Multiple Chemical Sensitivities

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October 27. 2021

To: Debbie Cress
Forest Supervisor
Santa Fe National Forest

Re: The Santa Fe Mountains Landscape Resiliency Project

The Multiple Chemical Sensitivities (MCS) Task Force is a statewide advocacy organization comprised of chemically sensitive New Mexicans and supporters. Formed in 1997, we are dedicated to increasing awareness of MCS and educating others about the hazards of high and low level environmental exposures.

Smoke from prescribed fires poses a significant public health threat, especially to those with chemical sensitivities, asthma, and other respiratory conditions.

Attached is a report entitled "*Human Health Effects of Wildland Smoke*" that summarizes the most up-to-date and available science on this issue.

The impact of smoke on the human environment needs to be carefully analyzed in an Environmental Impact Statement (EIS) for this project.

The proposal to use herbicides in the Santa Fe National Forest is also a public health threat. Herbicides are toxic chemicals that can get into the air and pollute land and water. An EIS should also examine the adverse health impacts of herbicides on the human environment, including impacts on vulnerable populations, such as those with chemical sensitivities.

Please keep me informed of developments regarding this project.

Thank you.

Ann McCampbell, MD

Co-Chair

MCS Task Force of NM

HUMAN HEALTH EFFECTS OF WILDLAND SMOKE

Ann McCampbell, MD

October 25, 2021

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Introduction

Smoke from wildfires and prescribed fires contain many hazardous chemicals and pose a significant public health threat. Fine particulate matter, PM2.5 (<2.5 micrometers) is most associated with causing adverse effects. Adverse health impacts can occur from both short-term smoke exposures (lasting hours to days) and long-term exposures.

"Even though woodsmoke [including wildland smoke] *is natural, it is not benign. Indeed, there is a considerable and growing body of epidemiologic and toxicologic evidence that both acute and chronic exposures to woodsmoke in developed country populations, as well as in the developing world, are associated with adverse health impacts. Woodsmoke contains thousands of chemicals, many of which have well-documented adverse human health effects, including such commonly regulated pollutants as fine particles, CO* [carbon monoxide], *and nitrogen oxides as well as ciliatoxic respiratory irritants such as phenols, cresols, acrolein, and acetaldehyde; carcinogenic organic compounds such as benzene, formaldehyde, and 1,3 butadiene; and carcinogenic cyclic compounds such as PAHs* [polyaromatic hydrocarbons]. *Woodsmoke contains at least five chemical groups classified as known human carcinogens by the International Agency for Research on Cancer (IARC), others categorized by IARC as probable or possible human carcinogens, and at least 26 chemicals listed by the U.S. EPA as hazardous air pollutants. Among the currently regulated pollutants in woodsmoke, fine particles (PM2.5) serve as the best exposure metric in most circumstances and, in addition, tend to be among the most elevated in relation to existing air quality standards"* (Naeher2007).

Wildfire and prescribed fire smoke also contain heavy metals, including mercury, as well as radionuclides. According to Carvalho, et al., forest fire smoke contains radionuclides at levels that can be greater than those in cigarette smoke (Carvalho2014).

Recent research has also found viable bacteria and fungi in wildland fire smoke (Kobziar2018), at levels above those present before burning occurred (Mirskaya2020). It has been hypothesized that these microorganisms could represent an infectious risk to the public. In 2019, researchers linked California wildfires with increased hospitalizations for invasive mold infections, including Aspergillus mold and Coccidioides fungus (causes Valley Fever) (Mulliken2019).

There is evidence that wildland smoke is more toxic than typical urban air pollution (Jaffe2020). Wildfire particulate matter tends to have a smaller particle size and contain more oxidative and proinflammatory components than urban particulates (Xu2020).

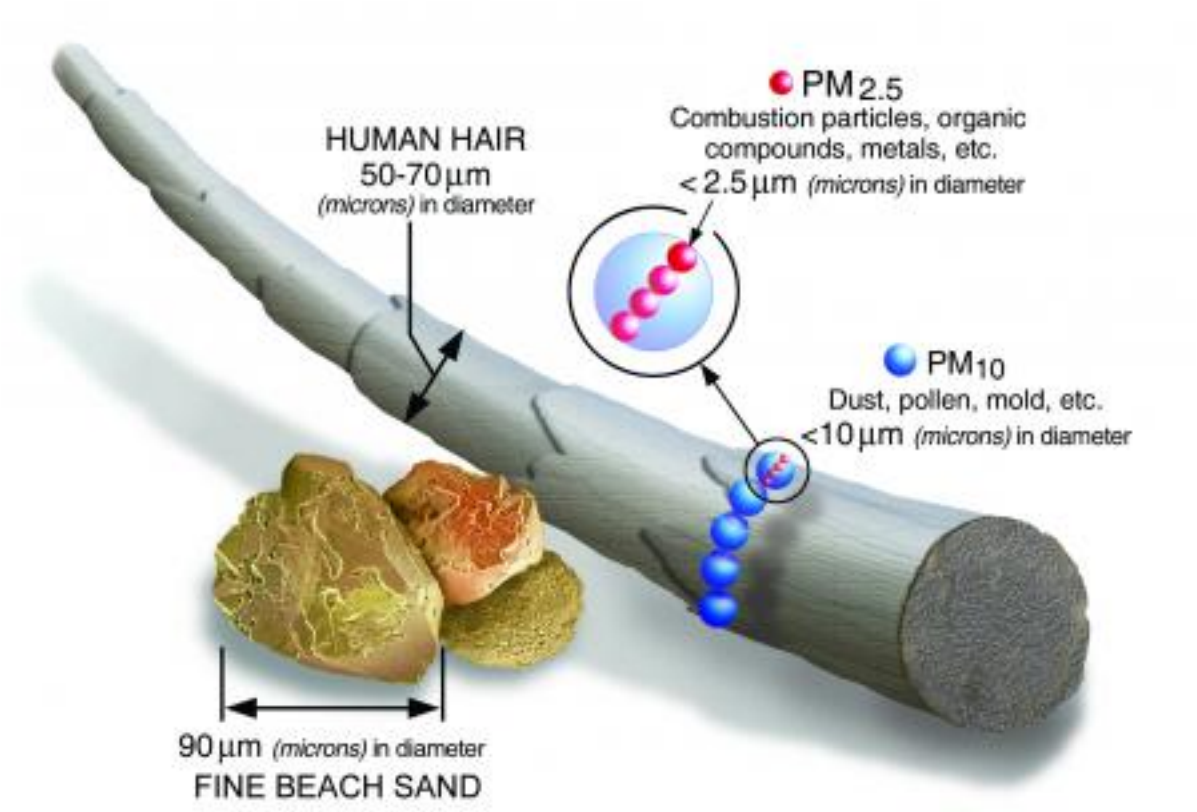
Exposure to wildfire and prescribed fire smoke can cause irritation of the eyes, nose, throat; wheezing, coughing, and shortness of breath; and headache. It can also aggravate lung disease, like asthma and chronic obstructive pulmonary disease

(COPD), and cardiovascular conditions. It is not unusual for people with chemical sensitivities to also experience severe fatigue, increased body pain, and brain "fog."

Wildland fire smoke can also have long-lasting effects on human health. Orr, et al., found a significant decrease in lung function among many community members one year following a wildfire event, and this remained decreased two years following the smoke exposure (Orr2020). Landguth, et al. found that higher daily average PM2.5 concentrations during a wildfire season was positively associated with increased influenza in the following winter influenza season (Landguth2020).

Particulate Matter (PM)

What is PM, and how does it get into the air?



Size comparisons for PM particles

PM stands for particulate matter (also called particle pollution): the term for a mixture of solid particles and liquid droplets found in the air. Some particles, such as dust, dirt, soot, or smoke, are large or dark enough to be seen with the naked eye. Others are so small they can only be detected using an electron microscope.

Particle pollution includes:

- **PM10:** inhalable particles, with diameters that are generally 10 micrometers [microns]

and smaller; and

- **PM2.5**: fine inhalable particles, with diameters that are generally 2.5 micrometers [microns] and smaller.

Sources of PM

These particles come in many sizes and shapes and can be made up of hundreds of different chemicals.

Some are emitted directly from a source, such as construction sites, unpaved roads, fields, smokestacks or fires.

Most particles form in the atmosphere as a result of complex reactions of chemicals such as sulfur dioxide and nitrogen oxides, which are pollutants emitted from power plants, industries and automobiles.

[www.epa.gov/pm-pollution/particulate-matter-pm-basics]

What are the Harmful Effects of PM?

Particulate matter contains microscopic solids or liquid droplets that are so small that they can be inhaled and cause serious health problems. Some particles less than 10 micrometers in diameter can get deep into your lungs and some may even get into your bloodstream. Of these, particles less than 2.5 micrometers in diameter, also known as fine particles or $PM_{2.5}$, pose the greatest risk to health.

The size of particles is directly linked to their potential for causing health problems. Small particles less than 10 micrometers in diameter pose the greatest problems, because they can get deep into your lungs, and some may even get into your bloodstream.

Exposure to such particles can affect both your lungs and your heart. Numerous scientific studies have linked particle pollution exposure to a variety of problems, including:

- premature death in people with heart or lung disease
- nonfatal heart attacks
- irregular heartbeat
- aggravated asthma
- decreased lung function
- increased respiratory symptoms, such as irritation of the airways, coughing or difficulty breathing.

People with heart or lung diseases, children, and older adults are the most likely to be affected by particle pollution exposure.

[www.epa.gov/pm-pollution/health-and-environmental-effects-particulate-matter-pm]

Both coarse particles (< 10 micrometers) and fine particles (<2.5 micrometers) enter the lungs and induce an inflammatory response. Fine particles can be absorbed into the blood stream and cause inflammation in all parts of the body.

One study found that PM samples collected during a wildfire event were more toxic than the same amount of PM from normal ambient air (Wegesser2009).

Another study notes "*there is some evidence to suggest that PM2.5 from wildfires may have a stronger adverse effect on respiratory morbidity at the same levels [emitted by other sources], and that there is a difference in toxicological response based on particulate matter source*" (Alman2016).

Shi, et al. found that both short- and long-term exposures to PM2.5 in a Medicare population were associated with all-cause mortality, even for exposure levels not exceeding U.S. EPA standards (12microgram/m3 annual average, 35 microgram/m3 daily). In addition, the association between short-term exposure and mortality appeared to be linear across the entire exposure distribution, indicating there was no safe level of exposure. (Shi2016).

Elliott, et al., compared dispensations for salbutamol [used to treat asthma] in forest fire-affected and non-fire-affected populations in British Columbia, Canada. Fire season PM2.5 was positively associated with salbutamol dispensations in all fire-affected populations (Elliott2013).

Sensitive Populations

Inhaling smoke is not good for anyone, even healthy people, but there are many populations at increased risk of harm from air pollution.

The following data on groups at risk from exposure to air pollution are provided by the American Lung Association in New Mexico (www.lung.org/our-initiatives/healthy-air/sota/city-rankings/states/new-mexico).

The total population of Santa Fe County is 150,358.

Under 18 years of age	26,394 (18%)
65 years and over	38,106 (25%)
Pediatric asthma	1,437 (1%)
Adult asthma	10,524 (7%)
COPD (chronic obstructive pulmonary disease)	7,951 (5%)
Lung cancer	51 (negligible)
Cardiovascular disease	11,248 (8%)
Poverty estimate	18,378 (12%)

It is also known that air pollution affects pregnant women. On average, pregnant women account for approximately 1% of a total population.

Those with chemical sensitivities are also at greater risk of harm from air pollution and can have serious physical reactions to exposures to even minute amounts of pollutants. A 1997 survey conducted by the NM Department of Health found that 16% of the state's respondents reported being unusually sensitive to everyday chemicals and 2% reported they had been diagnosed with multiple chemical sensitivities (MCS). The most recent national prevalence study found 25.9% of respondents reported being chemically sensitive and 12.8% of reported having been medically diagnosed with MCS (Steinemann2018).

Taken all together the above percentages of vulnerable populations totals 103%. Even though there are overlaps in these categories, it is clear that a significant portion of the population, possibly even the majority of the population, is at increased risk of harm from exposure to wildland fire smoke.

Respiratory Effects

Why is particle pollution a respiratory health concern?

Studies have linked particle pollution exposure to a variety of respiratory health effects, including:

- Respiratory symptoms including cough, phlegm, and wheeze.
- Acute, reversible decrement in pulmonary function.
- Inflammation of the airways and lung (this is acute and neutrophilic).
- Bronchial hyperreactivity.
- Acute phase reaction.
- Respiratory infections.
- Respiratory emergency department visits.
- Respiratory hospitalizations.
- Decreased lung function growth in children.
- Chronic loss of pulmonary function in adults.
- Asthma development.
- Premature mortality in people with chronic lung disease.

People with heart or lung disease, older adults, children, people with diabetes, and people of lower SES [socioeconomic status] are at greater risk of particle pollution-related health effects.

Though the respiratory system has remarkable resilience to air pollution via its repeated mobilization of defense and repair mechanisms, constant exposure to

elevated particle pollution will contribute to reduced respiratory function, even in apparently healthy people. Therefore, although we cannot completely avoid particle pollution exposure, taking simple steps to reduce exposure will reduce the severity of lung and systemic adverse health effects in both healthy and more sensitive people.

How does particle pollution affect the respiratory system?

Particles deposited in the respiratory tract in sufficient amounts can induce inflammation, which has been demonstrated in both animal and controlled human exposure studies. The extent of pulmonary inflammation depends on particle dose and composition. Controlled human exposure studies have demonstrated increased markers for pulmonary inflammation following exposure to a variety of different particle types. For example, organic carbon particles and transition metals from combustion sources can elicit a strong inflammatory response (U.S. EPA, 2009).

Airway inflammation increases airway responsiveness to irritants (e.g., cold air, particle pollution, allergens, lipopolysaccharides, and gaseous pollutants) and may reduce lung function by causing bronchoconstriction. At a cellular level, inflammation may damage or kill cells and compromise the integrity of the alveolar-capillary barrier. Repeated exposure to particle pollution aggravates the initial injury and promotes chronic inflammation with cellular proliferation and extracellular matrix reorganization (Berend, 2016).

Mobilization of the pulmonary immune system and other defense mechanisms is essential in the response to particle pollution. The overall balance between injury (inflammatory activity) and repair (anti-inflammatory defenses) plays an important role in the pathogenesis and progression of inflammatory respiratory diseases such as asthma. Inhalation of particle pollution may affect the stability or progression of these conditions through inflammatory effects in the respiratory tree.

What are the respiratory effects of acute exposure?

Studies have reported respiratory effects related to acute exposure to fine particles, including respiratory symptoms (especially in children and those diagnosed with asthma), reduction in pulmonary function, and increased airway inflammation and responsiveness. Additionally, epidemiologic studies have demonstrated that respiratory effects associated with particle pollution can be serious enough to result in emergency department visits and hospital admissions, including COPD and respiratory infections.

The relationship between exposure to ambient particle pollution concentrations and adverse respiratory effects was clearly demonstrated in a series of studies conducted in the Utah Valley by Pope (1989, 1991). When a steel mill, which was the source of 90 percent of local particle pollution emissions in the Utah Valley, was out of operation for one year, hospital admissions for bronchitis and asthma in the valley decreased by almost 50 percent and were comparable to those in other regions not polluted by the mill. Once mill operation resumed, hospital admissions increased. The mortality rate in the valley showed a similarly positive association with particle pollution levels during the same period.

The combination of experimental and epidemiologic studies has provided evidence of a relationship between short-term (daily) exposures to particle pollution and a number of respiratory-related effects, including elevated morbidity, higher frequency of emergency department visits and hospital admissions, as well as excess mortality. Often people with pre-existing diseases are at greatest risk for potential respiratory-related health effects due to short-term particle exposures (Ling and van Eeden, 2009).

What are the respiratory effects of chronic exposure?

Epidemiologic studies conducted in the U.S. and abroad provide evidence of associations between long-term exposure to fine particles and both decrements in lung function growth in children and increased respiratory symptoms.

The Children's Health Study (Gauderman et al., 2015) evaluated three separate cohorts of children who had longitudinal lung-function measurements recorded over the same 4-year age range (11 to 15 years) and in the same five study communities but during different calendar periods. The study shows an association between improvements in air quality in southern California and measurable improvements in lung-function development in children. Improved lung function (mean attained FEV1 and FVC values at 15 years of age) was most strongly associated with lower levels of particle pollution (PM2.5 and PM10) and nitrogen dioxide. These associations were observed in boys and girls, Hispanic white and non-Hispanic white children, and children with asthma and children without asthma, which suggests that all children have the potential to benefit from reduced exposure to particle pollution.

This same group conducted another epidemiological study that looked at the impact of improvement in particle pollution levels in Southern California between 1993 and 2012. It found that as ambient pollution levels improved there was a statistically significant decrease in bronchitis symptoms in children, especially among those with asthma (Berhane et al. 2016).

How does particle pollution affect people with asthma?

According to 2014 data, approximately 24 million Americans have asthma-- about 1 in 12 children (8.6 percent) and 1 in 14 adults (7.4 percent)(CDC, 2016). (For the most recent asthma data, go to CDC's asthma data page.) Asthma is a disease characterized by a variable degree of chronic airway inflammation associated with airway hyper-reactivity, reversible bronchoconstriction (used as an index of severity), and excessive mucus production. These abnormalities lead to symptoms and signs of asthma that include episodes of wheezing, coughing, chest tightness, and dyspnea. Asthma symptoms can be triggered by numerous environmental factors that can lead to bronchoconstriction and aggravate the disease. These environmental factors include exercise, humidity, temperature, allergens, viral infection, stress, and inhalation of air pollutants. Sensitivity to specific environmental triggers varies between individuals.

Several factors may cause people with asthma to be at increased risk of particle pollution-related health effects compared to healthy individuals.

- Airway hyper-reactivity and bronchoconstriction can affect particle deposition in a number of ways. Deposition can be increased in the conducting airways and some peripheral regions as a result of both obstruction and increased air flow to the better ventilated areas of the lung.
- Most particle pollution is pro-inflammatory and can aggravate pre-existing airway inflammation, which increases pro-inflammatory mechanisms and accelerates the inflammatory cascade.
- Allergens are a major factor in asthma development and exacerbation. The intensity of asthma symptoms and bronchial responsiveness varies with allergen sensitization, and people with allergic asthma are at increased risk for particle pollution-related health effects during times of high-allergen exposure (Silverman et al., 1992).

Biological particles (i.e., microbes, viruses, and spores) may lead to asthma exacerbation by aggravating inflammation and causing infection. In general, epidemiologic data provide substantial evidence for the association between particle pollution exposure and adverse effects in individuals with allergies and asthma, as assessed by frequency and severity of respiratory symptoms, pulmonary function changes, medication use, and ambient particle pollution levels. There is evidence that both the development of asthma and its exacerbation can be associated with particle pollution exposure.

What are the health disparities for asthma?

Asthma effects are more problematic in young children, older adults, minorities, and those with lower SES [socioeconomic status]. Minority children have higher

prevalence of asthma and higher rates of asthma-related emergency room visits, hospitalizations, and deaths than white children. Environmental factors related to SES may be important in contributing to these asthma disparities. For example, poor inner city children with asthma may be at increased risk from air pollution because they live near high-density traffic or industrial sources of particle pollution or because they have poor indoor air quality due to housing conditions. Because such children may have limited access to medical services and asthma education, these effects may be magnified (Gold et. al, 2005).

Children with asthma seem to be more affected by particle pollution than adults with asthma. This may be in part due to anatomical factors that lead to higher deposition of particle pollution in the tracheobronchial region of the lung in children. Other proposed factors that contribute to children being at increased risk of particle pollution-related health effects include behavioral factors such as increased exercise and time outdoors.

How does particle pollution affect people with COPD?

COPD is a major cause of disability and is the third leading cause of death in the United States (Ford et al, 2013). COPD is a lung disease characterized primarily by chronic airway inflammation, mucous hypersecretion, and progressive airflow limitation. These structural changes result in symptoms of cough, dyspnea, and increased sputum production. COPD comprises a spectrum of clinical disorders that include emphysema, bronchiectasis, and chronic bronchitis. COPD risk factors are both genetic and environmental. Elevated particle pollution contributes to the exacerbation of this disease and likely its pathogenesis. The role of other factors, such as developmental factors, is not well understood.

Like people with asthma, people with COPD are at greater health risk from particle pollution exposure than healthy individuals. There is a substantial overlap between the asthma and COPD phenotypes. The key underlying mechanisms are:

- Airway inflammation dominated by neutrophilic infiltration of the airways is aggravated by pro-inflammatory particle pollution.
- Increased sputum production combined with variable airway narrowing and uneven ventilation produces heterogeneous particle deposition, which creates localized regions (hot spots) with excessive particle accumulation. This accumulation, when combined with reduced particle clearance, substantially increases the probability of tissue injury beyond inflammation (Kim and Kang, 1997).

A few controlled human exposure studies of elderly COPD patients reported an

association between respiratory effects and fine particle pollution. Even fewer studies have explored the effects that ambient particle pollution may have on COPD development.

Epidemiological panel studies exploring the potential relationship between daily particle pollution levels and respiratory effects in people with COPD reported increased symptomatic response, increased use of evening medication (winter time), and small decrements in spirometric lung function in the days immediately following elevated particle pollution (PM10 and PM2.5) levels. Other endpoints showed an inconsistent response (Silkoff et al., 2005, Pope and Kanner, 1993). Though the induced effects may be insignificant, frequent exacerbation of symptoms and lung function impairment may accelerate COPD progression.

Time-series studies appear to show evidence of an association between acute exposures (i.e., daily) to particle pollution and morbidity (i.e., emergency department visits and hospital admissions) and mortality among individuals with COPD.

What is the role of fine particles in lung cancer incidence and mortality?

Prior to discussing the relationship between particle exposure and lung cancer, it is important to note the evolving scientific evidence. In the context of EPA, the evaluation of scientific evidence for cancer and other health effects for particle pollution occurs in an Integrated Science Assessment (ISA) as part of the National Ambient Air Quality Standards (NAAQS) review process.

The 2009 ISA (the most recent ISA for particle pollution) describes that epidemiologic studies generally demonstrated consistent positive associations between fine particle exposure and lung cancer mortality, but studies generally did not report associations between fine particles and lung cancer incidence (Pope et al., 1995; Dockery et al., 1993). Evidence from toxicological studies indicated that various combustion-related sources (e.g., wood smoke, coal combustion) are mutagenic and genotoxic, which provides biological plausibility for the effects observed in epidemiologic studies, and some components of particle pollution are known human carcinogens (e.g., specific arsenic, cadmium and chromium compounds).

More recently, the International Agency for Research on Cancer (IARC) conducted an evaluation on the carcinogenicity of outdoor air pollution, including particle pollution, and concluded that both are Group I agents (carcinogenic to humans). This IARC review focused on all routes of exposure and included an evaluation of individual components of particle pollution that are known human carcinogens.

Since 2009, there has been a dramatic increase in the number of epidemiologic studies that have examined chronic particle pollution exposures and both lung cancer incidence and mortality. Many of these studies are summarized in a meta-analysis by Hamra et al. (2014) that provide evidence of a relationship between fine particle exposure and lung cancer incidence and mortality. As part of the upcoming review of decisions to retain or revise the NAAQS for particle pollution, the EPA recently began an evaluation of evidence for cancer and other health effects resulting from particle pollution exposures that has been published since completion of the 2009 ISA. Information pertaining to publicly available drafts of EPA evaluations of the scientific evidence for particle pollution and lung cancer and other health effects can be found at EPA's Integrated Science Assessments website.

[www.epa.gov/particle-pollution-and-your-patients-health/health-effects-pm-patients-lung-disease]

Finlay et al. states that a review of the published evidence shows that human health can be severely affected by wildfires and that wood smoke has high levels of particulate matter and toxins. According to the authors, respiratory morbidity predominates, but cardiovascular, ophthalmic and psychiatric problems can also result (Finlay2012).

A study by Henderson, et al., found that forest fire smoke was associated with increases in self-reported symptoms, medication use, outpatient physician visits, emergency room visits, hospital admissions, and mortality. The associations were strongest for the outcomes most specific to asthma (Henderson2012).

U.S. Environmental Protection Agency (EPA) researchers investigated the relationship of PM2.5 levels with emergency department visits and acute hospitalizations for respiratory and cardiovascular outcomes during the 2012 Colorado wildfires. They found a positive association between PM2.5 and respiratory diseases, supporting evidence from previous research that wildfire PM2.5 is an important source for adverse respiratory health outcomes. (Alman2016).

Hutchinson, et al., examined the healthcare utilization of Medi-Cal recipients during the fall 2007 San Diego wildfires. They found that respiratory diagnoses, especially asthma, were elevated during the wildfires; wildfire-related healthcare utilization appeared to persist beyond the initial high-exposure period; increased adverse health events were apparent even at mildly degraded Air Quality Index levels; young children had bigger increases in healthcare visits during the peak fire period than older age groups; and very young children aged 0-1 were the most impacted experiencing a 243% increase in healthcare visits (Hutchinson2018).

Recently, Stowell, et al., studied the associations of wildfire smoke PM2.5 exposure with cardiorespiratory events in Colorado from 2011-2014. The authors found that for every 1 microgram/m3 increase in fire smoke PM2.5, statistically significant associations were observed for asthma and combined respiratory disease. Yet despite these associations, there was an absence of association with *total* PM2.5 concentrations. The authors state their findings point to potential toxic differences between smoke and non-smoke PM2.5 exposure, suggesting that PM2.5 from wildfire smoke could pose a significant threat to public health (Stowell2019).

Liu, et al., investigated wildfire-specific fine particulate matter and the risk of hospital admission in urban and rural counties. They found an increase in risk of respiratory admission during smoke wave days with high wildfire-specific PM2.5 (>37 micrograms/m3) compared to matched non-smoke wave days. They also concluded that "*Respiratory effects of wildfire-specific PM2.5 may be stronger than that of PM2.5 from other sources"* (Liu(a)2017).

Black, et al., evaluated the current literature on wildfire smoke and human health. The authors state that wildfire smoke has a distinct composition compared to other sources of air pollution. Wildfires produce proportionately more fine (under 2.5 microns) and ultrafine (under 1 micron) particulate, compared to coarse particulate, defined as particles fewer than 10 microns in size (PM10). The authors also note that wildfires also have a long smoldering phase, as wildfire containment strategies focus on extinguishing the flame phase while the smoldering phase is left to burn itself out, sometimes for months after a fire is considered contained. The smoldering phase of wood burning is associated with higher output of particulates, and can account for a large proportion of the total wildfire air pollutant emissions (Black2017).

Delfino, et al., studied the relationship of respiratory and cardiovascular hospital admissions during southern California wildfires of 2003. They found wildfire-related PM2.5 led to increased respiratory hospital admission, especially for asthma (Delfino2009).

Cascio addressed wildland fire smoke and human health. He states that systematic reviews conclude that a positive association exists between exposure to wildfire smoke or wildfire particulate (PM2.5) and all-cause mortality and respiratory morbidity. Respiratory morbidity includes asthma, chronic obstructive pulmonary disease (COPD), bronchitis and pneumonia. Susceptible populations include people with respiratory and possibly cardiovascular diseases, middle-aged and older adults, children, pregnant women and the fetus. The size of the population at risk from wildland fire smoke is increasing. Wildland fire smoke represents a costly and growing global public health problem. Studies have shown evidence that risks are greater for older women, African-Americans, and those with indicators of lower socio-economic status (Cascio2018).

Reid, et al., investigated health effects associated with fine particulate matter during 2008 wildfires in northern California. They observed a linear increase in risk for asthma hospitalizations and asthma emergency department (ED) visits with increasing PM2.5 during the wildfires. ED visits for chronic obstructive pulmonary disease (COPD) were associated with PM2.5 during the fires and this effect was significantly different from that found before the fires (Reid2016).

Roscioli, et al., employed models of human airway epithelium exposed to wildfire smoke-extract to examine changes in airway epithelial cell survival, fragility and barrier function. Primary epithelial models exposed to wildfire smoke-extract exhibited a significant blockade in autophagy, significant PARP cleavage indicative of apoptotic changes, and barrier dysfunction with significant increases in paracellular molecular permeability and reduction of tight junction proteins. These cultures also exhibited increased IL-6 secretion consistent with the aberrant and pro-inflammatory repair response observed in chronic obstructive pulmonary disease (COPD) airways. Further, blocks in autophagy and barrier disruption were significantly elevated in response to wildfire smoke-extract in comparison to similar exposure with cigarette smoke-extract (Roscioli2018).

Cardiovascular Effects

Why is particle pollution a cardiovascular health concern?

Cardiovascular disease accounts for the greatest number of deaths in the United States. One in three Americans has heart or blood vessel disease. According to the American Heart Association (AHA), one in every three deaths is attributed to cardiovascular disease, and expenses related to cardiovascular disease represent 17 percent of overall national health expenditures (Heidenreich et al., 2011).

Traditional risk factors for cardiovascular disease, such as male gender, age, increased blood pressure, high cholesterol, and smoking account for about 50 percent of cardiac events. Other factors acting independently, or together with established risk factors, likely contribute to the development of cardiovascular disease. Air pollution exposure is one such risk factor and is known to exacerbate existing, and contribute to the development of, cardiovascular disease.

Evidence linking ambient particle pollution exposure and adverse effects on cardiovascular disease is particularly strong (Newby et al., 2014). The AHA concluded both that exposure to increased concentrations of fine particle pollution over a few hours to weeks can trigger cardiovascular disease-related mortality and nonfatal events and that exposures of a few years or more to increased concentrations of fine particle pollution increases the risk of cardiovascular mortality and decreases life expectancy (Brook et al., 2010).

On an individual level, the risk of cardiovascular disease from particle pollution is smaller than the risk from many other well-established factors. At the population level, acute and chronic exposure to particle pollution can increase the numbers of cardiovascular events, including hospitalizations for serious cardiovascular events, such as coronary syndrome, arrhythmia, heart failure, and stroke, particularly in people with established heart disease.

Your patients with cardiovascular disease, including those who have angina, heart failure, particular arrhythmias, or that have risk factors for heart disease (e.g., those who are smokers, obese, or older adults) may be at greater risk of having an adverse cardiovascular event from exposure to fine particles. Unlike some risk factors that contribute to cardiovascular morbidity and mortality, people can take steps to reduce their exposure to particle pollution. Ninety-two percent of patients with cardiovascular disease are not informed of health risks related to air pollution (Nowka et al., 2011). Reducing population exposure to fine particle pollution has been shown to be associated with decreases in cardiovascular mortality (even within a few years of reduced exposure) (Pope et al., 2009; Correia et al., 2013).

How does particle pollution affect the cardiovascular system?

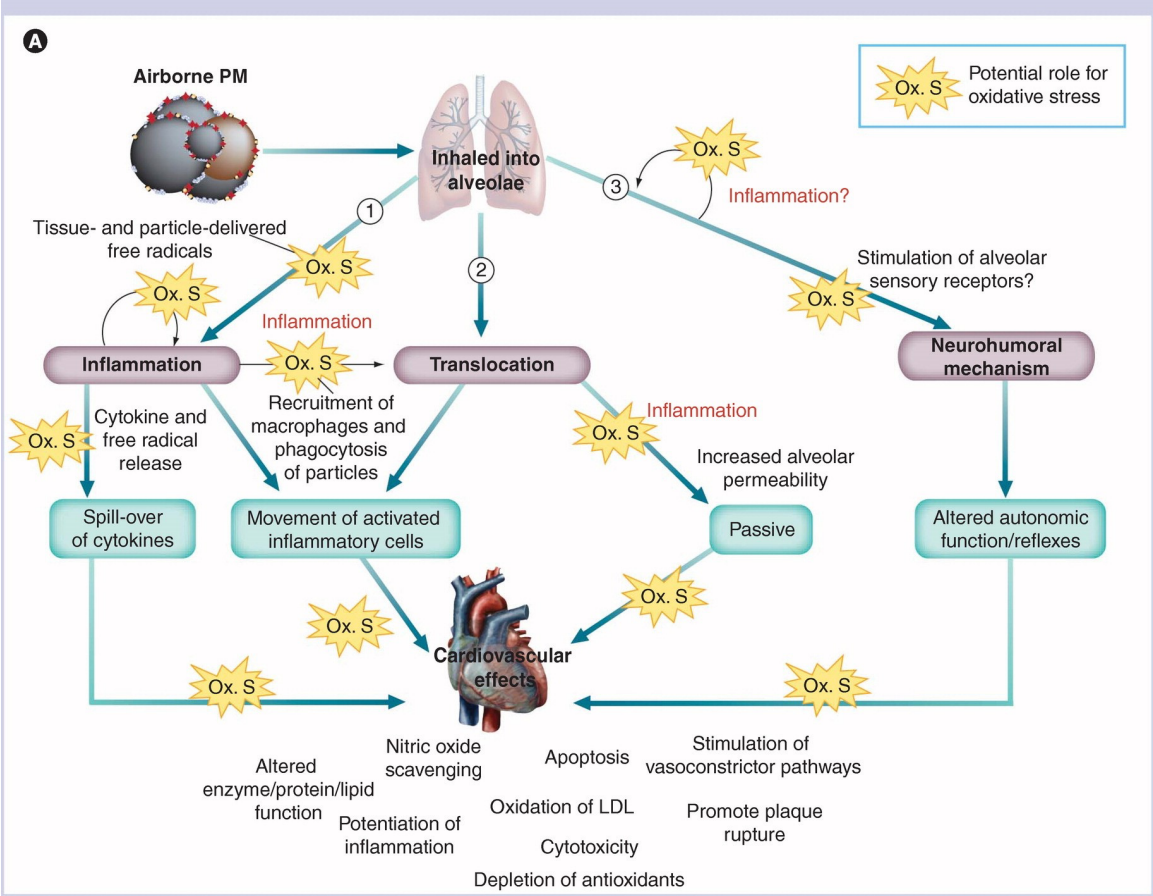
The mechanisms by which exposure to fine particle pollution can affect the cardiovascular system are under continuous examination. Exposure to inhaled fine particles appears to affect cardiovascular health through three primary pathways:

- Systemic inflammation.
- Translocation into the blood.
- Direct and indirect effects on the autonomic nervous system.

Oxidative stress is an underlying effect due to particle exposure that has been shown to impact endothelial function, pro-thrombotic processes, cardiac electrophysiology, and lipid metabolism.

The pathways by which inhaled particle pollution affects cardiovascular health are detailed in Figure 6. Inhaled particle pollution reaches the alveoli, at which point it can increase the formation of reactive oxygen species (ROS) and initiate an inflammatory response. Alveolar macrophages are likely to release pro-inflammatory cytokines with secondary effects on vascular control, heart rate variability, contractility, and rhythm. Alternatively, following deposition, small amounts (<1%) of ultrafine insoluble particles, or more soluble components of any size particles (e.g., metals), may translocate from the lung directly into the circulation where the particle might have direct impact on cardiovascular function

and/or have direct effects on the central nervous system with secondary effects on the heart and blood vessels via the autonomic nervous system.



6. Three possible mechanisms accounting for cardiovascular effects associated with particle pollution exposure. (1) Particles induce an inflammatory response in the lungs, leading to release of cytokines and other mediators that 'spill-over' into the systemic circulation. (2) Some ultrafine particles can translocate from the alveolus into the circulation and then interact directly with the heart and vasculature with or without the participation of inflammatory cells. (3) Particles might activate pulmonary sensory receptors and modulate the autonomic nervous system. Oxidative stress could play a role in exacerbating the stages of each pathway, as well as promoting interactions between pathways (e.g., in conjunction with inflammation). Reprinted with permission from Future Medicine Ltd. (Miller MR, Shaw CA, Langrish JP. 2012. From particles to patients: oxidative stress and the cardiovascular effects of air pollution. Future Cardiology 8(4):577-602).

Several studies identify an increase in inflammatory mediators and endothelial activation biomarkers after ambient particle pollution and urban air pollution

exposure (i.e., C-reactive protein (CRP), TNF-alpha, prostaglandin E2, CRP, interleukin-1b, and endothelin-1) (Pope et al., 2004; Calderón-Garcidueñas et al., 2008). Traffic-related particle pollution, which consists of a mixture of pollutants, has been shown to be positively associated with a number of subclinical effects including inflammation, oxidative stress, and autonomic nervous system balance, providing evidence that traffic-related air pollution is an important source of particle pollution (Chuang et al., 2007).

Studies using concentrated air particles provide important insights into the effects of exposure to particle pollution on cardiovascular endpoints in healthy adults. Ghio and colleagues studied the effects of either filtered air or particles concentrated from the immediate environment (averaging 120 μg/m3). After two hours of exposure, subjects underwent bronchoscopy and assessment of evidence of systemic inflammation. Exposure to fine particles produced no cardiopulmonary symptoms, yet bronchoalveolar lavage showed a mild increase in neutrophils in both the bronchial and alveolar fractions, and fibrinogen was increased the next day (Ghio et al., 2000).

What are the cardiovascular effects?

Acute and chronic exposure to fine particle pollution has been shown to increase the risk of hospitalizations for cardiovascular conditions and mortality. However, multi-city epidemiologic studies of mortality and hospital admissions have provided evidence of regional heterogeneity in risk estimates (Dominici et al., 2006; Zanobetti and Schwartz, 2009). It has often been hypothesized that the regional heterogeneity observed in epidemiologic studies may be a reflection of a number of factors including different sources and the chemical composition of fine particles varying between cities and regions, as well as demographic or exposure differences. To date, the underlying factors that contribute to this heterogeneity have yet to be identified.

Clinically important cardiovascular effects of inhaled particles include:

- Acute coronary syndrome, including myocardial infarction, unstable angina.
- Arrhythmia.
- Exacerbation of chronic heart failure.
- Stroke.
- Sudden cardiac death.

Such effects can be measured after acute exposure, and there is accumulating evidence that chronic exposure accelerates atherosclerosis and reduces life expectancy.

What are the acute exposure effects?

Population-based studies, small repeated-measure panel studies, and acute exposure studies in humans support the conclusion that inhalation of particle pollution induces small changes in blood pressure, oxygen saturation, endothelial function, systemic changes in acute phase reactants, coagulation factors, inflammatory mediators, and measures of oxidative stress. Systemic blood pressure and endothelial function changes, acute coronary syndrome (including myocardial infarction and unstable angina), increased ventricular arrhythmias in people with implantable (or internal) cardiac defibrillators (ICDs), exacerbation of heart failure, ischemic stroke, and cardiovascular mortality are all well-established clinical cardiovascular health effects associated with acute exposure to fine particles.

Blood pressure and endothelial function: Acute fine particle exposure causes a small increase in systolic and diastolic blood pressure (Liang et al. 2014). Some studies of persons without cardiovascular disease indicate a small increase in blood pressure associated with acute exposures to particle pollution (Auchincloss et al., 2008; Gong et al., 2003). Increased sympathetic tone and changes in vasomotor regulation caused by inflammation and oxidative stress are the most likely physiological changes to explain an increase in blood pressure (Brook et al., 2002). Because particle pollution is ubiquitous in the ambient air, exposures resulting in increases in blood pressure at the population level can have important public health implications (Brook, 2005).
Several studies indicate that filtering particles from the air either prevents or decreases particle-induced changes in physiological and biochemical determinants of heart and vascular health (Bräuner et al., 2008; Langrish et al., 2012). However, the clinical benefit of particle filters is not yet established.

Acute coronary syndrome: Several studies indicate that the onset of unstable angina and myocardial infarction are associated with exposure to ambient fine particle pollution (Pekkanen et al. 2002; Peters et al., 2001). Clinical studies show that particle pollution exposure increases the magnitude of ST-segment changes during ischemia, suggesting that exposure to particle pollution increases the severity of ischemia (Pekkanen et al., 2002).

Arrhythmias: An increase in ventricular and supraventricular arrhythmias in persons with ICDs (indicated by an increase in the discharge of the ICD) has been positively associated with increases in fine particle concentrations, which is supported by evidence of a linear exposure response (Peters et al., 2000; Rich et al., 2005; Dockery et al., 2005; Link et al., 2013). Stronger associations were found between air pollution and ventricular arrhythmias for episodes within a few days of a previous arrhythmia, suggesting that arrhythmias were triggered by air pollution episodes in combination with other factors that increased the

patient's susceptibility to arrhythmia.

Atrial fibrillation is the most common clinically important arrhythmia in older persons and imposes both a large societal burden and economic burden on the health-care system because of decreased quality of life, functional status, and hospitalizations for rhythm management, heart failure, and stroke (Rich et al., 2006).

While an increase in premature supraventricular beats is associated with long-term exposure to fine particle pollution (O'Neal et al., 2017a) and an increase in premature ventricular beats is associated with both short- and long-term exposure to increased concentrations of particle pollution (O'Neal et al., 2017b), the relationship between atrial fibrillation and exposure to particle pollution is less well established. Yet, a recent meta-analysis (Shao et al., 2016) showed an association between short-term exposure to fine particle pollution and the development of atrial fibrillation. The meta-analysis included some individuals with advanced heart disease managed with internal cardiac defibrillators (Link et al., 2013), and the positive association was not limited to fine particle pollution. Atrial fibrillation was also associated with increases in CO, NO and SO2.

Heart Failure: Several epidemiological studies indicate that acute exposures to fine particles contribute to hospitalization and mortality attributed to heart failure (Shah et al., 2013). For example, one large multi-city study conducted in 204 U.S. urban counties examined the association between daily changes in fine particle pollution concentrations and cardiovascular-related hospital admissions. The study reported that the largest association for hospital admissions is due to heart failure (Dominici et al., 2006). The authors reported a 1.28 percent increase in heart failure hospital admissions for a 10 μg/m3 increase in 24-hour average fine particle concentrations.

Stroke: Some studies have reported evidence of an increase in hospitalizations for stroke due to increases in the concentration of ambient fine particles (Wellenius et al., 2012). Recent meta-analyses have provided additional evidence supporting a relationship between both acute and chronic exposures to fine particles and various types of stroke (Shin et al., 2014; Shah et al., 2015). The mechanism for the increase in strokes is not known, but one study found a relatively small but independent effect of higher air temperature, dry air, upper respiratory tract infections, grass pollen, SO2, and suspended particles (Low et al., 2006).

Plaque stability and thrombus formation: Modulation of plaque stability and thrombus formation associated with fine particle exposure is suggested by epidemiological data indicating that the risk of unstable angina and myocardial

infarction may increase by as much as 4.5 percent for each 10 μg/m3 increase in 24-hour average fine particle concentrations (Pope et al., 2006).

What are the chronic exposure effects?

There is accumulating evidence that risk from chronic exposure (months to years) to inhaled fine particles accelerates atherosclerosis and reduces life expectancy.

Atherosclerosis: Several epidemiology studies, including the Multi-Ethnic Study of Atherosclerosis Air Pollution Study (MESA-Air), show that chronic air pollution exposure promotes atherosclerosis. This is indicated by the positive association between chronic particle exposure and an increase in coronary artery calcium (Kaufman et al., 2016), the severity of coronary artery disease (McGuinn et al., 2016), and increased thickness of the internal carotid artery (Künzli et al., 2005; Adar et al., 2013). Animal studies (Suwa et al., 2002, Araujo et al., 2008) have provided insights into the possible mechanisms that include inhibition of the anti-inflammatory capacity of plasma high-density lipoprotein, as well as increases in systemic oxidative stress, systemic inflammation, total amount of lipids in aortic lesions, and plaque turnover and extracellular lipid pools in coronary artery and aortic lesions.

Cardiovascular disease mortality: Fine particle pollution exposure is a risk factor for cardiovascular disease mortality via mechanisms that likely include pulmonary and systemic inflammation, accelerated atherosclerosis, and altered cardiac autonomic function (Dockery et al., 1993). The mechanisms of death associated with exposure to acute and chronic particle pollution are not fully known; however, prothrombotic effects precipitating myocardial infarction and stroke, autonomic instability precipitating arrhythmia, and increased oxidative stress worsening heart failure are speculated to account for the increased risk. Chronic exposure to particle pollution is most strongly associated with mortality attributable to ischemic heart disease, arrhythmia, heart failure and cardiac arrest (Pope et al., 2004).

Several seminal large cohort studies support the association of chronic exposure to air pollution and mortality. The Harvard Six Cities Study (Dockery et al., 1993) and American Cancer Society's Cancer Prevention II Study (Pope et al., 2002) both show an association between chronic exposure to ambient air pollution, particularly fine particle pollution, and an increased risk of death.

The Harvard Six Cities Study found statistically significant associations between chronic exposure to air pollution and mortality (Figure 7), specifically for fine particles and other pollutants strongly correlated with fine particles. Air pollution was also positively associated with cardiopulmonary disease deaths. A follow-up

study (Laden et al., 2006) assessing risk of death after considerable improvement in air quality in these six cities showed that the risk of mortality diminished in proportion to the reduction in air pollution.

The American Cancer Society's Cancer Prevention II Study also assessed the relationship between chronic exposure to fine particle pollution and mortality, but on a national scale (Pope et al., 2002). Similar to the Harvard Six Cities study, the ACS study reported evidence of a positive association between both all-cause and cardiopulmonary mortality and chronic exposure to fine particles.

In contrast to previous studies focusing on mortality in the entire population, Miller and colleagues examined the association between chronic exposure to fine particle pollution and clinical cardiovascular events in post-menopausal women without previous cardiovascular disease (Miller et al., 2007). In this study, one or more fatal or nonfatal cardiovascular event(s) occurred which included death from coronary heart disease or cerebrovascular disease, coronary revascularization, myocardial infarction, and stroke. The authors observed a marked increase in the risk of both cardiovascular events (24% increase), cerebrovascular events (35% increase), and cardiovascular-related mortality (76% increase) in this cohort of women for each 10 μg/m3 increase in the annual average concentration of fine particles.

[www.epa.gov/pmcourse/particle-pollution-and-cardiovascular-effects]

According to Huttunen, et al., short-term exposure to ambient air pollution is associated with increased cardiovascular mortality and morbidity and that this adverse health effect is thought to be mediated by inflammatory processes. They followed elderly individuals with ischemic heart disease. Average ambient PM2.5 concentration was 8.7 micrograms/m3. Of the studied pollutants, PM2.5 was most strongly associated with increased levels of inflammatory markers, most notably with C-reactive protein and IL-12 within a few days of exposure. There was also some evidence of an effect of particulate air pollution on fibrinogen and myeloperoxidase. The concentration of IL-12 was considerably (227%) higher during, rather than before, a forest fire episode. The authors state these findings show that even low levels of particulate air pollution from urban sources are associated with acute systemic inflammation and that particles from wildfires may exhibit pro-inflammatory effects (Huttunen2012).

Dennekamp, et al., found an association between exposure to forest fire smoke and in increase in the rate of out-of-hospital cardiac arrests (Dennekamp2015).

Zhao, et al., did a systematic review and meta-analysis of the impact of short-term exposure to air pollutants on the onset of out-of-hospital cardiac arrest. PM10, PM2.5, NO2 and ozone were found to be significantly associated with increase in

out-of-hospital cardiac arrest risk, with the strongest association being observed for PM2.5 (Zhao2017).

Haikerwal, et al., investigated the role of PM2.5 in triggering acute coronary events during the 2006-2007 wildfires in Victoria, Australia. They found PM2.5 exposure was associated with increased risk of out-of-hospital cardiac arrest and ischemic heart disease (Haikerwal(b)2015).

Jones, et al., studied cardiac arrests during California wildfires in 2015-2017 and found that out-of-hospital cardiac arrests increased with wildfire smoke exposure (Jones2020).

Deaths

Johnston, et al., estimated that worldwide exposure to fine-fraction PM2.5 from wildland fires during 1997-2006 were associated with approximately 340,000 deaths per year (Johnston2012).

Faustini, et al., analyzed the effects of wildfires and PM10 on mortality in 10 southern European cities. They found smoke was associated with increased cardiovascular mortality in urban residents, and PM10 on smoky days had a larger effect on cardiovascular and respiratory mortality than on other days (Faustini 2015).

Effects on Children and Pregnant Women

The American Pregnancy Association lists the following as being potential dangers of being exposed to air pollution during pregnancy: Low birth weight, preterm birth, autism, asthma, and fertility problems. Also noted is that particulate matter can cross the placenta and reach an unborn child. (https://americanpregnancy.org/pregnancy-health/how-air-pollution-impacts-pregnancy/)

According to an American Academy of Pediatrics Policy Statement, "*Ambient (outdoor) air pollution is now recognized as an important problem, both nationally and worldwide. Our scientific understanding of the spectrum of health effects of air pollution has increased, and numerous studies are finding important health effects from air pollution at levels once considered safe. Children and infants are among the most susceptible to many of the air pollutants. In addition to associations between air pollution and respiratory symptoms, asthma exacerbations, and asthma hospitalizations, recent studies have found links between air pollution and preterm birth, infant mortality, deficits in lung growth, and possibly, development of asthma*" (AAPeds2004).

Sraim, et al., reviewed studies looking at possible adverse effects of ambient air pollution on birth outcomes and concluded "*The evidence is sufficient to infer a causal relationship between particulate air pollution and respiratory deaths in the postneonatal period* [1 mo. – 1 yr. of age]". The authors further note that fetuses, in particular, are considered to be highly susceptible to a variety of toxicants, especially during critical windows (sensitive periods of development), because of higher rates of cell proliferation or changing metabolic capabilities (Sraim2005).

Tan-Soo, et al. found that prenatal exposure to smoke from the 1997 Indonesian forest fires resulted in decreased height at age 17. The authors state, "*Because adult height is associated with income, this implies a loss of 4% of average monthly wages for approximately one million Indonesian workers born during this period*" (Tan-Soo2019).

Kunzli, et al., investigated the health effects of the 2003 southern California wildfires on children. The authors found that fire smoke had a substantial effect on children's health. "*All symptoms (nose, eyes, and throat irritations; cough; bronchitis; cold; wheezing; asthma attacks), medication usage, and physician visits were associated with individually reported smoke exposure*." They also note that "*wildfire smoke contains numerous primary and secondary pollutants, including particles, polycyclic aromatic hydrocarbons, carbon monoxide, aldehydes, organic acids, organic compounds, gases, free radicals, and inorganic materials with diverse toxicologic properties" (*Kunzli2006).

According to Vicedo-Cabrera, et al., "*Exposure to wildfire smoke was associated with increased respiratory symptoms in this child population, particularly affecting susceptible individuals with asthma or rhinitis*." (Vicedo-Cabrera2016).

Lim, et al., did a systematic review and meta-analysis of the short-term effect of fine particulate matter on children's hospital admissions and emergency department visits for asthma. They found that children's hospital admissions and emergency department visits for asthma were positively associated with a short-term 10 microgram/m3 increase in PM2.5 (Lim2016).

Cancer

In an October 17, 2013 press release, the International Agency for Research on Cancer (IARC), an agency of the World Health Organization, announced that it had classified outdoor air pollution as *carcinogenic to humans* (Group 1). Particulate matter, a major component of outdoor air pollution, was evaluated separately and was also classified as *carcinogenic to humans* (Group 1)(IARC2013).

Kim and colleagues evaluated the mutagenicity and lung toxicity of particulate matter (PM) from flaming vs. smoldering phases of five biomass fuels (northern red oak, pocosin peat, ponderosa pine needles, lodgepole pine, and eucalyptus). They

found the greatest mutagenicity was for pine. Further, they concluded that smoldering emissions from wildland fires are highly mutagenic and support the notion that smoldering wood smoke is genotoxic and ultimately carcinogenic in humans (Kim2018).

Covid-19

Short-term and long-term exposure to PM2.5 is associated with an increased risk of Covid-19 cases and deaths.

According to the U.S. Centers for Disease Control and Prevention, "*wildfire smoke can irritate your lungs, cause inflammation, affect your immune system, and make you more prone to lung infections, including SARS-CoV-2, the virus that causes COVID-19*." [www.cdc.gov/disasters/covid-19/wildfire_smoke_covid-19.html]

Exposure to particulate matter increases the expression of angiotension-converting enzyme 2 (ACE2) in the lungs which facilitates SARS-CoV-2 viral adhesion.

Wu, et al., found that long-term exposure to air pollution was positively associated with higher mortality rates. They found that for every 1 microgram/m3 increase in PM2.5 exposure, there was an 11% increase in Covid-19 deaths (Wu2020).

Zhou, et al., investigated the number of Covid-19 cases and deaths in California, Oregon, and Washington during the 2020 wildfires. They concluded the overall number of Covid-19 cases and deaths attributable to daily increases in PM2.5 from wildfires was 19,742 and 748, respectively (Zhou2021).

In addition, the CDC also notes that *"[p]eople who currently have or who are recovering from COVID-19 may be at increased risk of health effects from exposure to wildfire smoke due to compromised heart and/or lung function related to COVID-19*." [www.cdc.gov/disasters/covid-19/wildfire_smoke_covid-19.html]

National Ambient Air Quality Standards (NAAQS)

The National Ambient Air Quality Standards (NAAQS) and EPA's associated Air Quality Index (AQI) do not adequately protect public health.

Several studies have found adverse health impacts from exposure to particulate levels below current standards, i.e. at levels AQI considers "*healthy*." There appears to be no threshold level of PM2.5 below which no adverse health effects occur. This has led some researchers to call for revising NAAQS standards.

According to a 2003 report by a World Health Organization (WHO) Working Group:

"Epidemiological studies on large populations have been unable to identify a threshold concentration below which ambient PM has no effect on health. It is likely that within any large human population, there is such a wide range in susceptibility that some subjects are at risk even at the lowest end of the concentration range."

Harvard researchers investigated the association between short-term exposures to ambient fine particulate matter (PM2.5) and ozone, and mortality. They found that in the U.S. Medicare population from 2000 to 2012, short-term exposures to PM2.5 and warm-season ozone were significantly associated with an increased risk of mortality. This risk occurred at levels below current national air quality standards, suggesting that these standards may need to be reevaluated. They also found no evidence of a threshold in the exposure-response relationship below which no increased mortality occurred (Di2017).

Schwartz, et al., investigated the concentration-response relation between PM2.5 and daily deaths. The authors state that several recent articles have reported that exposure to PM10 is associated with daily deaths with no evidence of a threshold. In this study, the authors found an association between exposure to PM2.5 and daily deaths with no level of a threshold down to the lowest levels of PM2.5. They state "*In fact, the curve is quite linear over the exposure range from 0 to 35 micrograms/m3*" and this is consistent with previous results (Schwartz2002).

Fire Accelerant Chemicals

A variety of chemical accelerants are used to start prescribed fires. These chemicals and their breakdown products get into the air and leave residues on the ground. Diesel fuel and gasoline are commonly used to start fires on the ground. Aerial release of ping pong-like balls containing potassium permanganate (KMnO4), ethylene glycol, and polystyrene shells are also used to start fires.

According to the International Chemical Safety Card for potassium permanganate, this chemical "*gives off irritating or toxic fumes (or gases) in a fire*." "*This substance is corrosive to the eyes, skin and respiratory tract*," and " … *may have effects on the lungs. This may result in bronchitis and pneumonia. Animal tests show that this substance possibly causes toxicity to human reproduction or development*" (IPSCpotassiumpermangate).

According to a Risk Assessment of Residues of Fire Accelerant Chemicals prepared for the Intermountain Region USDA Forest Service, Table 1-1, Chemicals Evaluated in Risk Assessment, the residues expected from the use of the above accelerants are diesel fuel, gasoline, MTBE, manganese dioxide, potassium hydroxide, and polystyrene. Styrene is also expected to be released as a gas.

This risk assessment evaluates the risk to humans of drinking contaminated water or fish, and ingesting contaminated soil. It gives recommendations for the quantity

of each kind of accelerant that can be used to avoid harm to humans. It did not, however, assess the human health risk of breathing fire accelerant chemicals (RiskAssessmentResiduesFireAccelerants2002; and companion literature search, LitSearchResiduesFireAccelerants2002).

Although this risk assessment contains useful information, it cannot be relied on to assess the risk to the public of exposure to fire accelerant chemicals because it is out-of-date and does not assess the impact of inhalation of fire accelerant chemicals, the most likely route of public exposure.

Prescribed Fires

According to the U.S. Environmental Protection Agency (EPA), " … *using prescribed fire is not without risk as it can result in smoke related air quality and public health impacts*". In its 2021 report "Comparative Assessment of the Impacts of Prescribed Fire Versus Wildfire (CAIF): A Case Study in the Western United States," the EPA states the goal of the report is to help risk managers take public health impacts of smoke into account when making decisions about using prescribed fire. [www.epa.gov/newsreleases/epa-releases-report-comparing-air-quality-and-public-health-impacts-prescribed-fire]

Even though health impacts from individual prescribed fires (or naturally-occurring fires to which accelerant is added) tend to be lower than those associated with severe wildfires, their cumulative impacts are often similar to or exceed the impact of wildfires, since they occur with much greater frequency.

In Australia, Arriagada et al., examined health impacts from elevated particulate air pollution from 2002-2017. They found that of the total estimated health costs resulting from particulate air pollution, 51% was attributable to prescribed burns and 41% to wildfires (Arriagada2020).

In Georgia, researchers found that the health burden of smoke from prescribed burning is comparable to that estimated for other major emission sectors, such as vehicles and industrial combustion. They say these findings call for greater attention to the air quality impacts of prescribed burning (Afrin2021).

In many ways prescribed fires are similar to wildfires, except they tend to be lower intensity burns that emit greater amounts of particulate matter per unit of biomass burned than wildfires.

"*Unlike wildfires that are of high intensity, prescribed fires are cool low-intensity burns and produce relatively short plumes … While low-intensity prescribed burns (low heat, light emissions) cause minimal risk to life and property, they can however emit large amounts of smoke particulates." "Smoke from prescribed burning can have a substantial impact on air quality and the environment. Prescribed burning is a*

significant source of fine particulate matter (PM2.5 aerodynamic diameter<2.5 micrometers) and these particulates are found to be consistently elevated during smoke events. Due to their fine nature PM2.5 are particularly harmful to human health" (Haikerwal(a)2015).

"*… There is a need to understand the influence of prescribed burning smoke exposure on human health. This is important especially since adverse health impacts have been observed during wildfire events when PM2.5 concentrations were similar to those observed during prescribed burning events* (Haikerwal2015).

According to Ward & Hardy, "*The smoldering combustion phase produces high emissions of particulate matter and CO [carbon monoxide]. Fires of low intensity (those in which the flaming combustion phase is barely sustained) produce high emissions of particulate matter." "For many fuel types, emissions from the smoldering phase overwhelm emissions produced through flaming combustion processes – typical of measurements of smoke from wildfires and during the later stages of prescribed fires"* (Ward&Hardy1991).

Kim et al. found that flaming combustion conditions were more efficient, converting much of the carbon to CO2, whereas more carbonaceous PM and CO (carbon monoxide) were emitted during smoldering. They also found that smoldering pine and pine needles had the highest levels of mutagenicity potencies (Kim2018).

Alves, et al., analyzed smoke from a wildfire in a mixed evergreen forest in Portugal and found that particulate matter and organic carbon emissions were significantly enhanced under smoldering fire conditions (Alves2011).

Navarro, et al, found that PM2.5 concentrations from wildfire smoke were significantly lower than PM2.5 concentrations from prescribed fire smoke (Navaffo2018).

Prescribed fires (and naturally-occurring fires to which is accelerant is added) also differ from wildfires in the application of fire accelerants. These are toxic chemicals that get into the air and can contaminate soil and water. And while prescribed fires can be timed to reduce smoke impacts, the increasing practice of adding accelerant to naturally-occurring fires removes this benefit.

<u>Mitigation</u>

In Air Quality Impacts from Prescribed Forest Fires under Different Management Practices, the authors state that large amounts of air pollutants are emitted during prescribed forest fires. Such emissions and corresponding air quality impacts can be modulated by different forest management practices. These include, but are not limited to, 1) making more use of mechanical thinning to reduce the amount of burning, 2) choosing to burn during seasons that emit fewer pollutants (in Georgia,

equivalent fires in the spring and winter were found to emit more PM2.5 than those in the summer), and 3) better controlling emissions from smoldering by, for example, burning before precipitation (Tian, 2007).

Ravi, et al., investigated the impacts of smoke from prescribed fires on air quality, health, and visibility in protected natural environments. They concluded that a 70% reduction in fire activities would result in significant improvement in air quality in areas in western Oregon, northern Idaho and western Montana where most prescribed fires occur. Using BenMAP, a health impact assessment tool, they showed that several hundred additional deaths, several thousand upper and lower respiratory symptom cases, several hundred bronchitis cases, and more than 35,000 work day losses can be attributed to prescribed fires and these health impacts decrease by 25-30% when a 30% fire emission scenario is considered. The authors also note that as prescribed burning activities become more frequent, they can be more detrimental for air quality and health (Ravi2018).

Environmental Justice

As noted above, people with lower socioeconomic status (SES) are at higher risk of suffering adverse health impacts from air pollution. This can occur because their exposures are higher than those with higher SES. But in addition, for any level of air pollution, they suffer disproportionately more harm. Forastiere, et al., investigated whether social class is an effect modifier of exposure to PM10 (particulate matter with a diameter < 10 microns) and found that their results confirmed previous suggestions of a stronger effect of particulate air pollution among people in low social class (Forastiere2007).

Liu, et al., found increased risks of respiratory admissions from wildfire smoke was significantly higher for blacks than whites (21.7% vs. 6.9%) and stated that their study raised important environmental justice issues (Liu(b)2017).

Executive Order 12898, issued in 1994, established the responsibility of each Federal agency to "*make achieving environmental justice part of its mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low income populations ....*" An accompanying Presidential Memorandum directed that human health, economic, and social effects, including effects on minority communities and low-income communities, be included in the analysis of environmental effects pursuant to NEPA. [https://ceq.doe.gov/nepa-practice/justice.html]

Therefore, analysis of the human health effects of smoke from prescribed fires must also include a breakdown of the severity of those impacts according to socioeconomic status (SES).

Costs

Jones, et al. found "*On average, wildfire smoke in the Western U.S. creates $165 million in annual morbidity and mortality health costs*" (Jones2017).

The costs to the public of exposure to smoke from wildland fires, including prescribed fires, can be considerable. Costs can include medical costs (doctor visits, ED visits, and hospitalization), increases in medication, evacuation costs (transportation, lodging, driver/attendant), purchase of air filters and masks, and lost days of work.

Rappold and colleagues evaluated the health impacts and economic value of wildland fire episodes in the U.S. from 2008-2012. Their models suggest that areas including northern California, Oregon and Idaho in the West, and Florida, Louisiana and Georgia in the East were most affected by wildland fire events in the form of additional premature deaths and respiratory hospital admissions. They estimated the economic value of these cases due to short term exposures as being between $11 and $20 billion (2010$) per year, with a net present value of $63 billion for the 5 years studied (95% confidence intervals $6-$170); and estimated the value of long-term exposures as being between $76 and $130 billion (2010$) per year, with a net present value of $450 billion for the 5 years studied (95% confidence intervals $42-$1,200)" (Rappold2014).

Borgschulte, et al., examined the importance of air pollution from wildfire smoke in the determination of national, annual labor income in the United States. Wildfires account for about 20% of the fine particulate matter emitted in the U.S. They note that air pollution exposure increases infant and elderly mortality and reduces long-run health and future income among those exposed in utero and infancy. Air pollution also negatively affects the broader adult population, for example, by reducing labor supply and productivity.

In summary, this paper found that smoke exposure reduces earnings in both the year of exposure (each day of wildfire smoke exposure caused a roughly linear reduction in labor income of 0.07% in the year of exposure) and the following year, lowers labor force participation, and increases Social Security claiming and payments. With an average of 17.7 days of annual smoke exposure per person, earnings losses sum to 1.26% of annual labor income. They further estimated that the welfare cost of these los earnings is higher than the mortality cost of wildfire smoke (Borgschulte2019).

Kochi, et al., summarized previous studies of the economic analysis of wildfire-smoke-induced health damage, noting that the omission of mortality costs may have resulted in substantial underestimates of total health costs. They further note, "*Work days lost, restricted-activity days, and minor restricted-activity days contribute substantially to total morbidity-related costs, and account for 36 to 74% of total estimated health costs in the studies that did not consider premature mortality.*"

The authors concluded, "*The economic costs of adverse health effects associated with exposure to wildfire smoke should be given serious consideration in determining the optimal wildfire management policy*." "*For example, concerns about adverse health effects from 2008 wildfires in northern California prompted the USDA Forest Service to actively suppress all wildfires in California*" (Kochi2010).

AMERICAN ACADEMY OF PEDIATRICS

POLICY STATEMENT

Organizational Principles to Guide and Define the Child Health Care System and/or Improve the Health of All Children

Committee on Environmental Health

Ambient Air Pollution: Health Hazards to Children

ABSTRACT. Ambient (outdoor) air pollution is now recognized as an important problem, both nationally and worldwide. Our scientific understanding of the spectrum of health effects of air pollution has increased, and numerous studies are finding important health effects from air pollution at levels once considered safe. Children and infants are among the most susceptible to many of the air pollutants. In addition to associations between air pollution and respiratory symptoms, asthma exacerbations, and asthma hospitalizations, recent studies have found links between air pollution and preterm birth, infant mortality, deficits in lung growth, and possibly, development of asthma. This policy statement summarizes the recent literature linking ambient air pollution to adverse health outcomes in children and includes a perspective on the current regulatory process. The statement provides advice to pediatricians on how to integrate issues regarding air quality and health into patient education and children's environmental health advocacy and concludes with recommendations to the government on promotion of effective air-pollution policies to ensure protection of children's health. *Pediatrics* 2004;114:1699–1707; *air pollution, adverse effects, children, asthma, environmental health.*

ABBREVIATIONS. $PM_{2.5}$, particulate matter with a median aerodynamic diameter less than 2.5 μm; PM_{10}, particulate matter with a median aerodynamic diameter less than 10 μm; EPA, Environmental Protection Agency; HAP, hazardous air pollutant; AQI, air quality index.

INTRODUCTION

Although it has been 3 decades since passage of the Clean Air Act in 1970 (Pub L No. 91–604), the air in many parts of the United States is far from clean. Air quality has improved in some areas but decreased in others.[1] In addition, there are important health effects from air pollutants at levels once considered safe. Children and infants are among the most susceptible to many of the air pollutants.

In 2002, approximately 146 million Americans were living in areas where monitored air failed to meet the 1997 National Ambient Air Quality Standards for at least 1 of the 6 "criteria air pollutants": ozone, particulate matter, sulfur dioxide, nitrogen dioxide, carbon monoxide, and lead (Table 1).[1] Although the standards for ozone and particulate matter were revised in 1997, legal barriers have delayed timely implementation.[2] Recent reports have identified adverse health effects at levels near or below the current standards for ozone, particulate matter, and nitrogen dioxide. Thus, the 1997 federal standards may not adequately protect children. Additionally, numerous other toxic air pollutants are of public health concern.[3]

Outdoor air pollution is also a major problem in developing countries. The World Health Organization found that the air quality in large cities in many developing countries is remarkably poor and that very large numbers of people in those countries are exposed to ambient concentrations of air pollutants well above the World Health Organization guidelines for air quality (www.who.int/ceh/publications/en/11airpollution.pdf).

Scientific understanding of the health effects of air pollution, including effects on children, has increased in the last decade. This statement updates a 1993 American Academy of Pediatrics (AAP) statement titled "Ambient Air Pollution: Respiratory Hazards to Children."[4]

EFFECTS OF AIR POLLUTION ON CHILDREN

Children are more vulnerable to the adverse effects of air pollution than are adults. Eighty percent of alveoli are formed postnatally, and changes in the lung continue through adolescence.[5] During the early postneonatal period, the developing lung is highly susceptible to damage after exposure to environmental toxicants.[5–7]

Children have increased exposure to many air pollutants compared with adults because of higher minute ventilation and higher levels of physical activity.[8] Because children spend more time outdoors than do adults, they have increased exposure to outdoor air pollution.[9,10]

Infants, children, the elderly, and those with cardiopulmonary disease are among the most susceptible to adverse health effects from criteria pollutants.[11–15] Lead is neurotoxic, especially during early childhood. Carbon monoxide interferes with oxygen transport through the formation of carboxyhemoglobin. Other criteria pollutants (ozone, sulfur dioxide, particulate matter, nitrogen dioxide) have respiratory effects in children and adults, including increased respiratory tract illness, asthma exacerbations, and decreased lung function (eg, changes in peak flow).[11–12] In adults, particulate air pollution is associated with respiratory and cardiovascular hos-

doi:10.1542/peds.2004-2166

TABLE 1. National Ambient Air Quality Standards for Criteria Air Pollutants, 1997

Pollutant	Primary Standards*
Ozone	
1-h average	0.12 ppm (235 $\mu g/m^3$)
8-h average	0.08 ppm (157 $\mu g/m^3$)
PM_{10}	
Annual arithmetic mean	50 $\mu g/m^3$
24-h average	150 $\mu g/m^3$
$PM_{2.5}$	
Annual arithmetic mean	15 $\mu g/m^3$
24-h average	65 $\mu g/m^3$
Sulfur dioxide	
Annual arithmetic mean	0.03 ppm (80 $\mu g/m^3$)
24-h average	0.14 ppm (365 $\mu g/m^3$)
Nitrogen dioxide	
Annual arithmetic mean	0.053 ppm (100 $\mu g/m^3$)
Carbon monoxide	
8-h average	9 ppm (10 mg/m^3)
1-h average	35 ppm (40 mg/m^3)
Lead	
Quarterly average	1.5 $\mu g/m^3$

Additional information on air quality standards are available at www.epa.gov/air/criteria.html.
* People residing in regions with pollutant concentrations above the primary standard may experience adverse health effects from poor air quality.

pitalizations, cardiovascular mortality,[16] and lung cancer.[17] Air pollution also has effects on indirect health indicators such as health care utilization and school absences.[11–13]

Although numerous studies have shown that outdoor air pollution exacerbates asthma, the effect of outdoor air pollution on the development of asthma has been less clear. Recently, a prospective study found that the risk of developing asthma was not greater, overall, in children living in communities with high levels of ozone or particulate air pollution. However, in communities with high levels of ozone, there was an increased risk of developing asthma in a small subset of children involved in heavy exercise (participation in 3 or more team sports per year [relative risk: 3.3; 95% confidence interval: 1.9–5.8]). This increased risk with heavy exercise was not seen in low-ozone communities. Time spent outside was also associated with new cases of asthma in high-ozone communities (relative risk: 1.4; 95% confidence interval: 1.0–2.1) but not in low-ozone communities.[18] Additional studies are needed to define the role of outdoor air pollution in the development of asthma.

Children in communities with higher levels of urban air pollution (acid vapor, nitrogen dioxide, particulate matter with a median aerodynamic diameter less than 2.5 μm [$PM_{2.5}$], and elemental carbon [a component of diesel exhaust]) had decreased lung function growth, and children who spent more time outdoors had larger deficits in the growth rate of lung function.[19,20] Ambient air pollution (especially particulate matter with a median aerodynamic diameter less than 10 μm [PM_{10}]) has also been associated with several adverse birth outcomes, as discussed in the next section.

Levels of ozone and particulate matter are high enough in many parts of the United States to present health hazards to children.[1] Additionally, National Ambient Air Quality Standards for nitrogen dioxide may not be protective. Findings on these pollutants are summarized here.

Ozone

Ambient ozone is formed by the action of sunlight on nitrogen oxides and reactive hydrocarbons, both of which are emitted by motor vehicles and industrial sources. The levels tend to be highest on warm, sunny, windless days and often peak in midafternoon, when children are most likely to be playing outside.

Ozone is a powerful oxidant and respiratory tract irritant in adults and children, causing shortness of breath, chest pain when inhaling deeply, wheezing, and cough.[11] Children have decreases in lung function, increased respiratory tract symptoms, and asthma exacerbations on days with higher levels of ambient ozone.[11,21–23] Increases in ambient ozone have been associated with respiratory or asthma hospitalizations,[24,25] emergency department visits for asthma,[26] and school absences for respiratory tract illness.[27] In Atlanta, Georgia, summertime children's emergency department visits for asthma increased 37% after 6 days when ozone levels exceeded 0.11 ppm.[25] In southern California, school absences for respiratory tract illness increased 63% in association with a 0.02-ppm increase in ozone.[27]

In healthy adults, ozone causes airway inflammation and hyperreactivity, decrements in pulmonary function, and increased respiratory tract symptoms.[11] Ozone exposures at concentrations of 0.12 ppm or higher can result in decrements in lung function after subsequent challenge with aeroallergen.[28] Although most of the controlled studies of ozone exposure have been performed with adults, it is reasonable to believe that the results of these findings could be extended to children.

Ozone may be toxic at concentrations lower than 0.08 ppm, the current federal regulatory standard. Field studies suggest potential thresholds of between 0.04 and 0.08 ppm (1-hour average) for effects on lung function.[29–31] Recent studies of hospitalizations for respiratory tract illness in young children and emergency department visits for asthma suggest that the effects of ozone may occur at ambient concentrations below 0.09 ppm.[32,33] Another study found associations of ozone and respiratory symptoms in children with asthma at levels below the current US Environmental Protection Agency (EPA) standards.[34] If these findings are confirmed, the ozone standards may need additional revision.

In addition to studies on short-term effects, 2 recent studies of college freshmen suggest that increasing cumulative childhood exposure to ozone may affect lung function when exposed children reach young adulthood, particularly in measures of flow in small airways.[35,36] Early childhood exposures may, therefore, be particularly important.[35]

Particulate Matter

PM_{10} is small enough to reach the lower respiratory tract and has been associated with a wide range of serious health effects. PM_{10} is a heterogeneous

mixture of small solid or liquid particles of varying composition found in the atmosphere. Fine particles ($PM_{2.5}$) are emitted from combustion processes (especially diesel-powered engines, power generation, and wood burning) and from some industrial activities. Coarse particles (diameter between 2.5 and 10 μm) include windblown dust from dirt roads or soil and dust particles created by crushing and grinding operations. Toxicity of particles may vary with composition.[37,38]

Particle pollution contributes to excess mortality and hospitalizations for cardiac and respiratory tract disease.[14,39–41] The mechanism for particulate matter–associated cardiac effects may be related to disturbances in the cardiac autonomic nervous system, cardiac arrhythmias, or increased blood concentrations of markers of cardiovascular risk (eg, fibrinogen).[16,42]

Daily changes in mortality rates and numbers of people hospitalized are linked to changes in particulate air pollution.[14,39–41] These studies and others have estimated that for every 10 $\mu g/m^3$ increase in PM_{10}, there is an increase in the daily mortality rate between 0.5% and 1.6%. Effects were seen even in cities with mean annual PM_{10} concentrations between 25 and 35 $\mu g/m^3$. These recent studies suggest that even the current federal standards for $PM_{2.5}$ (24-hour standard = 65 $\mu g/m^3$; annual standard = 15 $\mu g/m^3$) and PM_{10} (24-hour standard = 150 $\mu g/m^3$; annual standard = 50 $\mu g/m^3$) should be lowered to protect public health. In 2002, California adopted more stringent standards for particulate matter: the annual average standard for $PM_{2.5}$ is 12 $\mu g/m^3$ and for PM_{10} is 20 $\mu g/m^3$.[43]

In children, particulate pollution affects lung function[44–46] and lung growth.[19] In a prospective cohort of children living in southern California, children with asthma living in communities with increased levels of air pollution (especially particulates, nitrogen dioxide, and acid vapor) were more likely to have bronchitis symptoms. In this study, bronchitis symptoms refers to a parental report of "one or more episodes of 'bronchitis' in the past 12 months" or report that, "apart from colds, the child usually seems to be congested in the chest or able to bring up phlegm").[47] The same mix of air pollutants was also associated with deficits in lung growth (as measured by lung function tests).[19] Recent studies in different countries have also found associations between ambient air pollution (especially particulates and/or carbon monoxide) and postneonatal infant mortality (attributable to respiratory causes and possibly sudden infant death syndrome),[48,49] low birth weight,[50–53] and preterm birth.[51,54–56]

The relative contribution of fine versus coarse particles to adverse health effects is being investigated. In studies of cities on the East Coast, fine particles seem to be important.[57] In other areas, coarse particles have a stronger or similar effect.[58] Several studies have found that fine particles from power plants and motor vehicles[59] or industrial sources[60] may be more closely associated with mortality.

Nitrogen Dioxide

Nitrogen dioxide is a gaseous pollutant produced by high-temperature combustion. The main outdoor sources of nitrogen dioxide include diesel and gasoline-powered engines and power plants. Levels of nitrogen dioxide around urban monitors have decreased over the past 20 years. Currently, all areas of the country meet the national air quality standard for nitrogen dioxide of 0.053 ppm (100 $\mu g/m^3$), measured as an annual arithmetic mean. However, national emissions (overall production) of nitrogen oxides have actually increased in the past 20 years because of an increase in nitrogen oxide emissions from diesel vehicles.[1] This increase is of concern, because nitrogen oxide emissions contribute to ground-level ozone (smog) and other environmental problems such as acid rain.[1]

Controlled-exposure studies of people with asthma have found that short-term exposures (30 minutes) to nitrogen dioxide at concentrations as low as 0.26 ppm can enhance the allergic response after subsequent challenge with allergens.[61,62] These findings are of concern, because some urban communities that are in compliance with the federal standards for nitrogen dioxide (annual average) may experience substantial short-term peak concentrations (1-hour average) that exceed 0.25 ppm. Confirmation of these studies is needed.

Epidemiologic studies have reported relationships between increased ambient nitrogen dioxide and risks of respiratory tract symptoms[63,64] and asthma exacerbations.[65] As noted previously, children with asthma living in communities with increased levels of air pollution (especially nitrogen dioxide, acid vapor, and particulates) were more likely to have bronchitis symptoms.[47] The same mix of air pollutants was also associated with deficits in lung growth (as measured by lung function tests).[19] These effects were increased in children who spent more time outdoors.

The epidemiologic studies of health effects associated with nitrogen dioxide should be interpreted with caution. Increased levels of ambient nitrogen dioxide may be a marker for exposure to traffic emissions or other combustion-related pollution. An independent role of nitrogen dioxide cannot be clearly established because of the high covariation between ambient nitrogen dioxide and other pollutants. Nonetheless, these studies illustrate that adverse respiratory tract effects are seen in urban areas where traffic is a dominant source of air pollution.

Traffic-Related Pollution

Motor vehicles pollute the air through tailpipe exhaust emissions and fuel evaporation, contributing to carbon monoxide, $PM_{2.5}$, nitrogen oxides, hydrocarbons, other hazardous air pollutants (HAPs), and ozone formation. Motor vehicles represent the principal source of air pollution in many communities, and concentrations of traffic pollutants are greater near major roads.[66] Recently, investigators (primarily in Europe and Japan) have found increased adverse health effects among those living near busy roads.

Studies examining associations between adverse respiratory tract health and traffic have been reviewed.[67] Increased respiratory tract complications in children (eg, wheezing, chronic productive cough, and asthma hospitalizations) have been associated with residence near areas of high traffic density (particularly truck traffic).[68–71] Other investigators have linked various childhood cancers to proximity to traffic.[72–74]

Diesel exhaust, a major source of fine particulates in urban areas, is carcinogenic. Numerous studies have found an association between occupational exposure to diesel exhaust and lung cancer.[75] On the basis of extensive toxicologic and epidemiologic evidence, national and international health authorities, including the EPA and the International Agency for Research on Cancer, have concluded that there is considerable evidence of an association between exposure to diesel exhaust and an increased risk of lung cancer.[76,77] Additionally, fine particles in diesel exhaust may enhance allergic and inflammatory responses to antigen challenge and may facilitate development of new allergies.[78,79] Thus, diesel exhaust exposure may worsen symptoms in those with allergic rhinitis or asthma.

School buses operate in proximity to children, and most of the nation's school bus fleets run on diesel fuel. The EPA and some state agencies are establishing programs to eliminate unnecessary school bus idling and to promote use of cleaner buses to decrease children's exposures to diesel exhaust and the amount of air pollution created by diesel school buses (www.epa.gov/cleanschoolbus). A recent pilot study found that a child riding inside a school bus may be exposed to as much as 4 times the level of diesel exhaust as someone riding in a car.[80] These findings underscore the importance of advocating for school districts to replace diesel buses or retrofit them with pollution-reducing devices and limit school bus idling where children congregate as soon as possible.

Other Air Pollutants

Airborne levels of lead, sulfur dioxide, and carbon monoxide have decreased dramatically because of the implementation of control measures. However, levels of these pollutants may still be high near major sources. For example, high lead levels may be found near metals-processing industries, high sulfur dioxide levels may occur near large industrial facilities (especially coal-fired power plants), and high levels of carbon monoxide may occur in areas with heavy traffic congestion.[1]

In addition to criteria air pollutants, there are numerous other air pollutants produced by motor vehicles, industrial facilities, residential wood combustion, agricultural burning, and other sources that are hazardous to children. More than 50000 chemicals are used commercially, and many are released into the air. For most of these chemicals, data on toxicity are sparse.[81] Some pollutants remain airborne or react in the atmosphere to produce other harmful substances. Other air pollutants deposit into and contaminate land and water. Some toxic air pollutants such as lead, mercury, and dioxins degrade slowly or not at all. These pollutants may bioaccumulate in animals at the top of the food chain, including humans. Children can be exposed to toxic air pollutants through contaminated air, water, soil, and food.[3] One example of a persistent pollutant emitted into ambient air that leads to exposure through another route is mercury, a developmental neurotoxicant.[82] Industrial emissions, especially from coal-fired power plants, are the leading source of environmental mercury. Although the levels of airborne mercury may not be hazardous, mercury deposits into soil and surface waters and ultimately accumulates in fish.[82]

The HAPs, often referred to as "toxic air contaminants" or "air toxics," refer to 188 pollutants and chemical groups known or suspected to cause serious health effects including cancer, birth defects, and respiratory tract and neurologic illness.[3,83] The Clean Air Act directs the EPA to regulate HAPs, which include compounds such as polycyclic aromatic hydrocarbons, acrolein, and benzene from fuel or fuel combustion; solvents such as hexane and toluene; hexavalent chromium from chrome-plating facilities; perchloroethylene from dry-cleaning plants; asbestos; metals (eg, mercury and cadmium); and persistent organic pollutants such as polychlorinated biphenyls. In 2001, diesel exhaust was listed as a mobile-source HAP. Many of these compounds are included in a priority list of 33 HAPs that are of special concern because of their widespread use and potential carcinogenicity and teratogenicity.[81] The priority list and general sources of these compounds are available on the EPA Web site (www.epa.gov/ttn/atw/nata).

Limited monitoring data suggest that concentrations of some HAPs may exceed the goals of the Clean Air Act in many cities.[84] Mobile sources (on- and off-road vehicles) account for approximately half of the emissions[3] but may contribute to 90% of the cancer risk (www.scorecard.org/env-releases/hap/us.tcl). A number of studies assessing health risks have found that estimated levels of some of the HAPs are a potential public health problem in many parts of the United States.[3,84–86] For example, estimated concentrations of benzene, formaldehyde, and 1,3-butadiene may contribute to extra cases of cancer (at least 1 extra case per million population exposed) in more than 90% of the census tracts in the contiguous United States. Additionally, the most recent national cancer-risk assessment for HAPs (1996 data) did not include diesel exhaust in the risk estimates.[3] The health risks may also be underestimated, because there is limited information on toxicity values for many of the HAPs,[87] and the risk models did not consider the potential for increased risk in children. These findings underscore the need for better ways to decrease toxic air emissions and assess exposures and risks.

Air-pollution episodes created by disasters (eg, accidents, volcanoes, forest fires, and acts of terrorism) can also create hazards for children. A discussion of these events and of bioaerosols in ambient air (eg, fungal spores and pollen) is beyond the scope of this

policy statement. Additionally, this statement does not address the hazards of indoor air pollution.

PREVENTION

Public health interventions to improve air quality can improve health at the population level. A decrease in levels of air pollution in former East Germany after reunification was associated with a decrease in parent-reported bronchitis[88] and improved lung function.[89] During the 1996 Summer Olympics in Atlanta, Georgia, extensive programs were implemented to improve mass transportation and decrease anticipated downtown traffic congestion. These programs were successful and were associated with a prolonged decrease in ozone pollution and significantly lower rates of childhood asthma visits during this period.[90] Closure of a steel mill in Utah Valley and resultant reductions in particulate matter were associated with a twofold decrease in hospitalizations for asthma in preschool children.[91,92] Finally, lung function improved in children who moved away from communities with high particulate air pollution, compared with those who remained or moved to communities with comparable particulate air pollution.[93] These studies provide support for continued efforts to decrease air pollution and improve health via decreases in motor vehicle traffic and industrial emissions. Dietary factors may play a role in modulating the effects of air pollution in children. A recent study in Mexico City, Mexico, found that children with asthma given antioxidant supplements were less affected by ozone compared with a control group that did not receive supplementation.[94] Additional studies are needed to explore this issue further.

Air Pollution and the Regulatory Process

The Clean Air Act of 1970 mandated the EPA to establish the National Ambient Air Quality Standards (Table 1). Standards were set for criteria air pollutants because they are common, widespread, and known to be harmful to public health and the environment.[11,12,83,95] The standards are reviewed every 5 years and set to protect public health, including the health of "sensitive" populations such as people with asthma, children, and the elderly. These standards are set without considering the costs of attaining these levels.

The standards for ozone and particulate matter were revised in 1997 on the basis of numerous scientific studies showing that the previous standards were not adequate to ensure health protection. Legal challenges were made by the American Trucking Associations, the US Chamber of Commerce, and other state and local business groups. However, the Supreme Court ultimately supported the EPA and ordered implementation of the standards.[2] Establishing implementation plans will be a lengthy process that will require the coordinated efforts of the EPA, state and local governments, and industry and environmental organizations.

Population exposures to toxic air contaminants may be of substantial public health concern.[84,86] In contrast to criteria pollutants, monitoring of toxic air contaminants is more limited. Exposures are estimated on the basis of reported emissions and may underestimate actual exposures.[87] The EPA is mandated to develop regulations through a lengthy process that first sets standards to control emissions on the basis of best-available technology. After maximum available control technology emission standards are established, the EPA must assess the risk remaining after emission decreases for the source take effect (residual risk).

To date, the EPA has focused primarily on establishing technology-based emission standards,[3] and this has been a slow process for some sources (eg, mobile toxic air contaminants and mercury emissions). Nationwide, emissions of toxic air contaminants have dropped approximately 24% from baseline (1990–1993) because of regulation and voluntary decreases by industry. With the current plans for gradual fleet turnover and implementation of controls for motor vehicles and fuels, the EPA projects that toxic air-contaminant emissions from gasoline-powered and diesel mobile sources will not be decreased to 75% and 90% of baseline (1990–1993) levels, respectively, until the year 2020.[3] However, major decreases could be more rapidly achieved simply from a prompt, wider application of existing technology.

Protecting populations from exposure to the harmful effects of air pollutants will require effective control measures. Industry (eg, coal-burning power plants, refineries, and chemical plants) and motor vehicles (both gasoline- and diesel-powered) are major sources of criteria pollutants and HAPs.[11,12] For example, coal-fired power plants are important sources of nitrogen oxides (precursors of ozone), particulates, and sulfur dioxide and are the largest sources of mercury emission in the United States. Smaller sources such as dry cleaners, auto body shops, and wood-burning fireplaces can also affect air quality locally. Municipal and hospital waste incinerators release toxic air pollutants including mercury, lead, cadmium, and dioxin emissions. Depending on weather conditions and individual physicochemical properties, some pollutants can be carried by air currents to areas many miles from the source.

In numerous cities in the United States, the personal automobile is the single greatest polluter, because emissions from millions of vehicles on the road add up. Despite significant technologic advances that have led to tighter pollution control from vehicles, emissions vary substantially between vehicles, particularly between classes of vehicles, because of differences in fuel-economy standards set by regulatory agencies. For instance, the corporate average fuel-economy standards have less stringent fuel-economy requirements (average: 20.7 miles per gallon) for light-duty trucks, sport utility vehicles, and minivans, compared with passenger cars (average: 27.5 miles per gallon). The former group of vehicles tends to have higher emissions of air pollutants, higher fuel consumption, and higher emissions of greenhouse gases.[96,97] Information on emissions and fuel-economy ratings for recent models and a

guide for choosing clean, fuel-efficient vehicles are available from the EPA Web site (www.epa.gov/greenvehicles/index.htm). The high levels of particulate emissions from diesel-powered buses and trucks must also be addressed. More than 70% of fine particle emissions from traffic are attributable to diesel-powered buses and trucks.

Driving a private car is probably a typical citizen's most "polluting" daily activity, yet in many cases, individuals have few alternative forms of transportation. Thus, urban planning and smart growth are imperative. Urban sprawl affects land use, transportation, and social and economic development and ultimately has important implications for public health.[98] Ways in which individuals can help to decrease air pollution are available at www.epa.gov/air/actions and www.arb.ca.gov/html/brochure/50things.htm.

Air Quality Index

The air quality index (AQI) provides local information on air quality and potential health concerns at the observed (or forecasted) levels of air pollution and can be a useful tool for educating families about local air quality and health.[99] The AQI is reported daily in metropolitan areas, often as part of local weather forecasts on television or radio or in newspapers. The AQI divides air-pollution levels into 6 categories of risk for 5 common pollutants (ozone, PM_{10}, nitrogen dioxide, carbon monoxide, and sulfur dioxide). Each category has a descriptive name reflecting levels of health concern (ranging from good through very hazardous), an associated color, and an advisory statement. Information about air quality in a specific area can be obtained from www.epa.gov/air/urbanair/index.html, www.scorecard.org, or www.weather.com. Although many states and local air districts actively forecast and disseminate health warnings, the challenge is to have people take actions to protect themselves and decrease activities that cause air pollution.

Pediatric Environmental Health[100] from the AAP provides additional information about the outdoor air pollutants and the use of the AQI.

CONCLUSIONS

Ambient air pollution has important and diverse health effects, and infants and children are among the most susceptible. Currently, levels of ozone and particulates remain unhealthful in many parts of the United States, and the current National Ambient Air Quality Standards may not protect the public adequately. There is a compelling need to move forward on efforts to ensure clean air for all.

The assurance of healthy air for children to breathe is beyond the control of an individual pediatrician, and there are no easy solutions. State chapters of the AAP, as well as individual members, can play an important role as advocates for children's environmental health. Areas of involvement might include working with community coalitions in support of strong pollution-control measures and informing local and national representatives and policy makers about the harmful effects of the environment on children's health. Advocates for children's health are needed in discussions about land use and transportation issues. Pediatricians can also advocate for energy-saving (and pollution-minimizing) lifestyles to their patients' families, especially regarding vehicles driven.

In communities with poor air quality, pediatricians can play a role in educating children with asthma or other chronic respiratory tract disease and their families about the harmful effects of air pollution. Patients and families can be counseled on following the AQI to determine when local air-pollution levels pose a health concern. Ozone levels tend to be highest in the afternoon, and it may be possible to decrease children's exposure by scheduling strenuous outdoor activity earlier in the day.

As pediatricians become better informed about local air quality issues in their communities (eg, ozone, nearby industrial facilities, traffic, diesel buses, wood burning, etc), these local concerns can provide a starting point for discussion and education.

Pediatricians who serve as physicians for schools or for team sports should be aware of the health implications of pollution alerts to provide appropriate guidance to school and sports officials, particularly in communities with high levels of ozone.

RECOMMENDATIONS

1. The National Ambient Air Quality Standards are designed to protect the public. To achieve this, the following points should be addressed:
   - The revised standards for ozone and particulate matter adopted by the EPA in 1997 should be promptly implemented.
   - During implementation, the standards should not be weakened in any way that decreases the protection of children's health.
   - Because recent studies suggest that current standards for PM_{10}, $PM_{2.5}$, ozone, and nitrogen dioxide may not be protecting children, the standards should be promptly reviewed and revised.
   - Because the law requires that the most vulnerable groups be protected when setting or revising the air quality standards, the potential effects of air pollution on the fetus, infant, and child should be evaluated, and all standards should include a margin of safety for protection of children.
2. The current measures to protect children from exposures to HAPs are not effective and should be critically reevaluated. The EPA should focus on prompt implementation of the Clean Air Act Amendments of 1990 (Pub L No. 101–549) to decrease HAPs. Additional monitoring for HAPs should be undertaken to allow more accurate characterization of children's exposures to these compounds. Risk assessments for HAPs should be reviewed to ensure that goals are protective of children. Control measures that specifically protect children's health should be implemented.
3. States and local air districts with air quality concerns should actively implement forecasting and

dissemination of health warnings in ways that help people take actions to protect themselves and decrease activities that cause air pollution.
4. Children's exposure to diesel exhaust particles should be decreased. Idling of diesel vehicles in places where children live and congregate should be minimized. Ongoing programs to fund conversion of diesel school bus fleets to cleaner alternative fuels and technologies should be pursued.
5. Industrial emissions of mercury should be decreased.
6. Federal and state governments' policies should encourage reductions in mobile and stationary sources of air pollution, including increased support for mass transit, carpooling, retiring or retrofitting old power plants that do not meet current pollution-control standards, and programs that support marked improvements in fuel emissions of gasoline- and diesel-powered vehicles. Additionally, the development of alternative fuel fleets, low-sulfur diesel, and other "low-emission" strategies (eg, retrofit of existing diesel engines) should be promoted. Before promoting new alternative fuels, these alternative fuel sources should be critically evaluated and determined by governmental authorities to have a good safety profile.
7. The same overall fuel-economy standard should apply to all passenger vehicles. Programs that allow certain passenger vehicles to be exempt from the usual fuel-economy standards should be abolished.
8. City and land-use planning should encourage the design and redevelopment of communities to promote mass transit, carpooling, pedestrian walkways, and bicycle use.
9. Siting of school and child care facilities should include consideration of proximity to roads with heavy traffic and other sources of air pollution. New schools should be located to avoid "hot spots" of localized pollution.

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Ambient Air Pollution: Health Hazards to Children

Pediatrics 2004;114;1699
DOI: 10.1542/peds.2004-2166

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Ambient Air Pollution: Health Hazards to Children

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Sci Total Environ. 2011 Mar 15;409(8):1466-75. doi: 10.1016/j.scitotenv.2010.12.025. Epub 2011 Jan 31.

Emission of trace gases and organic components in smoke particles from a wildfire in a mixed-evergreen forest in Portugal.

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Abstract

On May 2009, both the gas and particulate fractions of smoke from a wildfire in Sever do Vouga, central Portugal, were sampled. Total hydrocarbons and carbon oxides (CO(2) and CO) were measured using automatic analysers with flame ionisation and non-dispersive infrared detectors, respectively. Fine (PM(2.5)) and coarse (PM(2.5-10)) particles from the smoke plume were analysed by a thermal-optical transmission technique to determine the elemental and organic carbon (EC and OC) content. Subsequently, the particle samples were solvent extracted and fractionated by vacuum flash chromatography into different classes of organic compounds. The detailed organic speciation was performed by gas chromatography-mass spectrometry. The CO, CO(2) and total hydrocarbon **emission** factors (g kg(-1) dry fuel) were 170 ± 83, 1485 ± 147, and 9.8 ± 0.90, respectively. It was observed that the particulate matter and OC emissions are significantly enhanced under smouldering fire conditions. The aerosol emissions were dominated by fine particles whose mass was mainly composed of organic constituents, such as degradation products from biopolymers (e.g. levoglucosan from cellulose, methoxyphenols from lignin). The compound classes also included homologous series (n-alkanes, n-alkenes, n-alkanoic acids and n-alkanols), monosaccharide derivatives from cellulose, steroid and terpenoid biomarkers, and polycyclic aromatic hydrocarbons (PAHs). The most abundant PAH was retene. Even carbon number homologs of monoglycerides were identified for the first time as biomarkers in biomass burning aerosols.

PMID: 21277615 DOI: 10.1016/j.scitotenv.2010.12.025

[Indexed for MEDLINE]

Publication type, MeSH terms, Substances

LinkOut - more resources

Exceedances of national air quality standards for particulate matter in Western Australia: sources and health-related impacts

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Ambient air quality in Australia is regulated by the National Environment Protection Measure (NEPM), which sets a maximum 24-hour mean concentration of 50 μg/m^3 for particulate matter less than 10 μm in diameter (PM_{10}) and 25 μg/m^3 for $PM_{2.5}$. Each state and territory is required by the NEPM to annually report all breaches of this standard, including the sources of pollution.[1]

We analysed NEPM reports for Western Australia to identify days during 1 January 2002 – 31 December 2017 on which atmospheric particulate matter levels exceeded air quality standard levels, and classified them according to the most frequently reported sources of pollution: prescribed burns, wildfires, and other (crustal particles such as dust, wood smoke, and indeterminate). During 2008–2013, exceedances caused by smoke from prescribed burns, wildfires, and wood smoke were all recorded by the WA Department of Environment Regulation as "smoke haze". For this period, we therefore applied a random forest algorithm, a machine learning method that uses a random sample of observations for known classifications to predict the classifications for new data.[2] We included the variables month, day of the week, temperature, and pollution level as model predictors.

To estimate background $PM_{2.5}$ level, we obtained historical hourly values for PM_{10} and $PM_{2.5}$ from the WA Department of Water and Environmental Regulation[3] and calculated historical monthly means, excluding days on which particle levels exceeded the air quality standard. We estimated daily $PM_{2.5}$ concentrations attributable to smoke events by subtracting the background $PM_{2.5}$ level from measured daily values.

Applying standard methods for assessing the health impact of air pollution,[4] we estimated the numbers of premature deaths, hospitalisations for cardiovascular and respiratory problems, and emergency department presentations with asthma attributable to elevated $PM_{2.5}$ levels. We used the value of statistical life (VSL)[5] to estimate costs associated with premature mortality. The VSL is based on the willingness to pay for reduced risk of premature mortality, and does not take into account underlying health status, age, or life expectancy of individuals. Deaths associated with acute exposure to increased air pollution are more likely among people at greater risk because of advanced age or chronic illness.[6] We estimated hospital service costs according to the mean cost of each episode of care as reported in the Independent Hospital Pricing Authority national cost data collection report[7] and the Health Policy Analysis emergency care costing report.[8]

We also undertook a sensitivity analysis in which we excluded data for 2008–2013, when exceedances caused by smoke from prescribed burns, wildfires, and wood smoke were all recorded in NEPM reports as "smoke haze". Further details on our methods, including underlying assumptions and limitations, are included in the online Supporting Information.

During 2002–2017, particulate air pollution exceeded the national standard on 271 of 5844 days (4.6%), including 197 days (73%) attributable to prescribed burns or wildfires. We estimated that 41 (95% confidence interval [CI], 15–68) premature deaths, 99 (95% CI, 19–182) hospitalisations for cardiovascular problems and 174 (95% CI, 0–373) for respiratory conditions, and 123 (95% CI, 70–179) emergency department visits with asthma were attributable to elevated $PM_{2.5}$ concentration (Box 1).

Total estimated health costs were $188.8 million (95% CI, $68.1–311.1 million); $97.1 million (51%) was attributable to prescribed burns and $77.7 million (41%) to wildfires. Mean estimated health costs were lower on days affected by smoke from prescribed burns ($703 984; 95% CI, $254 064–$1.2 million) than those affected by wildfire smoke ($1.3 million; 95% CI, $475 000–$2.2 million), although more days were affected by prescribed burns (138) than by wildfires (59). The estimated smoke-related costs of wildfires were highest in 2012 ($24.8 million); in many years, prescribed fires often accounted for most health-related costs, peaking in 2017 ($24.1 million) (Box 2). In our sensitivity analysis excluding the period 2008–2013, the relative costs by source were similar (prescribed burns, 53% [$58.4 million]; wildfires, 38% [$41.6 million]; Supporting Information).

Particulate matter in fire smoke is associated with adverse health outcomes,[9] even at relatively low concentrations.[10]

1 Estimated health burden attributable to elevated $PM_{2.5}$ concentrations, Western Australia, 2002–2017, by particulate matter source

	Estimated number of cases (95% confidence interval)			
Outcome	Prescribed burns	Wildfires	Other	Total
Excess deaths (any cause)	21 (8–35)	17 (6–28)	3 (1–5)	41 (15–68)
Hospital admissions, cardiovascular	51 (10–94)	41 (8–75)	7 (1–13)	99 (19–182)
Hospital admissions, respiratory	89 (0–192)	72 (0–154)	13 (0–27)	174 (0–373)
Emergency department attendances, asthma	63 (36–91)	51 (29–75)	9 (5–13)	123 (70–179)

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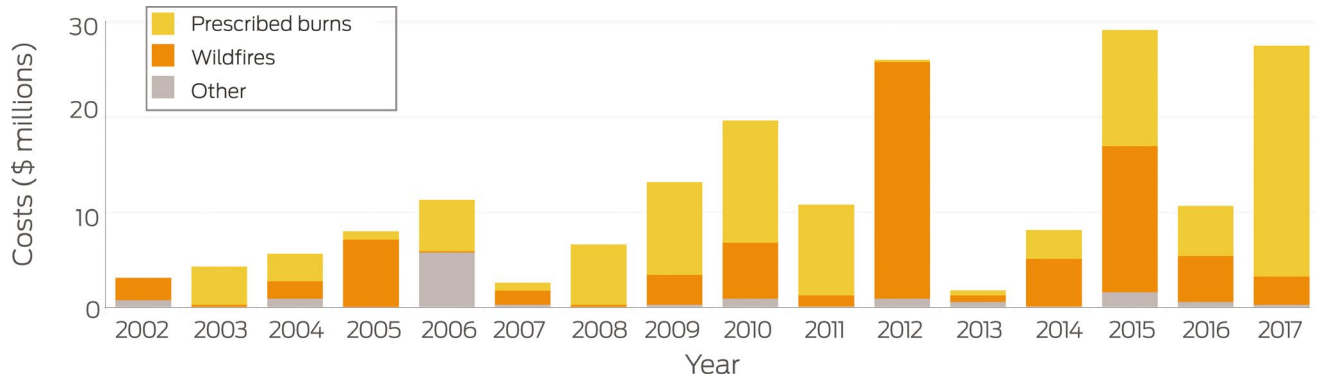
2 Estimated health costs tributable to elevated $PM_{2.5}$ concentrations, Western Australia, 2002–2017, by particulate matter source



Landscape fire smoke was the greatest contributor to excessive atmospheric particulate matter levels in WA during 2002–2017 and was associated with substantial health costs. Our estimates of the health impacts may be conservative, as we included only days when $PM_{2.5}$ concentrations exceeded the national standard, excluding smoky days on which the air quality standard was not breached. Further, our selection of health outcomes did not encompass the total health burden attributable to smoke exposure. Our study highlights the different smoke-related health effects and costs of infrequent severe wildfire and regular prescribed burning. While prescribed burning reduces the risk of wildfire, better understanding and incorporation into control strategies of the full health impacts of each type of fire are needed for sustainable fire management.[11]

Acknowledgements: We thank Arthur Grieco and the Department of Water and Environmental Regulation (Western Australia) for providing air quality monitoring network data. Nicolas Borchers Arriagada is supported by a Tasmania Graduate Research Scholarship from the University of Tasmania, a top-up scholarship from Asthma Australia, and a top-up scholarship from the New South Wales Department of Planning, Industry and Environment through the New South Wales Bushfire Risk Management Research Hub.

Competing interests: No relevant disclosures. ■

Received 16 September 2019, accepted 6 January 2020

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Supporting Information

Additional Supporting Information is included with the online version of this article.

HHS Public Access

Author manuscript

Environ Toxicol Pharmacol. Author manuscript; available in PMC 2018 October 01.

Published in final edited form as:

Environ Toxicol Pharmacol. 2017 October ; 55: 186–195. doi:10.1016/j.etap.2017.08.022.

Wildfire Smoke Exposure and Human Health: Significant Gaps in Research for a Growing Public Health Issue

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Abstract

Understanding the effect of wildfire smoke exposure on human health represents a unique interdisciplinary challenge to the scientific community. Population health studies indicate that wildfire smoke is a risk to human health and increases the healthcare burden of smoke-impacted areas. However, wildfire smoke composition is complex and dynamic, making characterization and modeling difficult. Furthermore, current efforts to study the effect of wildfire smoke are limited by availability of air quality measures and inconsistent air quality reporting among researchers. To help address these issues, we conducted a substantive review of wildfire smoke effects on population health, wildfire smoke exposure in occupational health, and experimental wood smoke exposure. Our goal was to evaluate the current literature on wildfire smoke and highlight important gaps in research. In particular we emphasize long-term health effects of wildfire smoke, recovery following wildfire smoke exposure, and health consequences of exposure in children.

Keywords

Wildfire; smoke; particulate; exposure; health; inhalation; toxicology

1. Introduction

Wildfire and other biomass smoke exposures are increasingly recognized as an important public health issue. While air quality in the United States has generally improved in recent decades due to increased regulatory control, emissions from wildfires have trended upward and are projected to increase as climate change increases the frequency and severity of

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Competing financial interests declaration

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

wildfires (Flannigan et al., 2000; Kinney, 2008). In 2012, wildfires in the US contributed over half of all estimated methane emissions, and 20% of all fine particulate emissions (EPA, 2011). While in Canada, approximately one third of all particulate emissions came from forest fires (Rittmaster et al., 2006). Recent American Thoracic Society reports highlight the growing interest in understanding the impact of climate change on human health, including better understanding how climate change will affect human exposures to respiratory irritants (Pinkerton et al., 2012; Rice et al., 2014). Changes in land cover and in policies concerning fire control and surveillance further complicate future projections of wildfire emissions estimates, however many aspects of climate change are directly related to wildfire risk, including temperature and drought (Flannigan et al., 2013). For example, half of fine particulate emissions were attributed to wildfire during the recent drought in California (California, 2012). Due to climate change and development, wildfire emissions are expected to increase an additional 19–101% in California through 2100 (Hurteau et al., 2014). Increasing numbers of wildfires and acreage burned is also expected to increase across the western United States and Europe (Abatzoglou and Williams, 2016; Lozano et al., 2016).

Wildfire smoke exposure affects millions of people. An estimated 212 million people lived in counties affected by smoke conditions in 2011, many of them far downwind of the actual wildfire burn site (Knowlton, 2013). The smoke from wildfires travels great distances and crosses geographical boundaries, so that states without fires may still be affected by smoke conditions. Epidemiologic evidence to date demonstrates that exposure to smoke from wildfires has direct effects on human health and increases healthcare use. Given that wildfires are growing in frequency and severity, we still know surprisingly little about the specific health effects of wildfire smoke compared to other sources of air pollution. In this review, we summarize the literature on wildfire health effects, human and animal wood smoke exposure studies, and *in vitro* studies. We will discuss the limitations of current studies, and emphasize critical research topics for the future.

2. Wildfire Smoke Composition

Wildfire smoke has a distinct composition compared to other sources of air pollution. The chemical species found in smoke from in a particular wildfire event are determined by many factors unique to the burn site, such as the type of vegetation burned and weather conditions (Urbanski, 2013). Much of the available data on wildfires and human health comes from studies performed on human populations living near burning forests and shrublands in North America, Europe, and Australia. This review focuses on those studies and excludes findings from burning savanna, grasslands, and agricultural burns (Urbanski, 2013).

Wildfire smoke is a major contributor to particulate air pollution. Wildfires produce proportionately more fine (under 2.5 microns) and ultrafine (under 1 micron) particulate, compared to coarse particulate, defined as particles fewer than 10 microns in size (PM_{10}) (Makkonen et al., 2010; Radke et al., 1991). Fine particles generally settle out of the atmosphere more slowly than coarse particles, and therefore disperse farther from the source (Kinney, 2008). Fine and ultrafine particulate is also of particular concern in human health because of its ability to penetrate more deeply into the lung. For this reason $PM_{2.5}$ has been

singled out for special consideration in government documents and guidelines (EPA, 2009). Ultrafine particulate constitutes a substantial proportion of wildfire-generated particulate, although the average size of smoke particulate depends on the intensity of the fire, type of fuel, and whether the fire is smoldering or flaming (Reid et al., 2005).

The particulate found in wildfire smoke is a heterogeneous mixture of chemical species. The chemical make-up of wildfire smoke depends on the type of biomass burned and the conditions for burning. Wet or green vegetation burns differently than dead and dry vegetation, burning hardwood produces different chemical species than burning softwood, and different stages of combustion (open flame vs. smoldering) produce different chemical profiles (Battye and Battye, 2002; Fine et al., 2001; Urbanski et al., 2008; Zhang et al., 2013). Therefore the composition of smoke particulate from natural or accidental wildfires burning in a dry season may differ substantially from prescribed burns performed by firefighters during the wet season (Urbanski, 2013; Zhang et al., 2013). Wildfires also have a long smoldering phase, as wildfire containment strategies focus on extinguishing the flame phase while the smoldering phase is left to burn itself out, sometimes for months after a fire is considered contained (Graham et al., 2004). The smoldering phase of wood burning is associated with higher output of particulate, and can account for a large proportion of the total wildfire air pollutant emissions (Radke et al., 1991; Tian et al., 2008; Urbanski, 2013)

Wildfires tend to occur under conditions that favor high intensity burning of biomass (Urbanski et al., 2008). Experimentally, higher wood combustion temperatures appear to yield more polycyclic aromatic hydrocarbons (PAH) in wood smoke, and in particular yield more oxy-PAH and quinones, which are implicated in oxidative stress (Kocbach Bølling et al., 2009). In real-world scenarios, particulate collected during wildfire events has more oxidative potential than ambient urban particulate due to the presence of more polar organic compounds (Verma et al., 2009). This is consistent with studies suggesting that particles from bushfire and forest fires may generate more free radicals and more oxidative stress in the lung than urban ambient particulate from the same region (Karthikeyan et al., 2006; Williams et al., 2013).

Many of the organic species found in wildfire smoke are unique to biomass combustion compared to fossil fuels, such as levoglucosan and other byproducts of cellulose combustion (Sillanpää et al., 2005). Surprisingly, no studies have been published on the health effects of these biomass smoke-specific species. The majority of wood smoke particulate is composed of organic carbon, compared to a higher level of elemental carbon found in fossil fuel emissions (Kocbach et al., 2006). Production of volatile and semi-volatile organic compounds is also higher in wood smoke compared to fossil fuel emissions, whereas nitrogen oxides and sulfur dioxide levels are lower (Mauderly et al., 2014). The health significance of PAH produced by wildfires is controversial. Studies comparing PAH in laboratory-generated wood smoke with PAH collected from traffic sources generally find higher levels in wood smoke (Bølling et al., 2012; Forchhammer et al., 2012). However, collection of ambient wildfire particulate demonstrates relatively low levels of PAH compared to urban sources, perhaps due to decomposition of PAH species during atmospheric transit (Jalava et al., 2006; Kocbach Bølling et al., 2009).

Beyond the particulate phase, wildfires also produce intermediate species that may participate in local ozone production under certain conditions. Wildfires generate both the nitrogen oxide species and the volatile organic compounds necessary for ozone production, however the relative abundance of each depends on the source of fuel and various other burn conditions. Nitrogen oxides tend to be the limiting factor in wildfire ozone production (Jaffe and Wigder, 2012). However, volatile organic compounds from wildfires may combine with anthropogenic nitrogen oxides in urban areas to generate ozone. Furthermore, much of the nitrogen released by wildfires is sequestered as peroxyacetyl nitrate, a stable nitrogen compound that decomposes to generate ozone downwind of a fire (Jaffe and Wigder, 2012). The presence of these precursor species mean that smoke from wildfires can contribute to local spikes in ozone far from the source, just as the oxidative species in fine particulate matter can travel far downwind of the source. This was the case in Maryland in 2015, when smoke from a large wildfire in Canada caused Maryland to exceed National Ambient Air Quality Standards (NAAQS) (Dreessen et al., 2016). Therefore, populations downwind of a wildfire may be at great risk of exposure to oxidative chemical species (Urbanski et al., 2008). All of these unique aspects of wildfire particulate need to be taken into account when evaluating human studies of wildfire smoke inhalation.

The United States has a growing air quality monitoring network that can provide real-time data for urban pollution events. However, that network is concentrated in heavily populated areas and consequently does not extend to the center of every wild fire. The current state of the art is to use computer simulations to estimate the emissions of individual species from a wildfire based on measurements of the amount and types of fuel burned and historical measurements of similar emissions. Research into wildfire emissions has lead to an extensive series of computer models, which the US Forest Service collects into the BlueSky Framework. In particular, the Fire Emissions Production Simulator (FEPS) model predicts methane, carbon monoxide, and particulate generated from a wildfire events. Expanding these models to include more chemical species would allow for principle component analysis in human studies to better associate the components of wildfire smoke and health outcomes. For instance, many PAH are known to have health effects in animal toxicity studies, but their significance in wildfire smoke exposure in a human population is unknown. Developing a model of wildfire PAH generation, dispersion, and atmospheric chemistry would help elucidate what role, if any, PAH may play in mediating the health effects of wildfire smoke. In the meantime, more consistent reporting of currently available wildfire smoke exposure parameters, including average, peak, and cumulative $PM_{2.5}$ exposure, and average and peak ozone exposure during an event, would allow for better comparison and perhaps help to explain inconsistencies in health effects between studies.

3. Health Effects of Wildfire Smoke

3.1 Population health effects

Exposure to wildfire smoke has been a longstanding concern in public policy. As outlined in Table 1, the major impact of wildfire smoke on the healthcare system comes from patients seeking care for respiratory symptoms. Emergency visits for respiratory symptoms increase in wildfire smoke-affected areas (Dohrenwend et al., 2013; Tham et al., 2009; Viswanathan

et al., 2006). More specifically, patients are more likely to visit the emergency room for asthma, bronchitis, dyspnea, and COPD symptoms (Chew et al., 1995; Delfino et al., 2009; Dennekamp and Abramson, 2011; Dohrenwend et al., 2013; Johnston and Kavanagh, 2002; Rappold et al., 2012; Schranz et al., 2010; Viswanathan et al., 2006). Hospital admissions for respiratory illness also increase during wildfire events, with one study suggesting increased risk for respiratory hospital admissions due to wildfire smoke in excess of what would be expected for a spike in $PM_{2.5}$ from other sources (Liu et al., 2017). In the outpatient setting, visits for respiratory symptoms also increase with wildfire smoke exposure (Henderson et al., 2011; Künzli et al., 2006).

In contrast to respiratory health risks, the data on cardiovascular visits are mixed. Multiple studies report no increase in hospital admissions or emergency department visits for cardiovascular events during wildfire events (Hanigan et al., 2008; Henderson et al., 2011; Johnston et al., 2007; Martin et al., 2013; Morgan et al., 2010; Schranz et al., 2010), with one study even reporting a protective effect from a wildfire in the U.S. (Lee et al., 2009). These findings are supported by data from human and animal wood smoke exposure studies showing no change in the coagulation pathway following exposure (Barregard et al., 2006; Mauderly et al., 2014; Stockfelt et al., 2013). However, other studies have shown increases in cardiac events during wildfires. Two Australian studies have reported an association between wildfire $PM_{2.5}$ and risk of out of hospital cardiac arrest (Dennekamp et al., 2015; Haikerwal et al., 2015). Increased outpatient cardiovascular visits among Native Australian populations following bushfires have also been reported (Johnston et al., 2007). A wildfire in Southern California was associated with an increase in hospital cardiovascular admissions for adults age 45–99 (Delfino et al., 2009). These studies raise the question whether wildfire smoke may be associated with cardiovascular health effects in specific populations or exposure conditions. In the future, discrepancies between studies may be explained by better characterizing the exposure in question.

Data on overall mortality risk during a wildfire event are mixed (Table 1). Too little data is available on the specific risks of wildfire smoke to make a conclusive comparison between wildfire smoke mortality and mortality from other air pollution sources. Many studies have found small associations between increased particulate matter due to wildfires and overall mortality (Johnston et al., 2011; Kollanus et al., 2017; Morgan et al., 2010; Sastry, 2002). However, at least one study suggests that the mortality rate observed during wildfire events is consistent with the increase in mortality from elevated $PM_{2.5}$, regardless of source (Hänninen et al., 2009). Thus it is difficult to determine whether any portion of the increased mortality risk observed during wildfire events is uniquely attributable to wildfire smoke.

Wildfire smoke exposure is associated with increases in specific symptom scores and surrogate markers (Table 2). Sutherland and colleges report a significant increase in COPD symptom scores on days when ambient particulate counts spike due to wildfires (Sutherland et al., 2005). In asthma patients, wildfire smoke exposure is associated with increases in asthma symptoms, and increased corticosteroid and rescue inhaler use (Elliott et al., 2013; Johnston et al., 2006).

It should be noted that in many of these human health studies, researchers have reported particulate under 10 microns in size (PM_{10}) as a measure of the severity of the wildfire. However, as we note in section 2, fine and ultrafine particulate is one of the largest emission components of wildfire smoke (Reid et al., 2005; Urbanski et al., 2008). This particulate size is also of greater concern in human health, as the smaller particles transit more deeply into the respiratory tract (EPA, 2009). It is therefore possible that associations between wildfire smoke and health outcomes are in fact stronger than reported, because PM_{10} is a less relevant marker for health outcomes and/or magnitude of smoke exposure compared to fine or ultrafine particulate matter.

3.2 Studies in firefighters

Studies performed on wildland firefighters are unique among human health effects studies because of their single-subject design. Table 3 provides a summary of parameters measured in firefighters in these studies. Two studies followed firefighters working at prescribed burns, while the rest followed crews working at wildfire sites (Adetona et al., 2011; Slaughter et al., 2004). By far the most striking and consistent finding is the decline in forced expiratory capacity in 1 second (FEV1) following a full season of firefighting compared to preseason values. Of the six studies reporting FEV1 decline, two were able to continue following up with firefighters after the fire season. These studies report an eventual return to baseline FEV1 in the post season, although the recovery period appears to be on the order of months following exposure (Betchley et al., 1997; Gaughan et al., 2008). One study by Swiston *et al.* comparing pre-shift and post-shift values instead of preseason and postseason values, reported no change in FEV1, suggesting that lung function decline is not an acute event but is rather associated with longer smoke exposures (Swiston et al., 2008).

Additionally, the study by Swiston *et al.* reported changes in respiratory symptoms, sputum neutrophils, and serum IL-6 and IL-8. The serum findings are in contrast with human experimental wood smoke exposure studies showing either no change or a decrease in serum IL-6, which may have to do with the extreme nature of the exposure in the Swiston study or the different chemical compositions of wildfire smoke and wood smoke (Ghio et al., 2012; Stockfelt et al., 2012). A recent study by Gaughan *et al.* showed an increase in two urinary markers of oxidative stress, 8-isoprostane and 8-oxo-2′-deoxyguanosine (Gaughan et al., 2014). This is consistent with one study of relatively high human exposure to wood smoke showing increased urinary 8-iso-prostaglandinF2α, while another study with lower concentration smoke exposure found decreased urinary 8-iso-prostaglandinF2α (Barregard et al., 2006; Stockfelt et al., 2013). While these findings need to be confirmed by independent investigators, they suggest that local lung inflammation and oxidative stress are important outcomes of wildfire smoke exposure.

3.3 Experimental animal and in vitro studies

A series of California wildfires in 2008 lead to the only animal studies on wildfire smoke exposure to our knowledge. Two studies instilled coarse or fine wildfire particulate collected during this event into mouse lungs to compare toxicity. Both coarse and fine wildfire particulate induced neutrophilic inflammation and reduced total macrophage counts in bronchoalveolar lavage, but fine particulate matter was the more potent fraction (Wegesser et

al., 2009). Both fractions also induced equal amounts of TNFα, MIP-1α, and CXCL1 in lavage. The fine particulate matter fraction was found to have approximately 50-fold more PAH compared to the coarse, whereas the coarse fraction was found to deplete more antioxidants in the lung lavage compared to fine. Coarse particulate from the wildfire was also compared with coarse urban particulate, and again found to be more oxidative (Wegesser et al., 2010). In a follow-up study, the same group showed that instillation of the coarse wildfire particles lead directly to macrophage death in the lungs, and also increased isoprostane in lavage a mere 30 minutes after instillation, suggesting that the coarse wildfire particulate induced rapid cytotoxicity in macrophages mediated by reactive oxygen species (Williams et al., 2013). These studies are corroborated by *in vitro* studies of RAW 264.7 cells, showing cell death 30 minutes after adding wildfire particulate matter to the culture medium (Franzi et al., 2011).

An *in vitro* study of wildfire particulate from the same event compared gene induction in primary human bronchial epithelial cells (HBE) cultured with wildfire particulate or an equal dose of urban ambient particulate (Nakayama Wong et al., 2011). Many of the genes induced by urban ambient particulate were not induced in controls treated with polymyxin B, indicating that much of the effect of urban particulate was due to LPS. The wildfire particles were found to be low in LPS, and yet they induced a significant fold-change in GM-CSF, IL-1α, IL-1β, CYP1A1, and CYP1B1. Gene induction was reduced when the wildfire particulate matter was treated with deferoxaminemesylate, indicating that iron played an important role in the inflammatory effect on HBE, and perhaps also in the cytotoxicity studies in macrophages discussed above.

Finally, a group of infant nonhuman primates housed outdoors at the California National Primate Research Center were exposed to smoke from the same wildfire event in 2008. A study of these animals performed 3 years later (as juveniles) found that compared to age-matched peers, these animals had reduced lung volumes. Furthermore, stimulation of peripheral blood mononuclear cells collected from the animals produced lower levels of inflammatory cytokines in response to Toll-like receptor ligands compared to age-matched controls that were not exposed to wildfire smoke as infants (Black et al., 2017).

Together, these studies suggest that smoke from California 2008 wildfire event had unique health effects compared to ambient air pollution exposures. However, a limitation of these studies is that only the PAH component was analyzed by Wegesser *et al.* 2010, while data on other chemical components of the wildfire smoke exposure was not included. The lack of chemical composition data can at least be partly attributed to the chance or unexpected occurrence of wildfires and the difficulty in collecting particulate matter samples of these events. More controlled animal studies coupled with more extensive chemical composition analysis are needed to elucidate which chemical components of wildfire smoke mediated health effects.

4. Conclusions

4.1 Improving air quality monitoring, prediction, and reporting

There are many challenges to be overcome in understanding the impact of wildfire smoke on human health, but improving health outcomes during and after wildfire events is a key priority. The risk of wildfire is growing with climate change. As other air pollution sources decline with regulatory efforts and wildfires increase, wildfire smoke will become a more significant source of human exposure to air pollution. In this review, we have discussed current literature regarding the human health impact of wildfire smoke exposure and the major gaps. Future research needs can be grouped into three major areas: improving air quality monitoring, prediction, and reporting.

Inconsistencies in exposure reporting and high variability between wildfire exposures make comparing studies difficult. Future human epidemiologic studies would benefit from more thorough and consistent reporting of air quality measures. $PM_{2.5}$ data is commonly available in many parts of the developed world. Reporting the mean and range of $PM_{2.5}$, both for wildfire periods and during any baseline air quality periods, would allow for more meaningful between-studies comparisons. Ozone measures are also commonly available, but rarely reported in wildfire studies despite examples of peaks in ozone due to wildfire activity. No studies looked at ultrafine particulate produced by wildfires, likely because ultrafine particulate is more difficult to measure and is less commonly reported by monitoring networks. Accuracy and availability of air quality metrics will improve as better wildfire combustion, dispersion, and photochemical reaction models develop. Air quality reporting will also continue to co-evolve with input from human and animal exposure studies identifying the contribution of different chemical species to overall health risk of wildfire smoke. It will be important to develop a scientific consensus on air quality reporting in epidemiologic studies as new air quality measures become available, in order to allow for comparison and meta-analysis of future studies.

4.2 Discovering a mechanistic link between components of wildfire smoke and health effects

Controlled exposures that replicate findings from epidemiologic studies are needed to identify mechanisms underlying the health effects of wildfire smoke exposure. Wildfires produce many species with strong oxidative potential, so the oxidative stress response can be hypothesized to play an important role in mediating the response to wildfire smoke. Many of the components of wildfire smoke particles are capable of inducing the oxidative stress pathway, including free radicals, transition metals, and PAH and quinone species. Animal studies show that wildfire particle deposition in the alveoli leads to increased oxidative stress and macrophage cytotoxicity (Williams et al., 2013). Human experimental exposures to wood smoke show changes in CC16, 8-isoprostane, and lung glutathione that are consistent with an oxidative stress response (Barregard et al., 2006; Sehlstedt et al., 2010; Stockfelt et al., 2012). However, there are no data relating the oxidative stress pattern observed in human wood smoke exposure studies to the effects of real-world wildfire. Furthermore, wildfire smoke components may participate in reactions with anthropogenic air pollution to contribute to spikes in ozone far downwind from the fire site (Jaffe and Wigder, 2012).

Despite this feature of wildfires, and despite the many known detrimental effects of ozone on human health, ozone levels are rarely reported in any studies looking at wildfire and human health outcomes. Future studies are needed to determine whether markers of increased oxidative stress are observed during a wildfire event, or whether a response to oxidative stress underlies the changes in respiratory symptoms and healthcare-seeking behavior seen after a wildfire event. We also look forward to studies to assess the effects of smoke from planned burns on individual health outcomes (O'Keeffe et al., 2016).

4.3 Assessing the long-term health impact of wildfire smoke

A better mechanistic understanding of the body's response to wildfire smoke would lead toward health-relevant biomarkers of exposure, which are needed to track long-term effects of wildfire smoke exposure in the human population. Studies in wildland firefighters showing lung function decrements after a wildfire season suggest that lung function can return to baseline over a long follow-up period (Betchley et al., 1997; Gaughan et al., 2008). However, the cumulative effect of repeated wildfire smoke injury and repair cycles on the lung is completely unknown. Likewise, little is known about the long-term effects on children. Exposure to air pollution during susceptible periods in childhood is associated with an altered growth trajectory in the lung (Gauderman et al., 2004; Gauderman et al., 2002; Gehring et al., 2013; Oftedal et al., 2008; Schultz et al., 2012). Animal models of lung development suggest a strong oxidant exposure in early life changes lung function by altering the pattern and timing of alveolarization and distal airway development (Avdalovic et al., 2012; Fanucchi et al., 2006). This is consistent with animal studies showing altered lung volumes in nonhuman primates exposed to wildfire smoke during infancy (Black et al., 2017). A mechanism explaining the health effects of wildfire smoke would help to predict how wildfire smoke interacts with the developing lung and other long-term health considerations in specific populations.

The broad research gaps identified in this review are heavily interrelated; making progress in any one area is dependent on progress in all. Ultimately, many different approaches can yield important insight into wildfire smoke exposures. A high degree of collaboration between experts in air quality monitoring, smoke exposure modeling, toxicology, physiology, and epidemiology, will be needed to fully understand the true health impacts of wildfire smoke.

Acknowledgments

This work was supported by California Air Resources Board Agreement #10-303, National Institute of Health P51OD011107 and T32HL007013.

Abbreviations

CC16	Clara cell secretory protein 16
COPD	chronic obstructive pulmonary disease
CXCL1	C-X-C motif chemokine ligand 1
CYP1A1	cytochrome P450 family 1 subfamily A member 1

CYP1B1	cytochrome P450 family 1 subfamily B member 1
EPA	Environmental Protection Agency
FEPS	Fire Emissions Production Simulator
FEV1	forced expiratory capacity in 1 second
GM-CSF	granulocyte macrophage colony-stimulating factor
HBE	human bronchial epithelial cells
IL-1α	interleukin-1 alpha
IL-1β	interleukin-1 beta
IL-6	interleukin-6
IL-8	interleukin-8
LPS	lipopolysaccharide
MIP-1α	macrophage inflammatory protein-1 alpha
NAAQS	National Ambient Air Quality Standards
PAH	polycyclic aromatic hydrocarbons
$PM_{2.5}$	particulate matter less than 2.5 μm
PM_{10}	particulate matter less than 10 μm
TNFα	tumor necrosis factor alpha

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Table 1

Healthcare-seeking behavior related to wildfire smoke exposure

Change in healthcare seeking	Age	Population	Exposure assessment, air quality range (during fire event unless otherwise noted), number of safety exceedance days where reported	Reference
Emergency room visits for respiratory symptoms	All ages	Victoria, Australia	Daily PM_{10}, average over study 22–24ug/mˆ3, PM_{10} >50ug/mˆ3 6 days,	(Tham et al., 2009)
	–	San Diego, US	Daily $PM_{2.5}$, $PM_{2.5}$ >65ug/mˆ3 1 day,	(Viswanathan et al., 2006)
	–	San Diego, US	Air quality index monitored, range from “good” to “very unhealthy”	(Dohrenwend et al., 2013)
Asthma visits	Children <12 years	Singapore, China	Daily PM_{10}, effect threshold reported at 158ug/mˆ3,	(Chew et al., 1995)
	All ages	Darwin, Australia	Daily average PM_{10}, range 2–70ug/mˆ3 over entire study period, >50ug/mˆ3 for six days	(Johnston and Kavanagh, 2002)
	–	San Diego, US	Daily $PM_{2.5}$, $PM_{2.5}$ >65ug/mˆ3 1 day	(Viswanathan et al., 2006)
	–	San Diego, US	Air quality index monitored, range from “good” to “very unhealthy,	(Dohrenwend et al., 2013)
	Ages 0–4, 20–99	Southern California, US	Daily $PM_{2.5}$ monitored, 24hr average 42–76ug/mˆ3,	(Delfino et al., 2009)
	Adults >18 years	North Carolina, US	Daily maximum $PM_{2.5}$, range 4–129ug/mˆ3 (peat wildfire)	(Rappold et al., 2012)
	All ages (females>males)	Northern California, US	Daily $PM_{2.5}$	(Reid et al., 2016)
	Age >20 (females)	Victoria, Australia	Daily $PM_{2.5}$, max >200ug/mˆ3	(Haikerwal et al., 2016)
	All ages, greatest effect 65+	Colorado, US	1 h max $PM_{2.5}$, 24 hour mean	(Alman et al., 2016)
	Age >65	New Mexico, US	Daily $PM_{2.5}$, 10–70ug/mˆ3	(Resnick et al., 2013)
Bronchitis visits	All ages	Southern California, US	Daily $PM_{2.5}$ monitored, 24hr average 42–76ug/mˆ3	(Delfino et al., 2009)
Chief complaint dyspnea	–	San Diego, US	Air quality index monitored, range from “good” to “very unhealthy”,	(Dohrenwend et al., 2013)
	–	San Diego, US	Daily $PM_{2.5}$, range 12–80ug/mˆ3, $PM_{2.5}$ >35ug/mˆ3 -3 days	(Schranz et al., 2010)

Change in healthcare seeking	Age	Population	Exposure assessment, air quality range (during fire event unless otherwise noted), number of safety exceedance days where reported	Reference
COPD symptom exacerbation	Age 20–64	Southern California, US	Daily $PM_{2.5}$ monitored, 24hr average 42–76ug/m^3,	(Delfino et al., 2009)
	–	Colorado, US	1 h max $PM_{2.5}$, 24 hour mean	(Alman et al., 2016)
Pneumonia	All ages	Southern California, US	Daily $PM_{2.5}$ monitored, 24hr average 42–76ug/m^3	(Delfino et al., 2009)
Hospital admissions				
Respiratory admissions	Adult, children	Brisbane, Australia	Daily PM_{10}, range 7.5–60ug/m^3, PM_{10} >50ug/m^3 2 days,	(Chen et al., 2006)
	–	Southeastern British Columbia, Canada	Daily PM_{10}, average 44+/−129	(Henderson et al., 2011)
	–	San Diego, US	Daily $PM_{2.5}$, range 12–80ug/m^3, $PM_{2.5}$ >35ug/m^3 -3 days	(Schranz et al., 2010)
	–	Darwin, Australia	Daily estimated PM_{10}, mean 21.2ug/m^3	(Hanigan et al., 2008)
	Age>65	Western US	Daily estimated $PM_{2.5}$ >37ug/m^3	(Liu et al., 2017)
Respiratory infection admissions	Indigenous Austrralians	Darwin, Australia	Daily estimated PM_{10}, mean 21.2ug/m^3	(Hanigan et al., 2008)
Asthma admissions	Adults 15–64 years	Sydney, Australia	Daily PM_{10}, range 43–117ug/m^3,	(Morgan et al., 2010)
	–	Darwin, Australia	Daily PM_{10}, range of 6.4–70ug/m^3 over entire study,	(Johnston et al., 2007)
	–	Sydney, Newcastle, Wollongong, Australia	Daily PM_{10}/Daily $PM_{2.5}$, range PM_{10} 47–281ug/m^3/range $PM_{2.5}$ 25–112ug/m^3,	(Martin et al., 2013)
	Age >20	Northern California, US	Daily $PM_{2.5}$	(Reid et al., 2016)
COPD admissions	–	Darwin, Australia	Daily PM_{10}, range of 6.4–70ug/m^3 over entire study,	(Johnston et al., 2007)
	–	Sydney, Newcastle, Wollongong, Australia	Daily PM_{10}/Daily $PM_{2.5}$, range PM_{10} 47–281ug/m^3/range $PM_{2.5}$ 25–112ug/m^3,	(Martin et al., 2013)
	>65 years	Sydney, Australia	Daily PM_{10}, range 43–117ug/m^3,	(Morgan et al., 2010)
	Age 20–64	Northern California, US	Daily $PM_{2.5}$	(Reid et al., 2016)
Cardiovascular admissions	Adults age 45–99	Southern California, US	Daily $PM_{2.5}$ monitored, 24hr average 42–76ug/m^3,	(Delfino et al., 2009)

Change in healthcare seeking	Age	Population	Exposure assessment, air quality range (during fire event unless otherwise noted), number of safety exceedance days where reported	Reference
	Age >65 years	Victoria, Australia	Daily $PM_{2.5}$, mean 15ug/mˆ3, max 163ug/mˆ3	(Haikerwal et al., 2015)
	Men >35	Melbourne, Australia	Hourly $PM_{2.5}$, mean 32.4ug/mˆ3, max 247ug/mˆ3	(Dennekamp et al., 2015)
Outpatient visits for respiratory symptoms	Adult, children	Southeastern British Columbia, Canada	PM_{10} 24 hr average 29ug/mˆ3,	(Henderson et al., 2011)
	Elementary and high school children	Southern California, US	Daily PM_{10}, 5 day average PM_{10} 30–252ug/mˆ3	(Künzli et al., 2006)
Upper respiratory tract illness visits	–	Singapore, China	Monthly PM_{10} average 60–100ug/mˆ3,	(Emmanuel, 2000)
Asthma visits	–	Singapore, China	Monthly PM_{10} average 60–100ug/mˆ3,	(Emmanuel, 2000)
		Native American reservation population, California, US	Weekly average PM_{10}, range 13–363ug/mˆ3, PM_{10} >150ug/mˆ3 12 days	(Lee et al., 2009)
Rhinitis visits	–	Singapore, China	Monthly PM_{10} average 60–100ug/mˆ3,	(Emmanuel, 2000)
Cardiovascular visits	–	Darwin, Australia	Daily PM_{10}, range of 6.4–70ug/mˆ3 over entire study,	(Johnston et al., 2007)
	Adults >18 years	Congestive heart failure patients	Daily maximum $PM_{2.5}$, range 4–129ug/mˆ3 (peat wildfire)	(Rappold et al., 2012)
Mortality	–	Southern Finland	Daily $PM_{2.5}$, increased 15.7ug/mˆ3 over normal levels for 2 weeks	(Hänninen et al., 2009)
	–	Sydney, Australia	Daily average PM_{10}, increased over 99 percentile (47.3ug/mˆ3)	(Johnston et al., 2011)
	–	27 European countries	Modeled daily $PM_{2.5}$ based on satellite data	(Kollanus et al., 2017)
	–	Kuala Lumpur, Malaysia	Daily PM_{10} levels above 210ug/mˆ3	(Sastry, 2002)
Respiratory mortality	>75 years age	Athens, Greece	Forest fire size by area burnt,	(Analitis et al., 2012)
Cardiovascular mortality	<75 years age	Athens, Greece	Forest fire size by area burnt,	(Analitis et al., 2012)
No change in healthcare-seeking	**Age**	**Population**	**Exposure assessment, air quality range (during fire event unless otherwise noted), number of safety exceedance days where reported**	**Reference**
Emergency room visits	–	San Diego, US	Daily $PM_{2.5}$, range 12–80ug/mˆ3, $PM_{2.5}$ >35ug/mˆ3 -3 days	(Schranz et al., 2010)

Change in healthcare seeking	Age	Population	Exposure assessment, air quality range (during fire event unless otherwise noted), number of safety exceedance days where reported	Reference
Asthma visits	–	Western Sydney, Australia	Hourly PM_{10},	(Smith et al., 1996)
	–	San Diego, US	Daily $PM_{2.5}$, range 12–80ug/mˆ3, $PM_{2.5}$ >35ug/mˆ3 -3 days	(Schranz et al., 2010)
	Age 5–18	Southern California, US	Daily $PM_{2.5}$ monitored, 24hr average 42–76ug/mˆ3,	(Delfino et al., 2009)
COPD symptom exacerbation	–	San Diego, US	Daily $PM_{2.5}$, range 12–80ug/mˆ3, $PM_{2.5}$ >35ug/mˆ3 -3 days	(Schranz et al., 2010)
Cardiovascular visits	–	San Diego, US	Daily $PM_{2.5}$, range 12–80ug/mˆ3, $PM_{2.5}$ >35ug/mˆ3 3 days	(Schranz et al., 2010)
	–	Southeastern British Columbia, Canada	Daily PM_{10}, average 44+/−129	(Henderson et al., 2011)
	–	Darwin, Australia	Daily estimated PM_{10}, mean 21.2ug/mˆ3	(Hanigan et al., 2008)
	–	Colorado, US	1 h max $PM_{2.5}$, 24 hour mean	(Alman et al., 2016)
Hospital admissions				
Cardiovascular admissions	–	Sydney, Australia	Daily PM_{10}, range 43–117ug/mˆ3	(Morgan et al., 2010)
	–	Darwin, Australia	Daily PM_{10}, range of 6.4–70ug/mˆ3 over entire study,	(Johnston et al., 2007)
	–	Sydney, Newcastle, Wollongong, Australia	Daily PM_{10}/Daily $PM_{2.5}$, range PM_{10} 47–281ug/mˆ3/range $PM_{2.5}$ 25–112ug/mˆ3,	(Martin et al., 2013)
	Age >65	Western US	Daily $PM_{2.5}$ >20ug/mˆ3	(Liu et al., 2017)
	–	Northern California, US	Daily average $PM_{2.5}$	(Reid et al., 2016)
Protective effect observed	–	Native American reservation population, California, US	Weekly average PM_{10}, range 13–363ug/mˆ3, PM_{10} >150ug/mˆ3 12 days,	(Lee et al., 2009)
Outpatient visits				
Asthma (Protective effect)	Children 1–14 years	Sydney, Australia	Daily PM_{10}, range 43–117ug/mˆ3	(Morgan et al., 2010)
Mortality	–	Singapore, China	Monthly PM_{10} average 60–100ug/mˆ3,	(Emmanuel, 2000)
	–	Sydney, Australia	Daily PM_{10}, range 43–117ug/mˆ3	(Morgan et al., 2010)

Table 2

Markers and symptoms evaluated following human exposure to wildfire smoke

Change in symptom or marker	**Population**	**Location**	**Exposure assessment, PM range or average (during fire event unless otherwise noted), exceedance days where reported**	**Reference**
COPD exacerbation, increase in COPD symptom score	COPD patients	Denver, Colorado, US	Daily average PM_{10}, 89.4ug/mˆ3/Daily average $PM_{2.5}$, 63.1ug/mˆ3	(Sutherland et al., 2005)
Asthma exacerbation				
Increase in asthma symptoms	Adults and children with asthma	Darwin, Australia	Daily average PM_{10} 2.6–43.3ug/mˆ3/Daily average $PM_{2.5}$ 2.2–36.5ug/mˆ3 over entire study, PM_{10} >50ug/mˆ3 1 day, $PM_{2.5}$ > 25ug/mˆ3 5 days	(Johnston et al., 2006)
Commencing asthma medication use or increase in medication dispensed	Adults and children with asthma	Darwin, Australia	Daily average PM_{10} 2.6–43.3ug/mˆ3/Daily average $PM_{2.5}$ 2.2–36.5ug/mˆ3 over entire study, PM_{10} >50ug/mˆ3 1 day, $PM_{2.5}$ > 25ug/mˆ3 5 days	(Johnston et al., 2006)
	Pharmaceutical dispensation database	British Columbia, Canada	Daily average $PM_{2.5}$ 4.2–7.4ug/mˆ3	(Elliott et al., 2013)
	Obese asthmatic children	San Diego, California, US	levels not reported, 2–3 fold increase in particulate compared to before fire	(Tse et al., 2015)
Systemic inflammation				
Band cells in peripheral blood	National service men	Singapore	Daily average PM_{10}, range 47–216ug/mˆ3	(Tan et al., 2000)
Respiratory symptoms				
Upper respiratory (cold, rhinitis, congestion)	Elementary and high school children	Southern California, US	Daily PM_{10}, 5 day average PM_{10} 30–252ug/mˆ3	(Künzli et al., 2006)
Lower respiratory (cough, wheeze, chest tightness)	Nonasthmatic 16–19 year olds,	Southern California, US	Not reported,	(Mirabelli et al., 2009)
	Elementary and high school children	Southern California, US	Daily PM_{10}, 5 day average PM_{10} 30–252ug/mˆ3	(Künzli et al., 2006)
Lower birth weight	Full-term births	Southern California, US	Daily PM_{10}, mothers exposed to >40ug/mˆ3 average daily PM_{10} during fire classified as “exposed”	(Holstius et al., 2012)
No change in symptom or marker	**Population**	**Location**	**Exposure assessment, PM range or average (during fire event unless otherwise**	**Reference**

Change in symptom or marker	**Population**	**Location**	**Exposure assessment, PM range or average (during fire event unless otherwise noted), exceedance days where reported**	**Reference**
			noted), exceedance days where reported	
Asthma exacerbation				
No change in peak expiratory flow rate	Children with wheeze	Sydney, Australia	Daily PM_{10}, ~40–110ug/m3 for 8 days	(Jalaludin et al., 2000)
Systemic inflammation				
No change in total WBC counts, lymphocytes, monocyte, granulocyte counts	National service men	Singapore	Daily average PM_{10}, range 47–216ug/mˆ3	(Tan et al., 2000)
Lung function				
No change in FEV1 or FVC	National service men	Singapore	Daily average PM_{10}, range 47–216ug/mˆ3	(Tan et al., 2000)

Table 3

Studies of wildfire smoke exposure in wildland firefighters

Change in markers	**Exposure measurement**	**Reference**
Lung function		
Decline in FEV1	None, comparison of values before/after fire season	(Liu et al., 1992)
	None, comparison of values before/after fire	(Gaughan et al., 2008)
	None, comparison of values before/after fire season	(Adetona et al., 2011)
	Personal CO, NO, VOC measurement, comparison of values before/after fire season	(Miranda et al., 2012)
	None, comparison of values before/after fire season	(Jacquin et al., 2011)
	None, comparison of values before/after work shift and before/after fire season	(Betchley et al., 1997)
	$PM_{3.5}$ range 235–1317ug/m^3, average 24 hour $PM_{3.5}$ 882ug/m^3	(Slaughter et al., 2004)
Upper respiratory symptom score	None, comparison of values before/after fire season	(Gaughan et al., 2008)
Lower respiratory symptom score	None, comparison of values before/after fire season	(Gaughan et al., 2008)
Increase in airways hyperresponsiveness	None, comparison of values before/after fire season	(Liu et al., 1992)
Systemic inflammation		
increase in serum IL-6	Measured before and after 8h shift, estimated PM3.5 exposure >1000ug/mˆ3 for at least 6h	(Swiston et al., 2008)
increase in serum IL-8	Measured before and after 8h shift, estimated PM3.5 exposure >1000ug/mˆ3 for at least 6h	(Swiston et al., 2008)
increase in urinary 8-isoprostane	None, comparison of values before and after fire & correlation with urinary levoglucosan	(Gaughan et al., 2014)
increase in urinary 8-OHdG	None, comparison of values before and after fire & correlation with urinary levoglucosan	(Gaughan et al., 2014)
Blood cell counts		
Increase in band cells	Measured before and after 8h shift, estimated PM3.5 exposure >1000ug/mˆ3 for at least 6h	(Swiston et al., 2008)
Sputum		
Increase in neutrophils	Measured before and after 8h shift, estimated PM3.5 exposure >1000ug/mˆ3 for at least 6h	(Swiston et al., 2008)
Increase in macrophage inclusions	Measured before and after 8h shift, estimated PM3.5 exposure >1000ug/mˆ3 for at least 6h	(Swiston et al., 2008)
Exhaled airway markers		
Decrease in exhaled NO	Personal CO, NO, VOC measurement, comparison of values before/after fire season	(Miranda et al., 2012)
No change in markers	**Exposure measurement**	**Reference**
Lung function		
FEV1	Measured before and after 8h shift, estimated PM3.5 exposure >1000ug/mˆ3 for at least 6h	(Swiston et al., 2008)
Systemic inflammation		
serum GM-CSF	Measured before and after 8h shift, estimated PM3.5 exposure >1000ug/mˆ3 for at least 6h	(Swiston et al., 2008)
CRP	Measured before and after 8h shift, estimated PM3.5 exposure >1000ug/mˆ3 for at least 6h	(Swiston et al., 2008)

Change in markers	Exposure measurement	Reference
Blood cell counts		
WBC and PMN increases	Measured before and after 8h shift, estimated PM3.5	(Swiston et al., 2008)
not significantly different from strenuous exercise controls	exposure >1000ug/mˆ3 for at least 6h	

Air Pollution and the Labor Market: Evidence from Wildfire Smoke*

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June 2019

Abstract

We estimate labor market responses to transient air pollution events using a novel linkage of satellite images of wildfire smoke plumes to pollution monitor data and labor market outcomes in the United States. Smoke exposure reduces earnings in both the year of exposure and the following year, lowers labor force participation, and increases Social Security claiming and payments. With an average of 17.7 days of annual smoke exposure per person, earnings losses sum to 1.26 percent of annual labor income. We estimate that the welfare cost of these lost earnings is higher than the mortality cost of wildfire smoke.

JEL Classification: I10, J21, Q51, Q52, Q53, Q54
Keywords: air pollution, labor supply, mortality, wildfires

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Pollution imposes large costs on human well-being. Air pollution exposure increases infant and elderly mortality (Chay and Greenstone, 2003; Jayachandran, 2009; Deryugina et al., 2019) and reduces long-run health and future income among those exposed in utero and infancy (Sanders, 2012; Chen et al., 2013; Isen, Rossin-Slater and Walker, 2017). While infant and elderly impacts are thought to constitute the vast majority of the welfare costs of poor air quality (U.S. Environmental Protection Agency, 2011; OECD, 2016), air pollution also negatively affects the broader adult population, for example, by reducing labor supply and productivity (Graff Zivin and Neidell, 2012; Hanna and Oliva, 2015). It remains largely unknown, however, whether responses in the adult population constitute an important share of the overall costs of air pollution. These effects, if important in the aggregate, may significantly alter our understanding of how pollution affects human welfare and the design of efficient pollution-abatement policies.

This paper examines the importance of air pollution in the determination of national, annual labor income in the United States. A key challenge involved in measuring the causal effect of air pollution on countrywide labor market outcomes is finding geographically widespread fluctuations in pollution that are not themselves driven by economic factors, such as regulations, that directly impact economic activity. To sidestep the joint determination of air quality and economic activity, our analysis leverages nationwide variation in U.S. air quality induced by wildfire smoke. Wildfires account for about 20 percent of the fine particulate matter emitted in the United States (U.S. Environmental Protection Agency, 2014), and wind can transport wildfire smoke for thousands of miles, generating plausibly exogenous air pollution events that are both geographically dispersed and widespread (Langmann et al., 2009).

Our analysis relies on a novel linkage of high-resolution satellite remote sensing data on wildfire smoke plumes in the United States with ground pollution monitors and labor market data over the period 2006-2015.[1] We exploit year-over-year variation in wildfire smoke exposure in a given region to estimate the medium- to long-run impacts of air pollution events. In this way, our approach is most similar to that of Deschênes and Greenstone (2011), who use annual variation in

[1] We use wildfire smoke exposure data developed by Miller, Molitor and Zou (2017) and adapt it to fit the unit of analysis for the labor market data.

daily weather to estimate the effects of temperature on annual mortality. Moreover, we estimate and compare the effects of the wildfire smoke events on both labor market and mortality outcomes. This comparison benchmarks the importance of labor market responses in the costs of air pollution for social welfare, following a strategy that is similar to that of Deschênes, Greenstone and Shapiro (2017), who use mortality effects to benchmark the welfare effects of defensive investments.

Several features of wildfire smoke combine to create an attractive natural experiment for studying the effects of air quality on labor market outcomes. Wildfire-related smoke events occur frequently throughout the United States: the average person in our sample experienced about 17.7 days of smoke exposure per year, and nearly every U.S. county was exposed to wildfire smoke in the sample period. Drifting wildfire smoke plumes induce sharp air pollution shocks that typically last a few days and have magnitudes typical of normal daily variation in U.S. air quality. At the daily level, we estimate that exposing a county to an additional day of smoke increases concentrations of particulate matter smaller than 2.5 microns ($PM_{2.5}$) by an average of 2.2 $\mu g/m^3$, or one-third of the daily standard deviation.[2] At the annual level, we estimate that increasing smoke exposure by an additional day raises a county's annual average $PM_{2.5}$ concentration by 0.019 $\mu g/m^3$, or about 0.9 percent of the annual standard deviation. The regularity and broad geographic coverage of wildfire smoke events underscore the importance of understanding the impact of these shocks on human welfare and suggest that the results are informative of the effects of short-term fluctuations in air pollution more generally.

Three primary results emerge from the analysis. First, we find that wildfire smoke exposure leads to statistically and economically significant losses in annual labor income. Specifically, each day of smoke exposure over the year causes a roughly linear reduction in labor income of 0.07 percent in the year of exposure.[3] We also find evidence of income losses in the year following

[2]Biomass burning may be more harmful to human health than car exhaust and most sources of industrial pollution, because it contains a higher share of volatile organic compounds (VOCs), a particularly harmful class of particulate matter. However, our mortality estimates are in line with previous effects estimated for particulate matter exposure, suggesting that wildfire smoke is comparable to other sources of pollution. We discuss this further in Section 1, and estimate the full range of EPA-monitored pollutant responses in Section 3.

[3]Our primary estimates exclude county-years where wildfires burned. The expected direct effect of wildfires on labor market outcomes is ambiguous. Wildfires may destroy businesses and reduce economic activity while burning, but firefighting and rebuilding may temporarily increase incomes. In robustness checks, our estimates of smoke effects

exposure, indicating lasting reductions in health or wages. After adjusting for the concentration of smoke days in the middle and second half of the year, the magnitude of losses in the year following exposure are around one-half as large as the effects in the year of exposure. Because we measure income at the annual level and allow for lagged effects of smoke exposure, our estimates capture medium- to long-run effects of transient pollution exposure, about which previous research has produced little evidence. In particular, these estimates incorporate possible intertemporal substitution in work effort and lasting effects of illness which show up as changes in health capital. As a placebo test, we find no effect of smoke on income in years prior to the exposure. We calculate that wildfire smoke reduced U.S. labor income by 1.26 percent each year in our sample, or $93 billion in 2018 dollars. Summing the smoke effects in the year of and year after exposure produce losses of 1.98 percent, or about $147 billion in 2018 dollars.

Second, we show that smoke can have lasting effects on the labor market through changes in employment. We estimate that an additional day of smoke exposure reduces employment by 302 per million individuals aged 16 and older, which is approximately a 0.046 percent reduction. Under reasonable assumptions, the effects associated with extensive margin responses can explain half of the overall decrease in income due to smoke exposure. Proportional effects are largest among older workers, suggesting that greater vulnerability to air pollution may amplify the effects in the labor market.[4] We also find evidence that wildfire smoke exposure increases the receipt of Social Security income. To the best of our knowledge, these results provide the first evidence linking air pollution to extensive margin and retirement responses and indicate a channel through which short-run changes in air quality may have lasting impacts on the labor market.

Third, we find the labor market cost of wildfire smoke in the United States to be substantially higher than the mortality cost. Using a similar strategy as employed in our analysis of labor market effects, we find mortality responses are concentrated among individuals aged 60 and older and

change little when we include counties experiencing wildfire burn.

[4]Medical and public health studies find that vulnerability to respiratory and circulatory illness rises with age, suggesting older workers may be particularly responsive to air pollution (e.g., Bentayeb et al., 2012; Schlenker and Walker, 2016). For examples of the mortality literature, see Dockery et al. (1993) and Pope et al. (2009). See Chan and Stevens (2001) for evidence related to job search at older ages.

within six months of smoke exposure. Specifically, each day of exposure to smoke leads to 9.3 additional deaths per million residents. Using an approach that values each lost life year at $100,000, we estimate that premature mortality due to wildfire smoke imposes costs of $15.6 billion per year. Using a model of health and labor supply, we analyze the welfare implications of the $86 billion in lost labor market earnings each year due to wildfire smoke.Our approach indicates that the welfare costs of lost labor income are more than four times as high as the costs arising from mortality, and may be many times higher. These results contrast with previous estimates that find that the costs of premature death represent over 80 percent of the total welfare costs of air pollution.[5] In the absence of quasi-experimental variation, however, previous estimates of the aggregate labor market costs of air pollution have relied on strong assumptions in lieu of direct estimation.[6] Our results overcome this limitation and provide a comparison of mortality and labor market effects that arise from the same quasi-experimental variation in pollution exposure.

In addition to providing the first empirical evidence on the aggregate effects and relative importance of labor market channels in the evaluation of the costs of air pollution, our paper makes several other contributions to the literature. First, for pollution-abatement policy, the pollution variation we study consists primarily of variation in levels that do not exceed regulatory standards set by the Environmental Protection Agency. Nevertheless, our findings indicate that such pollution may significantly reduce labor market earnings. Failure to consider labor market costs may therefore lead to inefficient pollution standards and regulations. Second, our finding that reductions in air pollution can increase labor income through improvements in health or increased productivity indicates the possibility of a "double dividend": reducing air pollution to align the private marginal cost of abatement with social marginal benefits can improve population health and productivity and can also generate additional income tax revenue that could be used to lower tax rates and the distor-

[5] See OECD (2016) for a summary of the literature. The World Bank (2016) estimates that labor market costs comprise less than 5 percent of the total welfare costs of air pollution worldwide and in North America. The U.S. Environmental Protection Agency (2011) estimates that 85 percent of the benefits of the Clean Air Act come from reductions in premature mortality.

[6] The EPA's usual method measures lost earnings by multiplying the dose-response function, estimated from medical records or taken from previous literature, and associated number of days lost due to a given illness, usually taken from survey evidence. For example, this method would involve gathering data on the increase in asthma attacks on smoky days, and then multiplying this by the number of days of work lost due to a typical asthma attack.

tions they impose (Williams III, 2003). The national representation of our estimated income effect from pollution reductions enhances the relevance of our estimates for evaluating the magnitude of revenue effects from other air pollution regulations.

Moreover, our findings shed light on how changes in health can lead to changes in employment, earnings, and retirement behavior. The propagation of short-run labor market shocks, especially those that generate job loss, are of long-standing interest in labor and macroeconomics (Jacobson, LaLonde and Sullivan, 1993; Neal, 1995; Jarosch, 2015). Our findings that pollution shocks translate into reductions in labor income and labor force participation add to a relatively small body of literature that documents the lasting impacts of changes in health on labor supply using quasi-experimental evidence (Coile, 2004; Stephens Jr and Toohey, 2018). We find significantly larger responses in urban areas, and larger point estimates in poorer areas with more black residents, suggesting that high rates of hourly work and narrow coverage under the Family Medical Leave Act may play a role in transmitting short-run shocks to lasting changes in income. In light of on-going population aging, the link between air pollution and premature retirement is of increasing policy relevance.

Finally, our research extends the growing body of literature on the economics and social costs of natural disasters to the study of wildfire. Like the economic losses caused by other natural disasters, the damage from wildfires can be mitigated or exacerbated by policy. Our findings suggest, however, that, unlike the losses caused by most other natural disasters, the damage from wildfire arises largely from externalities, as the costs may be concentrated in locations far from the fires themselves. These social costs should be considered alongside traditional considerations of damage to property, natural resources, and the costs of firefighting, and may significantly alter optimal policy in local land use and fire management.[7] Climate change has the potential to multiply the damage done by wildfires, as the National Research Council estimates that each degree Celsius increase in global temperature may lead to a quadrupling of acreage burned.[8] More broadly, these

[7] Kochi et al. (2010) surveys the literature, finding only six studies that have quantified the economic cost of wildfire smoke, and none that include economic costs manifested through the labor market.

[8] Climate change is projected to increase temperatures and reduce precipitation, leading to longer and more intense fire seasons; for example, every one-degree-Celsius increase in global temperature is projected to quadruple acreage

findings contribute to a growing body of literature on trans-boundary pollution with international implications, as an important share of wildfire smoke in the U.S. originates in Canada or Mexico (Lipscomb and Mobarak, 2016; Monogan, Konisky and Woods, 2017; Yang and Chou, 2017).

Section 1 provides background on wildfire and a model of the links between air pollution and labor market outcomes. Section 2 describes our data, and Section 3 explains our empirical strategy. Section 4 reports our main results on earnings, and labor force participation and retirement. Section 5 discusses the welfare costs of wildfire smoke exposure, with particular attention paid to the comparison of labor market impacts to mortality costs. Section 6 concludes.

1 Background and Conceptual Framework

How do transient air pollution events, such as wildfire smoke, affect labor market earnings? A well-developed body of literature in biomedical sciences, public health, and economics demonstrates negative effects of air pollution exposure on short-run performance and health outcomes.[9] Wildfire smoke, like other air pollution, contains particulate matter that enters the lungs and can pass into the bloodstream. This fine particulate matter carries with it numerous pollutants, such as ozone, carbon monoxide, atmospheric mercury, and a range of volatile organic compounds (VOCs). Exposure to these pollutants diminishes human health and performance. Inert particulate matter may also be harmful to human health. Health effects of exposure can have direct consequences on labor supply, leading to missed work days and reduced productivity. The EPA and other large research

burned by wildfires. See National Research Council (2011) for more details on this projection, and Moritz et al. (2012) for more on modeling of climate-and-wildfire linkages. Consistent with predictions generated by these models, recent fire seasons have set records in number of fires, acreage burned, and property damage.

[9]The literature has made important progress in documenting the effects of air pollution on outcomes such as worker productivity, usually in narrow settings chosen to minimize the confounding effects of changes in economic activity. See Hanna and Oliva (2015) and Aragon, Miranda and Oliva (2016) for air pollution effects on hours worked; Hausman, Ostro and Wise (1984), Hansen and Selte (2000) and Holub, Hospido and Wagner (2016) for sick leave; Graff Zivin and Neidell (2012) and Chang et al. (2014) for the productivity of agricultural workers; He, Liu and Salvo (2018) and Adhvaryu, Kala and Nyshadham (2016) for the productivity of Chinese and Indian manufacturers, respectively; Chang et al. (2016) for the productivity of indoor call center workers; Lichter, Pestel and Sommer (2015) and Archsmith, Heyes and Saberian (2016) for the performance of soccer players and baseball umpires, respectively; Ebenstein, Lavy and Roth (2016) and Roth (2016) for test score performance. See Graff Zivin and Neidell (2009) and Aldy and Bind (2014) for effects on demand for goods and services, such as for entertainment, hospitality and tourism.

organizations have traditionally focused their research on concurrent hours responses lost due to illness. Although a growing body of literature documents productivity effects, we know of no estimates of the general incidence on workers.

While wildfire smoke is understood to operate through the same channels as other sources of pollution, the composition of wildfire smoke may make it more harmful to human health per unit of measured particulate matter than most industrial sources of pollution.[10] The most comprehensive evidence pertaining to the effects of wildfire smoke exposure on health in the United States comes from Miller, Molitor and Zou (2017), who use national-scale variation in daily smoke exposure to document a link between smoke exposure and adult mortality and morbidity. Using conventional figures for the value of a statistical life-year, they find that the mortality cost of wildfire smoke is significantly higher than the hospital-related morbidity cost as captured by health care spending. Other case studies of wildfire smoke anomalies have also found suggestive evidence that the mortality cost of wildfire smoke exceeds the morbidity cost (e.g. Kochi et al., 2012, 2016). Another strand of research documents costs to infant health, which may have large valuations attached to them in cases of long-lasting damage (Jayachandran, 2009; McCoy and Zhao, 2016).

In addition to direct health effects, it is increasingly recognized that behavioral responses to air pollution pose a deep challenge to translating currently available estimates of the effects of air pollutants to policy-relevant parameters. Graff Zivin and Neidell (2013) survey the literature on avoidance behavior and discuss the challenges it presents for estimating the effects of air pollution.[11] For wildfire smoke in particular, survey research has documented a number of margins of behavioral responses to wildfire smoke, such as spending more time indoors, running air conditioners for longer times, and missing work (Richardson, Champ and Loomis, 2012; Jones et al., 2015). Richardson, Champ and Loomis (2012), examining a single large wildfire in California

[10] Research on the differences in the composition of smoke from biomass burning and car exhaust find higher reactivity of VOCs in smoke, which is consistent with the incomplete burning of the carbon material in a fire relative to internal combustion (e.g., Verma et al., 2009, 2015; Bates et al., 2015). Wildfire is noteworthy for containing a more noxious mix of chemicals and higher levels of extremely fine particulate matter than most biomass burns Liu et al. 2017.

[11] For examples of the literature on air pollution and avoidance behavior, see Chay and Greenstone (2005) for long-run responses, including residential sorting, and Moretti and Neidell (2011) for short-run avoidance behavior.

in 2009, estimate that the economic costs of health effects are comprised primarily of avoidance, defensive actions, and disutility, with only about 10 percent of costs due to illness.

The previous literature suggests that labor market effects may arise from health effects and avoidance during and immediately after air pollution events. More significant losses may occur if these short-run effects catalyze longer-run labor market responses, yet little is known about the long-run effects of transient air pollution shocks in adulthood on either health or labor markets. Theoretically, short-run health effects of air pollution may result in lasting earnings losses over a longer time period through either health channels or interactions with the labor market. Biomedical mechanisms exist through which short-run exposure may affect medium- and long-run health. Most directly, once particulate matter enters the body, it may take weeks or months for it to be cleared. In addition, transient exposure may result in adverse health events, such as heart attacks or the onset of asthma, reducing health capital and leaving exposed individuals more vulnerable to future health shocks. For example, exposure to adverse economic and environmental conditions in early childhood has been found to lower educational attainment and earnings later in life (Case, Lubotsky and Paxson, 2002; Isen, Rossin-Slater and Walker, 2017). Temporary labor market disruptions can also have lasting impacts on earnings and welfare, as shown in numerous studies of displaced workers and labor market entrants (Jacobson, LaLonde and Sullivan, 1993; Kahn, 2010; Oreopoulos et al., 2012; Borgschulte and Martorell, 2017). Workers in the United States have varying and often weak job protections when they or family members fall ill.[12] Wages may respond to more serious illnesses due to lasting changes in workers' productivity or employment. We know of no evidence on the effects of such responses to air pollution; however, their importance has been demonstrated in other contexts. For example, lower wages resulting from a health shock is the primary source of earnings losses following hospitalization (Dobkin et al., 2016).

To illustrate the multiple channels of action implied by the combination of direct health effects, behavioral responses, and long-run wage effects, we build a simple model of health and labor

[12]The Family Medical Leave Act covered 59% of workers in 2012, and allowed them to take up to 12 weeks of unpaid leave for their own serious health condition, or that of a spouse, parent, or child (Klerman, Daley and Pozniak, 2012).

supply to connect exposure to airborne pollutants with labor market earnings, our primary outcome measure. We model the utility of a representative agent in response to a fixed dose-response function, $s(c)$, relating exposure to pollution concentration, c, to sick days, $s(c)$. Pollution concentration may represent a vector of harmful components in wildfire smoke. An agent maximizes utility that depends on consumption, X, leisure, l, sick days, s, and exposure, c:

$$\mathbf{max}_{X,l} U(X,l,s,c)$$

$$\text{s.t. } Y + wh \geq X$$

$$l = T - s - h$$

Consumption will equal non-labor income, Y, and earnings, wh. Wages respond to pollution, $w = w(c)$, due to a combination of responses through three channels: changes in the returns to work arising from a decay in human capital after an illness, the incidence on workers of labor demand changes, and direct productivity effects during periods of high pollution. T reflects the total time endowment, from which days of illness, $s(c)$, are directly subtracted. Hours of work, $h = h(w(c),c)$, respond to wages and direct avoidance of high pollution.

The resulting earnings function is:

$$E(c) = w(c) \cdot h(w(c), s(c), c) \tag{1}$$

Taking derivatives and re-arranging yields a decomposition of the reduced-form effect:

$$\frac{dE(c)}{dc} = w\left[\frac{\partial h}{\partial s}\frac{ds}{dc} + \frac{\partial h}{\partial c}\right] + h\left[\frac{dw}{dc}\right](1+\eta_s) \tag{2}$$

The first bracketed term in Equation (2) captures the direct effects of pollution on labor supply. The first term inside the brackets, $\frac{\partial h}{\partial s}\frac{ds}{dc}$, denotes the loss of hours of work to illness, and the second term, $\frac{\partial h}{\partial c}$, reflects avoidance behavior. The second bracketed term, $\frac{dw}{dc}$, captures the effect of pollution on wages. The final term, $(1+\eta_s)$, scales the endogenous labor supply response to changes in the

wage; as wages fall with pollution exposure, workers may reduce their hours of work. Thus, we expect the effect of air pollution on earnings to be the sum of the effects working through the direct effect on hours, and the combined effects on wages and endogenous labor supply response.

The primary focus of the paper is on estimating $\frac{dE(c)}{dc}$, the total response of earnings to variation in air quality. We also examine evidence for the components of the losses, especially the response of hours through a labor force participation channel. Following our main estimates, we return in Section 5 to Equation 2 to guide our analysis of the welfare effects of the lost earnings.

2 Data

Our analysis relies on a novel, nationwide linkage of wildfire smoke exposure, air pollution, weather conditions, labor market outcomes, and mortality. These data sources include a rich set of remote sensing, environmental monitoring, federal income statistics, national representative surveys, and death records data files. This section describes the construction of the database and the definitions of our key variables used in the analysis.

2.1 Wildfire Smoke Data

A key innovation of this analysis is that we are able to observe labor market outcomes linked to annual counts of wildfire smoke exposure at a fine geographic level over a broad geographic scope. These daily smoke exposure data were originally developed by Miller, Molitor and Zou (2017) using wildfire smoke analysis produced by the National Oceanic and Atmospheric Administration's (NOAA) Hazard Mapping System (HMS). The HMS is a program that utilizes a variety of satellite and spacecraft observations to identify fire and smoke emissions over the contiguous United States (Ruminski et al., 2006). An important output of the HMS is the daily geo-referenced smoke plume files, drawn manually by smoke analysts, that represent the outlines of smoke plumes emitted by wildfires. We obtain digital archives of the daily plume files from 2006 to 2015 and construct our key smoke-exposure variable separately at the county level. To construct our measure of wildfire

smoke exposure, we first code the fraction of county that is covered by any smoke plume detected by the HMS on that day.[13] We then code a county as exposed to smoke if it is on the interior of the plume. The results are qualitatively similar, but slightly smaller magnitude, when we measure exposure using partial coverage with a smoke plume.

We complement satellite smoke observations with wildfire records from the National Fire and Aviation Management group of the U.S. Forest Service, which combines records from seven major fire and wildland management agencies.[14] We use these data primarily to distinguish areas directly affected by the burning of fires.

2.2 Pollution Data

We link satellite smoke observations with ambient air pollution monitoring data obtained from the U.S. Environmental Protection Agency's (EPA) Air Quality System (AQS). We extract monitor-daily readings for EPA "criteria pollutants," including fine particulate matters ($PM_{2.5}$), coarse particulate matter (PM_{10}), ozone (O_3), carbon monoxide (CO), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2). We focus on these pollutants because their concentrations are expected to elevate during wildfire events and they are recognized by the EPA to be among the most important pollutants that affect human health.

To measure air pollution at the local level, for each pollutant we take the weighted average of all readings from monitors that fall within 20 miles of a county's centroid, where the weights used are inverse monitor-to-centroid distance. Depending on the sparsity of the monitoring network, a significant number of areas have no pollution monitors within a 20-mile radius and therefore are missing pollution data. Pollution readings can differ by pollutant, as well: 1,642 counties have O_3 data, while we are able to obtain NO_2 observations for only 691 counties.

[13] In producing smoke plume outlines, the HMS uses data from the Geostationary Operational Environmental Satellite (GOES) visible band imagery available at the 1 km resolution and infrared bands at the 2 km resolution. The granular resolution enables us to explore geographically fine variations in exposure at the county level.

[14] These include the Bureau of Indian Affairs, the Bureau of Land Management, the Bureau of Reclamation, the California Department of Forestry and Fire Protection, the National Park Service Fire and Aviation Management, the U.S. Fish & Wildlife Service, and the U.S. Forest Service.

2.3 Weather Data

The analysis includes a flexible set of control variables for temperature, precipitation, and wind patterns. Temperature and precipitation data are collected from the National Climatic Data Center's Global Historical Climatology Network (GHCN), which provides station-daily-level information on minimum temperature, maximum temperature, and total precipitation. To construct weather conditions at the local level, we average daily weather readings from stations that fall within 20 miles of each county's centroid, weighting readings by inverse station-to-centroid distance.

We obtain wind speed and wind direction data from the National Centers for Environmental Information's North American Regional Reanalysis (NARR). NARR divides the United States into 32km$\times$32km grids, and for each grid-day it provides data on the East-West wind vector ("u-wind") and the North-South wind vector ("v-wind"), which together characterize windspeed and direction. Given the resolution of the data, we construct wind conditions at the county level by first linearly interpolating u-wind and v-wind vectors at the grid centroids to the county centroid, and then converting u-wind and v-wind at the county centroid into wind speed and wind direction.

2.4 Earnings Data

Our main earnings analysis uses outcome data from four sources: the Quarterly Workforce Indicators (QWI), the County Business Patterns (CBP), the Regional Economic Information System (REIS), and the Internal Revenue Service's (IRS) Individual Income Tax Statistics.While all four earnings sources approach full coverage of labor earnings in the United States, each source has a distinct construction. For example, the IRS data, which are based on stratified probability samples of individual income tax returns as reported on Forms 1040, 1040A, and 1040EZ, will include only workers who file tax returns by the end of the calendar year that follows the year of tax liability; of course, under- and mis-reporting may occur in these data.[15] CBP, which is based on the Census Bureau's Business Register, excludes data on self-employed individuals, employees of private households, railroad employees, agricultural production employees, and most government

[15] <https://www.irs.gov/pub/irs-soi/sampling.pdf>

employees, whereas REIS includes these workers.[16] QWI, on the other hand, excludes members of the armed forces, self-employed individuals, proprietors, and railroad employees. By using four distinct data sources, we can replicate the main findings and demonstrate robustness to the construction of aggregate labor income.

2.5 Labor Force Status and Social Security Data

We draw county-level labor force participation information from two sources. First, we use Local Area Unemployment Statistics (LAUS) published by the Bureau of Labor Statistics. LAUS contains county-level labor force estimates produced through a building-block approach that combines data from national representative surveys and state unemployment insurance systems.[17] Second, We use the Quarterly Workforce Indicators (QWI) published by the Census Bureau to measure employment jobs both at the county aggregate and by age groups. We measure number of retirement claimants and benefits at the county level using the Social Security Administration's (SSA) annual publications of Old-Age, Survivors, and Disability Insurance (OASDI) beneficiaries statistics. The SSA produces these statistics using its Master Beneficiary Record, which covers the universe of Social Security beneficiaries who are ever entitled to receive retirement and survivors insurance or disability insurance benefits. For each county-year, we observe retirement benefits paid out to claimants in that county. In addition to the SSA data, we also make use of the "retirement and disability insurance benefits" field available in the county-level REIS data.

2.6 Mortality Data

Mortality outcomes are measured in micro-data provided by the National Vital Statistics System. The underlying data are taken from death certificates which contain age of death. We use the

[16] CBP's payroll and employment information are derived from administrative records for the universe of firms. CBP's payroll measure is based exclusively on administrative records for single-unit companies. For multi-unit companies, CBP's payroll information comes from a combination of administrative records with Census data. See <http://www.census.gov/econ/cbp/methodology.htm>.

[17] LAUS county level estimates use survey data from the Current Population Survey, the Current Employment Statistics survey, and the American Community Survey (ACS). See <http://www.bls.gov/lau/lauov.htm>.

restricted data files containing month of death and all counties in the United States to link mortality outcomes to smoke exposure. The data are available for the entire sample period, 2006-2015, and contains 25.2 million deaths.

3 Empirical Framework

Researchers who study the labor market effects of environmental hazards face three primary challenges when seeking to identify the causal effects of air pollution on labor markets. First, observational correlations between air pollution and economic activity may be due in part to the causal effects of the economic activity on air pollution. As a result, finding a valid instrument for air pollution that does not have a direct effect on labor markets is difficult. For example, policy instruments which reduce air pollution may impose direct effects on the regulated markets. Second, transient changes in air pollution may induce short-run effects that reflect intertemporal substitution, rather than true welfare-reducing labor market effects. Third, existing evidence of a relationship between pollution and labor markets has generally focused on case studies of specific industries or regions. These specific settings may not produce nationally representative effects and may not generate sufficient variation to study relatively rare but significant outcomes, such as retirement or mortality.

3.1 Wildfire Smoke Exposure

We use annual variation in regional wildfire smoke exposure to identify the causal effects of transient air pollution shocks on labor markets. A few key features of wildfire smoke permits a research design that addresses the identification challenges described above.

First, wildfire smoke plumes are a natural source of air pollution, traveling hundreds or even thousands of miles downwind, affecting cities at great distances from the fire itself. Figure 1 provides summary statistics for the frequency of the events. The average county appears on the interior of a smoke plume for 17.7 days.[18] As can be seen, smoke exposure is concentrated in the upper

[18] Appendix Figure A.1 depicts an example of smoke exposure across much of North America during the Fort McMurray fires in northern Canada. Fires in the U.S. Southeast also appear in the figure.

Midwest, but exhibits significant year-to-year variation. Importantly for a study focused on air pollution, the pattern of smoke exposure differs markedly from wildfire footprints (see Appendix Figure A.2). Thus, we can study the effects of downwind smoke exposure separately from direct damages caused by occurrences of wildfires. The majority of our analysis excludes counties where fires burn.

Second, smoke shocks give rise to spikes in air pollution concentration, with the average magnitude large enough for us to expect significant health and behavioral responses. The upper panel of Table 1 shows that wildfire smoke increases concentrations of the six EPA criteria pollutants we examine, with the largest responses in particulate matter and ozone. An average smoky day increases $PM_{2.5}$ by 2.2 $\mu g/m^3$ on the day of exposure, about one-third of a standard deviation in the distribution of daily particulate matter. When we examine the cumulative effect of smoke days on annual pollution measures in the lower panel of Table 1, we estimate that increasing smoke exposure by one day increases the annual $PM_{2.5}$ by 0.019 $\mu g/m^3$; evaluated at the annual average number of smoke days (17.7), smoke raises a county's annual average $PM_{2.5}$ concentration by 0.336 $\mu g/m^3$, or about 16 percent of the annual standard deviation. This implies that each day of smoke exposure contributes 6.92 $\mu g/m^3$ to the annual sum of daily $PM_{2.5}$ concentrations. This annual concentration effect of one day of smoke exposure is over three times as large as the same-day effect, implying that particulate matter lingers in the air, increasing pollution levels on days that are not coded as smoke exposure days in the satellite data.

Third, most wildfire smoke events induce modest, but largely humanly imperceptible, changes in air quality, meaning that our estimates are not driven by a small number of intense smoke exposure days. A potential concern with using wildfire smoke is that extreme wildfire smoke events generate substantial news coverage, possibly triggering behavioral responses that would not be present with normal sources of air pollution. The vast majority of smoke exposure days in our data lie within the normally experienced levels of air quality, helping to allay this concern. Additional evidence on the clustering of smoke days appears in Figure 2, which depicts an event study with $PM_{2.5}$ as the outcome variable and time since a smoke day as the event time. Smoke

days are associated with elevated levels of fine particulate matter for four days, with an average increase of just over 2 $\mu g/m^3$ on the day of exposure off an average of 10.3 $\mu g/m^3$ (see Table 1). To put this into context, the EPA long-run standard for $PM_{2.5}$ is 15 $\mu g/m^3$, while the daily $PM_{2.5}$ standard is 35 $\mu g/m^3$, far above most exposure levels. Thus, although wildfire smoke is a unique source of pollution, it should not trigger dramatically different behavioral responses, as compared with other changes in air quality. Further details on the distribution of air quality appear in the Appendix.

3.2 Identifying the Effect of Smoke Exposure on Labor Market Outcomes

Our identification exploits variation in the annual, cumulative number of wildfire smoke days at the county level to identify the labor market effects of smoke exposure. We identify over 720,000 county-day smoke exposure events from 2006 through 2015, and we aggregate to the annual level to construct $SmokeDays_{ct}$, the number of days in year t to which county c was exposed to wildfire smoke. We then estimate the following regression equation:

$$Y_{ct} = \beta \cdot SmokeDays_{ct} + State_c \times Year_t + \alpha_c + X_{ct}\gamma + \varepsilon_{ct} \tag{3}$$

where Y_{ct} denotes labor market outcomes such as the log of per capita earnings in county c and year t. The primary coefficient of interest, β, can be interpreted as the effect of an additional day of wildfire smoke on annual earnings in the exposed county. We include county fixed effects, α_c, to control for time-invariant differences in county labor market outcomes. Our smoke exposure effects are therefore identified using year-over-year variation in smoke exposure within the same county. In addition, we control for state-by-year effects to capture time-varying changes at the state level, such as the Great Recession.[19] X_{ct} includes time-varying weather controls that may independently affect labor market outcomes. These include the summed exposure to various levels

[19] In the raw data, each additional day of smoke exposure in a county in a year is associated with 0.65 additional days of smoke exposure in the following year. Including county fixed effects, state-by-year fixed effects, and weather controls reduces this association to 0.07 additional days of smoke. Thus, conditional on these controls, there is little year-over-year serial correlation in wildfire smoke exposure.

of heat (10-degree Fahrenheit bins of daily temperature), rain (quadratic annual precipitation), and wind (60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed). To reflect the smoke-exposure effect on a representative resident, we weight the regressions by county-annual population. To adjust for both within-county and state-year autocorrelation, we two-way cluster standard errors at the county and the state-by-year levels.

4 Results

4.1 Annual Earnings

Table 2 presents results from the estimation of Equation 3 where the outcome is the log of per capita labor income at the county level. We use earnings as measured by four distinct data sources, as well as a simple average of the four separate earnings measures. The results reported in the main text drop counties that experienced wildfires in the same year.

Starting in column 1, we find that each day of wildfire smoke exposure in a county reduces average annual wage and salary earnings reported in the QWI by 0.059 percent. Across the four measures of annual labor income, exposure to wildfire smoke significantly lowers the average annual income in a county by between 0.054 and 0.080 percent. We cannot reject the equality of the estimates from the four data sources. We report the average income estimates in the remaining columns, and also add one lead and various lags of smoke exposure; we lose one year of data with the addition of each lead or lag, somewhat reducing the statistical power of the analysis. The effect across the average of the four income measures is 0.071 percent, as reported in column 5. When we add the first lead and the first lag of smoke exposure as shown in column 6, we find effects of 0.069 percent in the preceding year, and effects of 0.043 percent in the year of exposure. When comparing these magnitudes, it is important to note that most smoke occurs in the second half of the year. The average smoke day occurs in mid-July, day 196 of the year, meaning that we should multiply the year-of effects by 2.1 when comparing them with effects in the following years; we report the raw numbers in tables and figures, but this interpretation can be applied throughout the paper. In this

case, earnings drop by 0.090 percent in the remainder of the year of exposure, consistent with a larger effect in the immediate period following the smoky day. The sum of smoke exposure in the year before and the year of income measurement is a 0.112 percent reduction in earnings with a standard error of 0.035; summing over the year of and two years preceding income measurement gives an estimate of 0.106 percent reduction in earnings with standard error of 0.041. Multiplied by the average number of smoke days in the sample period, these estimates imply a total loss of just under 2 percent of national labor income in the United States each year as a consequence of wildfire smoke. Total losses in 2018 dollars are $147.2 billion using one lag, and $139.3 billion using two lags.[20]

The linear fit can be visually assessed in the residual plot in Figure 3. The figure reports a binned scatter plot of the average income residual in ten equally-sized bins of smoke exposure. The fit line represents our main estimate in column 5 of Table 2. Most of the residuals fall close to the regression line, and the results do not appear to be driven by extreme smoke exposure events. As suggested by this figure, in unreported analysis, we find that higher-order terms in exposure are not significant.

We perform several additional analyses and robustness checks, with detailed results reported in the Appendix. First, we test whether smoke exposure affects migration in and out of a county. We measure net migration flows (i.e. population size) using the total number of tax exemptions claimed in an area using IRS data. We also directly measure in- and out-migration using IRS county-to-county flows. The results, reported in Appendix Table A.1, indicate that population migration does not respond to smoke exposure to an economically or statistically significant degree. If anything, the insignificant point estimates indicate small *positive* effects of smoke exposure on the total population, and near-zero coefficients on both inflows and outflows. The lack of population migration response to smoke exposure suggests that our main effects are not an artifact of changes in population composition across regions. Next, we explore how the average smoke exposure effects reported in Table 2 vary by the intensity of smoke exposure. While our satel-

[20]The calculation uses $6.4 trillion as total wage and salary compensation in 2010, as reported by the Bureau of Economic Analysis, and the Consumer Price Index to translate losses in 2018 dollars.

lite measure of exposure captures the number of days of complete coverage in smoke, we could have used alternative measures based on partial coverage. We report the results of two alternative specifications in Appendix Table A.2, showing that similar results hold when we use the sum of (possibly) partial coverage or adopt a binary indicator based on greater than 75 percent (the average coverage conditional on *any* coverage) of the county covered in smoke. As a final robustness check, we examine robustness to alternative clustering choices for the calculation of the standard errors. Appendix Table A.4 shows that the choice to cluster at the county and state-by-year level has almost no effect on inference.

4.2 Extensive Margin and Retirement Behavior

An important and unanswered question in the literature on the labor market effects of air pollution is whether transitory air pollution episodes leave lasting impacts on labor markets. Two channels are suggested by the model in Section 1. First, air pollution may cause health events, such as asthma episodes or heart attacks, which lead to chronic health conditions. These chronic conditions may reduce workers' productivity and labor supply, in extreme cases, causing them to leave the labor force altogether. Second, diminished health, whether temporary or chronic, may affect labor market opportunities. An extensive literature in labor economics documents the lasting effects of job loss, suggesting that particularly large losses may occur with changes in extensive margin labor force attachment. Further, because the health of older workers may be more sensitive to pollution shocks, we hypothesize that smoke effects should be strongest among older workers, potentially generating losses associated with labor market transitions and retirements.

We test for effects on employment in the following ways. First, we test for labor force participation (LFP) responses in the LAU data. Column 1 of Table 3 shows the results when we use employment as the outcome in Equation 3, including a lead and a lag in smoke. Results suggest that each day of wildfire smoke reduces LFP in the county by 147 per million aged 16 and over. Second, we use the Census Bureau's Quarterly Workforce Indicators (QWI) to test explicitly for employment responses in total employment to smoke exposure. Column 2 contains results for all

workers, and shows a drop of 289 employees per million residents. Off an average employment rate of 63 percent, this implies that each day of exposure reduces employment by 0.046 percent.

In column 3 we focus on workers above age 55, and find a decline in employment of 177 employees per million residents.[21] Reduction in employment of older workers may reflect retirements, and for this reason, we next examine retirement-related outcomes, such as Social Security retirement benefit claiming, using data from the REIS and the Social Security Administration (SSA). For the REIS analysis, we examine total payments of retirement and disability benefits in the same framework as in the primary earnings analysis, finding a 0.026 percent increase in benefit payments for each day of exposure, as seen in column 4. In the SSA analysis, we use the same specifications as above applied to data derived from SSA's administrative records. Table 3 contains results for SSA benefits per capita (column 5). Importantly, the SSA data are annual, so retirements must be shifted significantly to be captured by this measure. We present the results of testing for a change in Security benefits paid, finding that the benefit per claimant rises 0.015 percent per day of smoke exposure. Comparing this result with the preceding estimates, we estimate that a share of the change in participation and employment may be associated with new Social Security claimants.[22] In all cases, we can interpret the increase in per beneficiary payments as evidence of increased need for financial resources among the elderly population.

A significant portion of the earnings results can be explained by the extensive margin effects. If those who leave the labor force earn average incomes and the reduction in labor supply lasts one year, the average annual exposure of 17.7 days implies a 0.81 percent reduction in earnings. Comparing this to the total effect of 1.26 percent reduction in earnings, implies that more than half of the earnings effect could be explained by extensive margin responses. This calculation illustrates the potential for relatively small but recurring shocks to employment to have large effects on total earnings. It is also important to note that we lack the power to examine how movements in and out of the labor force impact wages, an important channel in the job loss literature.

[21] In Appendix Figure A.3, we report the age-profile of response in the QWI. The largest responses are in the ages 55 to 64, and 65 and over.

[22] The Social Security rules do not require claimants to quit work to claim benefits, and it is possible that some claimants do not show up as labor force dropouts.

4.3 Heterogeneity by County Characteristics and Industry

With our national variation, we can perform heterogeneity analyses that it has not been possible to conduct in previous studies of air pollution and labor markets. Patterns in heterogeneity may provide suggestive evidence of underlying mechanisms behind the earnings and employment losses. To maintain consistency with our previous analysis, we examine how county-level characteristics predict the size of earnings losses. We split the sample into above- and below- median values of a number of characteristics: fraction that is urban, fraction in poverty, median home value, fraction that is black, and average smoke days. We then re-run the earnings models with interactions between smoke exposure and indicators for the characteristics.

In Table 4 we report the results of the earnings heterogeneity analysis. We base the analysis off of the dynamic specification shown in column 6 of Table 2, and we focus on the interaction between the current year smoke effect and county-level characteristics. In the first column, we find that counties with above average urban fractions explain the majority of the earnings losses we find, with a 0.049 percent reduction in labor income, while in areas with below average fraction urban, there is no detectable effect. We test whether we can reject the equality of these coefficients, finding a p-values less than 0.01 . In this case, we strongly reject the equality of the effect in more- and less-urban areas, and conclude that responses are concentrated in urban areas. This finding is important for excluding a direct effect of fires as a primary mechanism behind the earnings losses. Effects are also concentrated in high home-value areas, consistent with the urban-rural heterogeneity. Continuing across the columns, we report that we find larger earnings losses in areas with higher poverty rates and higher fraction black, though we cannot reject the equality of the coefficients in both cases very precisely (p-values > 0.06). In the last column, we stratify counties based on county's 10-year (2006-2015) average $PM_{2.5}$ concentration. One possibility is that that high pollution areas may have more fully adapted to exposure, which could be reflected in a lower responsiveness to smoke. The evidence suggests, however, that there is no significant heterogeneity across high vs. low pollution areas.

We also conducted additional analyses of heterogeneity by industry with results reported in

Figure 4. The industry analysis is conducted using the QWI data, which identifies earnings at the industry level. Point estimates are generally insignificant, but suggest that smoke has stronger effects in the mining, real estate, and construction industries; weighted by industry size, the largest losses are in manufacturing, professional services, and construction. We find a relatively sharp zero in agriculture, in spite of previous evidence of reduced worker productivity in previous studies. Note, however, that the agriculture sector is rather broadly defined in our study and includes sectors such as crop and animal production, logging, fishing, etc. One interpretation of this evidence is that, in some settings, intertemporal substitution compensates for daily fluctuations in output that have been identified by previous studies.

5 Welfare

5.1 Air Pollution and Welfare

In the preceding sections we have demonstrated earnings losses and changes in labor force participation as a result of wildfire smoke exposure. Of course, the earnings response does not necessarily reflect individual or social welfare responses, which depend on the mechanisms that explain the decreased earnings. In particular, at least some portion of the lost earnings may be explained by increased leisure, for example, if workers stay home on high-pollution days. Similarly, if some workers are forced into early retirement by smoke-related illness, we would like to account for the replacement of market work with home production. Optimal policy should weigh the marginal cost of reduced pollution against the marginal damage to social welfare.

5.1.1 The Double Dividend through Increased Labor Income

Studies in public and environmental economics consider how air pollution regulation interacts with the tax-distorted labor markets. While taxes on pollution may or may not generate any benefits in the labor market (Goulder, 1995; Fullerton and Metcalf, 2001), pollution regulations that improve labor incomes through health and productivity channels are shown to produce a "double dividend"

(Schwartz and Repetto, 2000; Williams III, 2002, 2003). This second source of welfare gains arises because increases in labor supply alleviate pre-existing tax distortions associated with payroll, income, and sales taxes.

Calibrating the changes in welfare through this channel is straightforward in partial equilibrium. On the margin, increases in labor supply will reduce deadweight loss by an amount that equals the change in labor times the average marginal tax rate for affected individuals. While we do not have a direct measure of this tax rate, we can use a moderate value of 25% to calculate that welfare increases by one-quarter of the $93 billion total loss, or $23 billion. This is a conservative lower bound on the total welfare loss, as it does not consider the effects of changes in individual welfare through either increased post-tax income or utility gained from health and amenities.

5.1.2 Individual and Social Welfare

For individual welfare, we can perform a simple calculation building off the models in Section 1 and Dobkin et al. (2016), and estimates reported in Table 2. To focus attention on the labor market costs, we separate workers' losses that occur through consumption and leisure, x and l, from direct losses arising from changes in health and amenities, s and c. We label utility from the first two terms as $U^{LM}(x,l)$; normalizing by the marginal utility of consumption gives the labor market component of welfare, W^{LM}. In the next subsection, we return to the issue of costs arising from illness. We also simplify the model by dropping avoidance behavior, and focusing on long-run effects, motivated by the persistent losses we find in the earnings analysis. Individual welfare losses arise from reductions in the wage, endogenous labor supply responses, and reductions in the time endowment due to illness. Social welfare losses include these changes in addition to changes in deadweight loss, i.e. the double dividend channel.

Considering a small change in pollution concentration, c, the loss in money-metric utility to the worker is

$$\frac{dW^{LM}}{dc} \equiv \frac{dU^{LM}/dc}{MU_x} = h\frac{dw}{dc} - w\frac{ds}{dc}.$$

The first term relates to the change in the wage, which leads to a welfare loss in proportion to

labor supply, h. Intuitively, a lower wage directly subtracts dollars from consumption; then, hours change in response to reflect a re-optimization at this lower utility frontier. The second term reflects the direct loss of time due to illness, valued at the wage. We can then take the ratio of the above individual welfare loss to the lost earnings in order to calibrate the appropriate scaling of the earnings losses.

Absent detailed data on time use and illness, we require some assumptions to calibrate the percentage of share of earnings losses that reflect true welfare costs to individuals. We focus on the case where all responses arise from changes in the wage, as in Dobkin et al. (2016), but also consider changes in the time endowment to provide an informative upper bound. Specifically, individual welfare losses as a share of earnings losses lie between the wage response, $\frac{1}{1+\eta_{h,w}}$, and an upper bound of unity, the case when all earnings losses reflect time spent sick. Should welfare costs arise entirely due to changes in the wage, we can take a conservative value of the labor supply elasticity, $\eta_{h,w} = 0.5$ (drawing from Keane (2011), as in Dobkin et al. (2016)), to estimate that two-thirds of the earnings loss reflect true costs to the worker.

Moving from individual to social welfare involves considering both individual welfare losses and changes in deadweight loss from taxation. In the case where earnings losses arise from responses to the wage, social welfare losses are the sum of individual losses and the deadweight loss of the labor supply response due to taxation, which can be calculated by multiplying the marginal tax rate by the difference between earnings responses and the individual welfare loss.[23] Assuming a marginal tax rate of 25% and $\eta_{h,w} = 0.5$ implies a social welfare effect of 75% (two-thirds from labor supply plus one-twelfth from deadweight loss) of lost earnings.

Applying the above model to the estimates reported in Table 2, we find that the welfare losses working through labor market responses are $70 billion in 2010 dollars. The lasting damage to labor market opportunities show up as lower wages, but may reflect either reduced health capital following an acute smoke-induced illness (i.e. lower productivity of workers following the health shock), or worker transitions to lower-paid jobs induced by illness or labor-demand effects. Losses

[23] Intuitively, lost earnings that arise from labor supply response are replaced by leisure in the individual's utility. However, this leisure is subsidized by the government at the marginal tax rate, leading to deadweight loss.

may approach an upper bound of $93 billion, if responses occur entirely through perfectly inelastic responses, as when workers are constrained from working by illness. Alternatively, at a lower bound where all lost income arises from perfectly elastic labor-supply responses, social welfare falls by 25% of lost earnings, or $23 billion. We regard this scenario as unrealistic; it is informative primarily because it generates important welfare responses entirely through the double dividend channel, and applies under the most pessimistic model of individual behavior. Costs associated with mortality, health care expenditures, the disutility of smoke-induced illness, and other costs would then be added to this figure to reach the total damage done by wildfire smoke.

5.2 Comparison with Mortality Costs

To evaluate the importance of incorporating labor market effects into estimates of air pollution costs, we benchmark the welfare cost of lost earnings against that of premature deaths due to smoke exposure. We estimate mortality effects of wildfire smoke using a regression specification that closely mirrors Equation 3 from our earnings analysis. We regress the mortality rate M_{cmy} in county, c, month of the year, m, and year, y, on the number of days, $SmokeDays_{cmy}$, in which the county was exposed to wildfire smoke that month:

$$M_{cmy} = \beta \cdot SmokeDays_{cmy} + State_c \times Year_y + County_c \times Month_m + \alpha_c + X_{cmy}\gamma + \varepsilon_{cmy}. \quad (4)$$

The primary coefficient of interest is β, which describes the effect of an additional day of smoke exposure on mortality. We measure mortality as deaths per million in the month of smoke exposure. To account for delayed mortality effects as well as possible short-run mortality displacement (harvesting), we also estimate specifications where mortality is measured over 3-, and 6-month windows beginning with the month of exposure. We include the same weather controls, X_{zt}, as in Equation 3 and add fixed effects for county by month to control for seasonality in mortality. Standard errors are two-way clustered at the county and state by year levels.

Table 5 reports the results of the mortality analysis. Across all ages (column 1), mortality in

the month of exposure increases by 0.23 deaths per million individuals (panel A), with effects growing to 0.37 deaths per million within three months of exposure (panel B). Extending the mortality window to six months following exposure (panel C) yields a precisely measured mortality estimate of 1.5 deaths per million. Further extending the post-exposure window to a year produces a positive but insignicant estiamte of 0.92. Taken together, these estimates indicate medium-run mortality effects of smoke that level off between 3 to 6 months following smoke exposure.

In columns 2 and 3 of Table 5 we report estimates of the mortality effect of smoke exposure separately by age group. We find mortality responses are concentrated among individuals aged 60 and older. Among this group, each day of smoke increases mortality by 1.8, 3.8, 9.3 and (imprecisely estimated) 4.0 deaths per million within 1, 3, 6, and 12 months of smoke exposure, respectively. The pattern suggests that the 6-month mortality estimates capture the extent of premature mortality from smoke, including potentially lagged effects. Any additional deaths at between 6 and 12 months are approximately equal to those deaths shifted from this period to the first 6 months following exposure.

To calculate the welfare cost of smoke mortality, we scale the age group-specific mortality effects by the average life expectancy among individuals in these groups and assume a $100,000 value for each year of life lost, following Deschênes and Greenstone (2011).[24] If individuals who die from smoke exposure are less healthy than average among their age group, this approach will generate an upper bound on welfare cost of smoke mortality. Because of the small and statistically insignificant effects among younger age groups, we assume a negligible mortality cost among all age groups except the oldest group.

Following this approach, we calculate the welfare cost of smoke mortality by multiplying the mortality effect of smoke among the population aged 60 and older (9.265 deaths per million per day of smoke), the average life expectancy among this population (16.1 years of life per death), the size of this population in millions (59.0, per 2012 U.S. Census), the average number of smoke days per year (17.7), and the value of a life-year ($100,000 per life-year lost). This generates an

[24] We calculate average life expectancy within each age group from the 2014 period life table for the Social Security area population, downloaded from https://www.ssa.gov/oact/STATS/table4c6.html#ss on September 8, 2017.

estimate of $15.6 billion in mortality costs due to wildfire smoke each year.

A key feature of the analysis is that we use the same measured variation in both the earnings and mortality analyses. The estimates from the labor market can then be directly compared with the mortality costs. We find that mortality costs ($15.6 billion) are approximately one-sixth of the lost labor market earnings ($93 billion). Our preferred estimate of $70 billion in welfare cost of these lost earnings implies that the labor market cost of air pollution due to wildfire smoke exposure is more than four times the mortality cost. Even taking our lower bound of $23 billion in welfare cost due to lost earnings implies a labor market cost that is 1.5 times as large as the mortality cost. A smaller ratio would be estimated if we consider only earnings losses in the year of exposure; since most exposure occurs in the second half of the year, this more closely corresponds to the 6-month mortality window. Earnings losses in the same year represent between 0.6 and 1.7 times the costs to mortality. These welfare calculations demonstrate that lost labor market earnings represent a source of welfare losses that should be considered as large or larger than mortality costs, which are usually thought to comprise the majority of health costs. A full welfare accounting would consider elements such as medical costs, losses to firms, the deadweight loss of additional tax revenue required to make Social Security payments, as well as adaptive and defensive investments. However, the literature has traditionally focused on mortality as the primary driver of costs, and including these elements would not alter the primary conclusion, that labor market responses impose an important, or even primary, share of the welfare costs of wildfire smoke.

6 Discussion and Conclusion

Wildfires cause severe damage to the areas they burn, destroying homes and property and threatening human lives in their path. Wildfires also produce a harmful and prevalent source of air pollution, to which most of the U.S. population is exposed at some point each year. We analyze annual variation in wildfire smoke exposure across the United States and find that increases in smoke ex-

posure cause significant decreases in earnings, which in turn are associated with decreased labor force participation and increases in Social Security benefits. These findings suggest that the impacts of reduced air quality on worker productivity do not just fall on firms, such as through higher sick leave expenses, but are at least partly passed on to workers in the form of lower earnings. In addition, we find that the welfare cost of these lost earnings is significantly larger than the cost of increased mortality due to the same wildfire smoke events.

The findings in this paper have broad implications for environmental policy and the growing body of literature on the labor market effects of air pollution. Many agencies that engage in environmental policy making, such as the WHO, OECD, World Bank, and EPA, have traditionally treated pollution damages arising from lost labor market hours and earnings as considerably smaller than the mortality cost of air pollution. Our results, which provide the first quasi-experimental evidence of the effect of air pollution events on labor markets at a national scale, indicate that environmental policies that ignore the labor market effects of air pollution ignore a significant cost, and may be designed inefficiently as a result. In addition, our results imply that the employment-reducing effects of environmental regulation are at least partially offset by earnings and employment gains to workers resulting from improved air quality. Although wildfire smoke has a different chemical composition from that of industrial pollution or vehicle exhaust, the large labor market costs of wildfire-emitted pollutants—which comprise a significant share of all U.S. particulate matter emissions—suggest that other EPA-monitored pollutants that negatively affect health may similarly have large labor market costs.

Our findings also have direct implications for wildfire policy and management. A primary implication of our results is that downwind labor market effects of wildfire smoke generation create large externalities in land use and fire management. These effects call for greater coordination of fire policy efforts, including a focus on preventing smoke-producing wildfires from starting and spreading in addition to the narrower goal of defending land and property exposed to fire damage. For example, fires generating large smoke plumes that may reach urban centers should be prioritized over fires that burn far from or downwind of population centers. Management of forest

fires, which typically consume dense biomass and therefore generate large, thick smoke plumes, should be prioritized over prairie fires that consume less biomass. The use of prescribed fires to remove fuel and limit the scope for larger future burns should likely expand, although such fires should be set only after taking into account wind patterns to avoid population exposure. Finally, estimates of the marginal cost of firefighting and prevention—which we note are sorely lacking in the literature—should consider both the cost of reducing acreage burned and the cost of reducing population smoke exposure. While wildfires and smoke cannot and should not be completely eliminated, planning and optimal policy can mitigate damages from these events.

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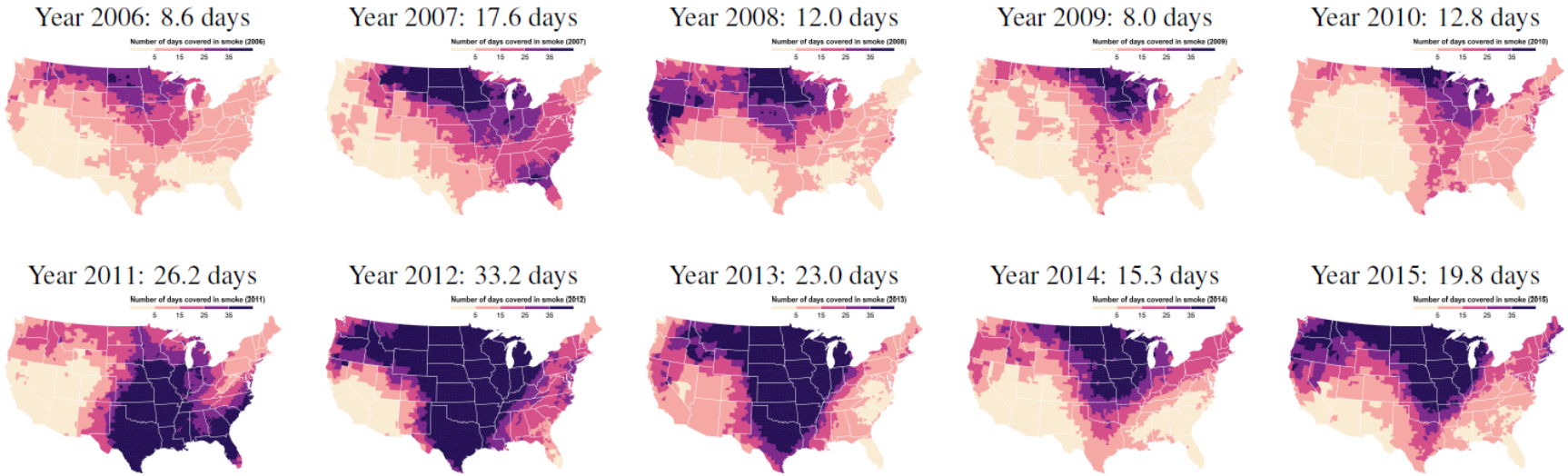
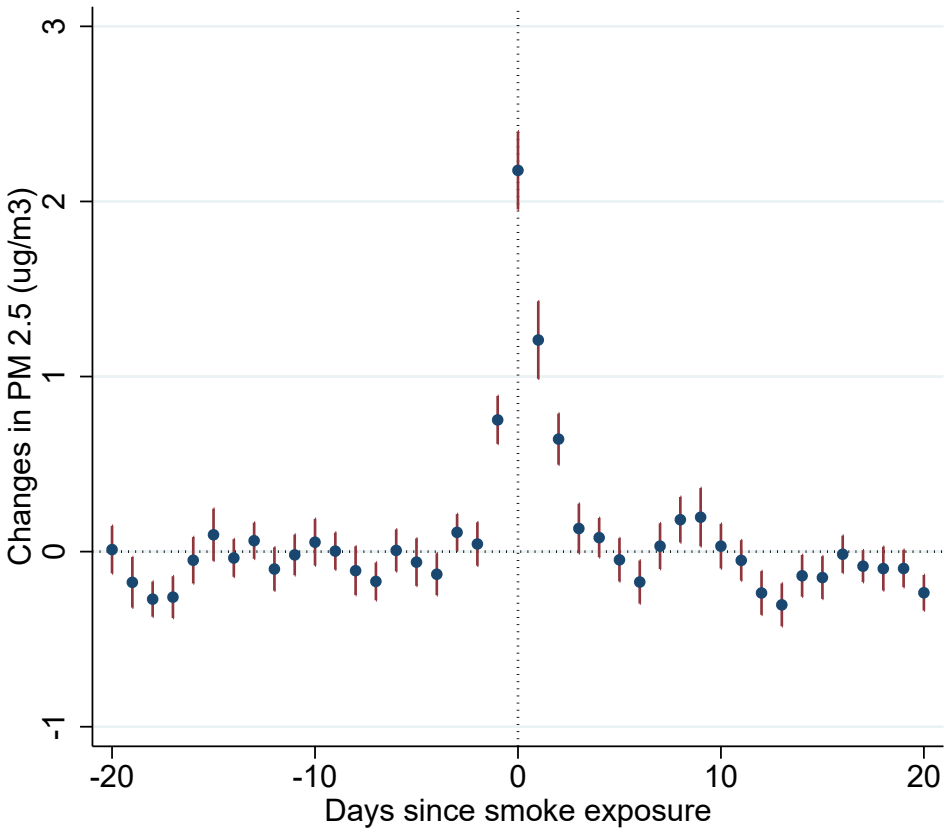
Figure 1: County Annual Wildfire Smoke Exposure 2006 – 2015



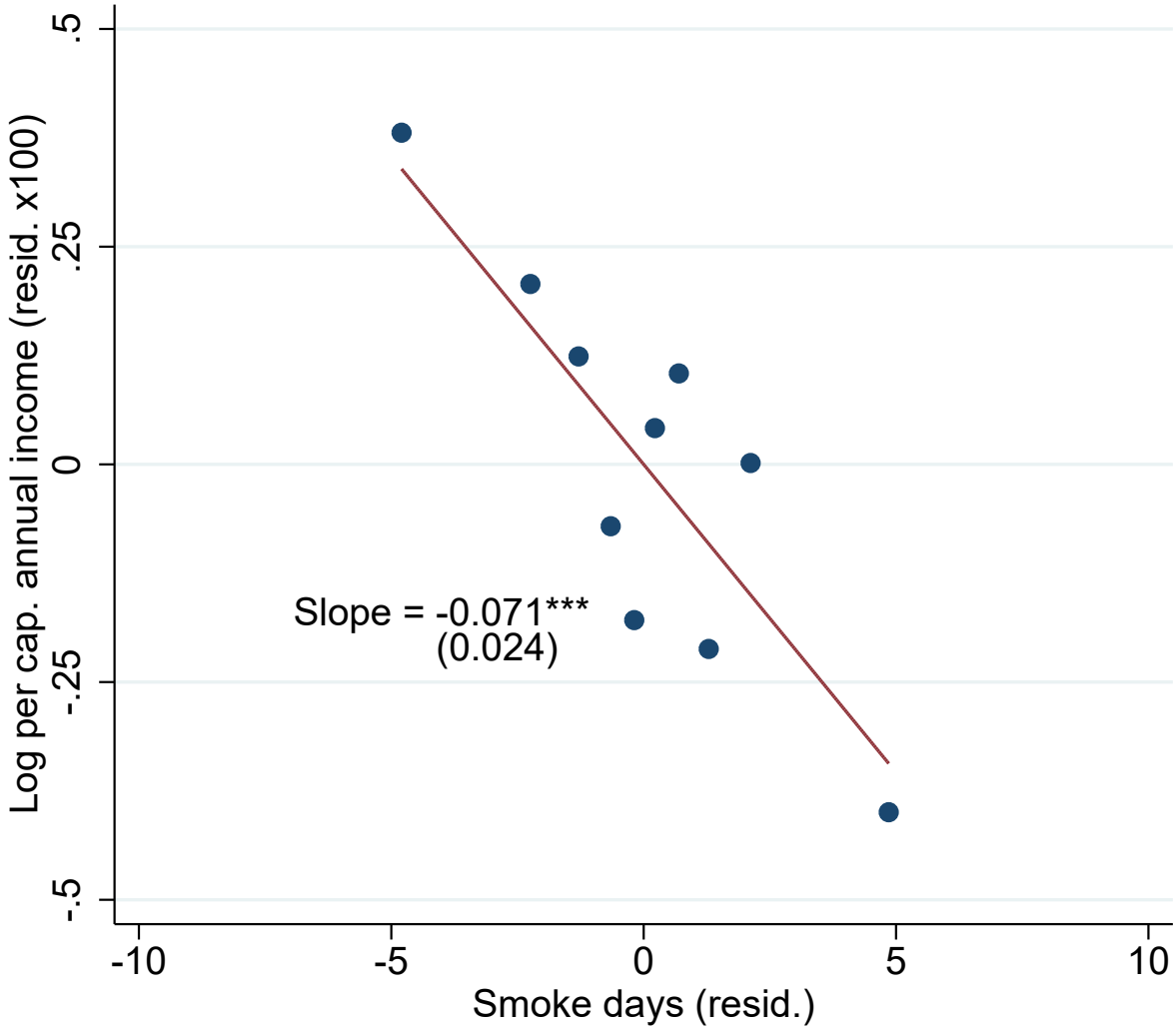
Notes: This figure plots county-level average annual days of smoke exposure for the lower 48 states for each year from 2006 to 2015. Average population-weighted exposure during this period was 17.7 days per year. Grayscale shading indicates quintiles of smoke exposure: 0 – 5 days (lightest shading), 6 – 15 days, 16 – 25 days, 26 – 35 days, or more than 35 days (darkest shading).

Figure 2: Daily Air Pollution Effects of Wildfire Smoke: Event Study



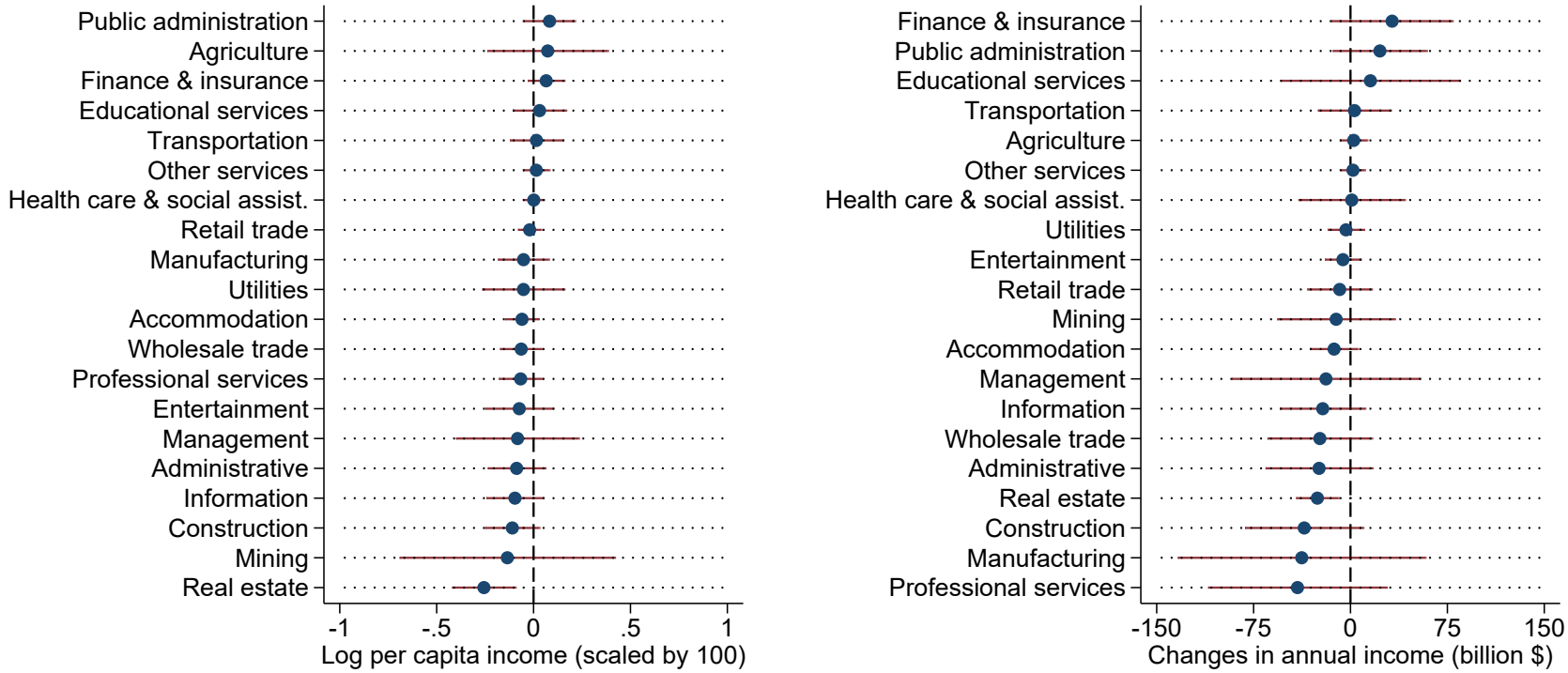
Notes: This figure shows coefficients from a regression of daily $PM_{2.5}$ on indicators of daily smoke exposure up to 20 days before and after the day of observation. In addition to the 41 smoke indicators, the regression controls for county-by-week-of-year fixed effects, state-by-year fixed effects, day-of-week fixed effects, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic daily precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. Daily observations for years in which a county experienced a wildfire are excluded. Standard errors are clustered at both the county and the state-by-year levels.

Figure 3: Annual Wage Income Effect of Wildfire Smoke: Residualized Plot



Notes: This graph shows a binscatter of residualized log per capita wage income by residualized annual smoke days. The dependent variable is the log of the average per capital annual income across IRS, CBP, REIS, and QWI data, scaled by 100. The focal independent variables capture the number of days in a year on which a county was exposed to wildfire smoke. The slope coefficient in this graph reflects percentage changes in per capita income per day of smoke. All regressions include county fixed effects, state-by-year fixed effects, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic annual precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. All regressions exclude observations for years in which a county experienced a wildfire.

Figure 4: Annual Wage Income Effects of Wildfire Smoke: Industry Profile



Notes: The figure reports estimated effects of an additional day of wildfire smoke exposure on log per capita (left) and economy-wide total (right) annual income separately for 2-digit NAICS industries. The focal independent variables capture the number of days in a year on which a county was exposed to wildfire smoke. All regressions include county fixed effects, state-by-year fixed effects, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic annual precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. All regressions exclude observations for years in which a county experienced a wildfire. Standard errors are clustered at both the county and the state-by-year levels.

Table 1: Daily and Annual Air Pollution Effects of Wildfire Smoke

	(1)	(2)	(3)	(4)	(5)	(6)
Unit of measurement:	$PM_{2.5}$ ug/m3	PM_{10} ug/m3	O_3 ppb	CO ppb	NO_2 ppm	SO_2 ppm
Panel A. Daily effect						
1(Smoke)	2.173***	3.743***	3.162***	11.190***	0.530***	0.157***
	(0.118)	(0.179)	(0.147)	(1.063)	(0.068)	(0.022)
Mean dep. var.	10.38	21.11	27.39	360.17	11.89	1.94
SD dep. var.	6.02	13.26	10.59	172.16	7.57	2.53
Number of observations	2,338,609	1,347,701	3,640,761	1,594,882	1,714,565	2,566,112
Panel B. Annual effect						
Smoke days	0.019*	0.077**	0.032***	-0.413	-0.021*	0.007
	(0.010)	(0.037)	(0.010)	(0.636)	(0.012)	(0.006)
Mean dep. var.	10.26	20.68	27.83	358.64	11.73	1.93
SD dep. var.	2.12	6.26	3.77	109.60	4.99	1.52
Number of observations	11,040	7,428	12,766	4,516	4,937	7,249

Notes: The table reports estimated effects of a day of wildfire smoke on pollution that day (panel A) and an additional day of wildfire smoke exposure on annual pollution (panel B). Each panel-column corresponds to a separate regression using county-daily/annual observations and county population weights. Daily-level regressions include county-by-week-of-year fixed effects, state-by-year fixed effects, day-of-week fixed effects, 3 leads and 3 lags of smoke day indicators, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic daily precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. Annual-level regressions include county fixed effects, state-by-year fixed effects, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic annual precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. All regressions exclude observations for years in which a county experienced a wildfire. Standard errors are clustered at both the county and the state-by-year levels.

Table 2: Annual Wage Income Effect of Wildfire Smoke

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Dependent variable: log of per capita annual income (scaled by 100)							
Smoke days (year $t+1$)						-0.033	-0.026
						(0.021)	(0.018)
Smoke days (current year t)	-0.059**	-0.080***	-0.054**	-0.065***	-0.071***	-0.043**	-0.035*
	(0.026)	(0.029)	(0.027)	(0.025)	(0.024)	(0.018)	(0.018)
Smoke days (year $t-1$)						-0.069***	-0.038**
						(0.024)	(0.017)
Smoke days (year $t-2$)							-0.032*
							(0.018)
Wage data source	QWI	CBP	REIS	IRS	All	All	All
Mean per capita income	20,496	17,299	22,154	21,494	20,533	20,359	20,449
Number of observations	22,803	22,711	22,579	22,861	22,851	18,309	16,045

Notes: The table reports estimated effects of an additional day of wildfire smoke exposure on annual income. Each column corresponds to a separate regression using county-year observations and county population weights. In columns (1)–(4), the dependent variable is the log of per capita annual income as measured by the data source indicated in the bottom panel, scaled by 100. In columns (5)–(7), the dependent variable is the log of the average per capital annual income across all (four) data sources, scaled by 100. The means of per capita income are in 2010 dollars. The focal independent variables capture the number of days in a year on which a county was exposed to wildfire smoke. Coefficients reflect percentage changes in per capita income per day of smoke. All regressions include county fixed effects, state-by-year fixed effects, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic annual precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. All regressions exclude observations for years in which a county experienced a wildfire. Standard errors are clustered at both the county and the state-by-year levels.

Table 3: Annual Employment and Retirement Effects of Wildfire Smoke

	(1)	(2)	(3)	(4)	(5)
	LFP (per million pop.16+)	Employment (per million pop.16+)	Employment (per million pop.55+)	Retire. & DI benefits (log per cap.$\times$100)	Retire. benefits (log per cap.$\times$100)
Smoke days (year $t+1$)	-17.0	-96.3	-96.0	0.009	0.009
	(137.8)	(119.7)	(75.1)	(0.019)	(0.022)
Smoke days (current year t)	-146.9*	-288.9***	-176.6***	0.026*	0.015
	(88.5)	(103.6)	(62.8)	(0.016)	(0.018)
Smoke days (year $t-1$)	-78.1	-153.6	-177.0**	0.037*	0.033
	(110.2)	(107.8)	(73.7)	(0.021)	(0.026)
Mean dep. var.	633,873	634,295	366,257	86.73	45.08
Data source:	BLS LAU	QWI	QWI(55+)	REIS	SSA
Number of observations	18,309	18,273	18,273	18,092	17,888

Notes: The table reports estimated effects of an additional day of wildfire smoke exposure on annual labor force participation, employment and retirement outcomes. Each column corresponds to a separate regression using county-year observations and relevant county population weights. The focal independent variables capture the number of days in a year on which a county was exposed to wildfire smoke. In columns (1) (2) and (3), coefficients reflect changes in labor force participation and employment outcomes (counts per relevant population) per day of smoke. In columns (4) and (5), coefficients reflect percentage changes in per capita income receipt per day of smoke. All regressions include county fixed effects, state-by-year fixed effects, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic annual precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. All regressions exclude observations for years in which a county experienced a wildfire. Standard errors are clustered at both the county and the state-by-year levels.

Table 4: Heterogeneous Wage Income Effects of Wildfire Smoke

	(1)	(2)	(3)	(4)	(5)
Dependent variable: log of per capita annual income (scaled by 100)					
Characteristics k:	Frac. urban	Median home value	Frac. poverty	Frac. black	Avg. $PM_{2.5}$
Smoke days $\times$ 1($k <$median)	-0.015	-0.023	-0.035*	-0.034*	-0.038**
	(0.018)	(0.018)	(0.018)	(0.019)	(0.017)
Smoke days $\times$ 1($k \geq$median)	-0.049***	-0.049**	-0.050**	-0.052***	-0.040**
	(0.019)	(0.019)	(0.020)	(0.019)	(0.020)
p-value	0.000	0.006	0.061	0.061	0.748
Number of observations	18,309	18,309	18,309	18,309	10,139

Notes: The table reportes heterogeneous effects of an additional day of wildfire smoke exposure on annual wage income. Each column corresponds to a separate regression using county-year observations and relevant county population weights. The focal independent variables capture the number of days in a year on which a county was exposed to wildfire smoke. Interaction terms are county-level above- and below-median indicators for fraction of urban population (column 1), median home value (column 2), fraction of population living under 100% of the Federal Poverty Line (column 3), fraction of population that is African American (column 4), average $PM_{2.5}$ during the study period (column 5). p-value corresponds to the null that there is no differential effect of smoke across above- and below-median groups. All regressions include county fixed effects, state-by-year fixed effects, one lead and one lag in smoke exposure, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic annual precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. All regressions exclude observations for years in which a county experienced a wildfire. Standard errors are clustered at both the county and the state-by-year levels.

Table 5: Monthly Mortality Effect of Wildfire Smoke

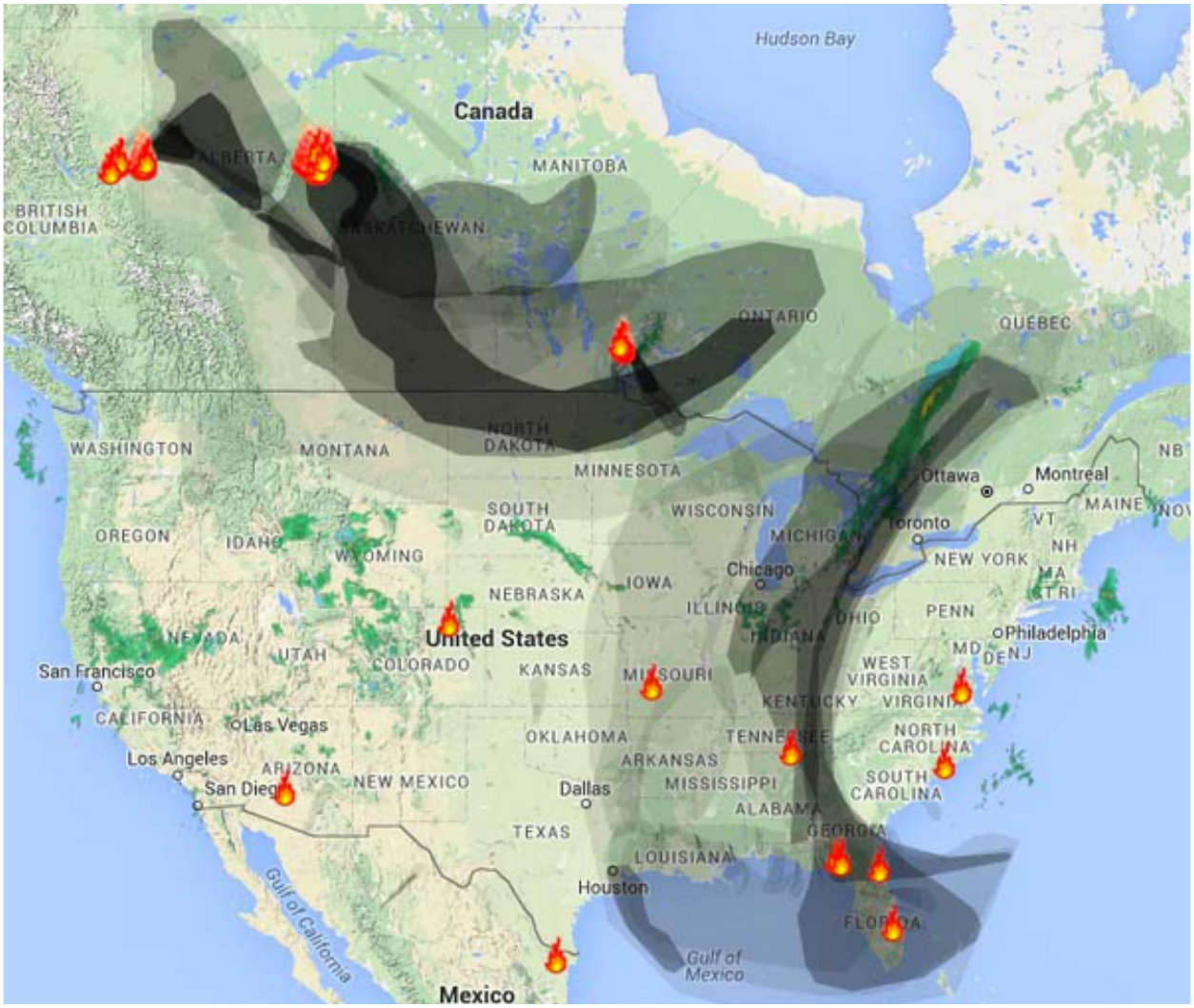
	(1)	(2)	(3)
	All-age	Ages below 60	Ages above 60
Panel A. 1-month mortality effect			
Smoke days	0.232**	0.091**	1.846***
	(0.110)	(0.042)	(0.573)
Mean dep. var.	703.2	173.7	2944.6
Panel B. 3-month mortality effect			
Smoke days	0.368	0.141	3.781***
	(0.262)	(0.087)	(1.241)
Mean dep. var.	2108.9	520.9	8846.3
Panel C. 6-month mortality effect			
Smoke days	1.499***	0.197	9.265***
	(0.536)	(0.143)	(2.453)
Mean dep. var.	4218.7	1041.7	17747.3
Panel D. 12-month mortality effect			
Smoke days	0.917	-0.118	4.043
	(0.642)	(0.227)	(2.958)
Mean dep. var.	8457.4	2082.6	35816.8
Avg. life years lost	44.5	50.4	16.1

Notes: The table reports estimated effects of an additional day of wildfire smoke exposure on monthly mortality rate. k-month mortality is the number of deaths in the next k months (including the current month) divided by relevant population. Each panel-column corresponds to a separate regression using county-monthly observations and relevant county population weights. The focal independent variables capture the number of days in a month on which a county was exposed to wildfire smoke. All regressions include county-by-month-of-year fixed effects, state-by-year fixed effects, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic monthly precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. Regressions in panels B, C, and D control additionally for number of smoke days in the corresponding look-ahead windows. All regressions exclude observations for years in which a county experienced a wildfire. Standard errors are clustered at both the county and the state-by-year levels.

A Online Appendix

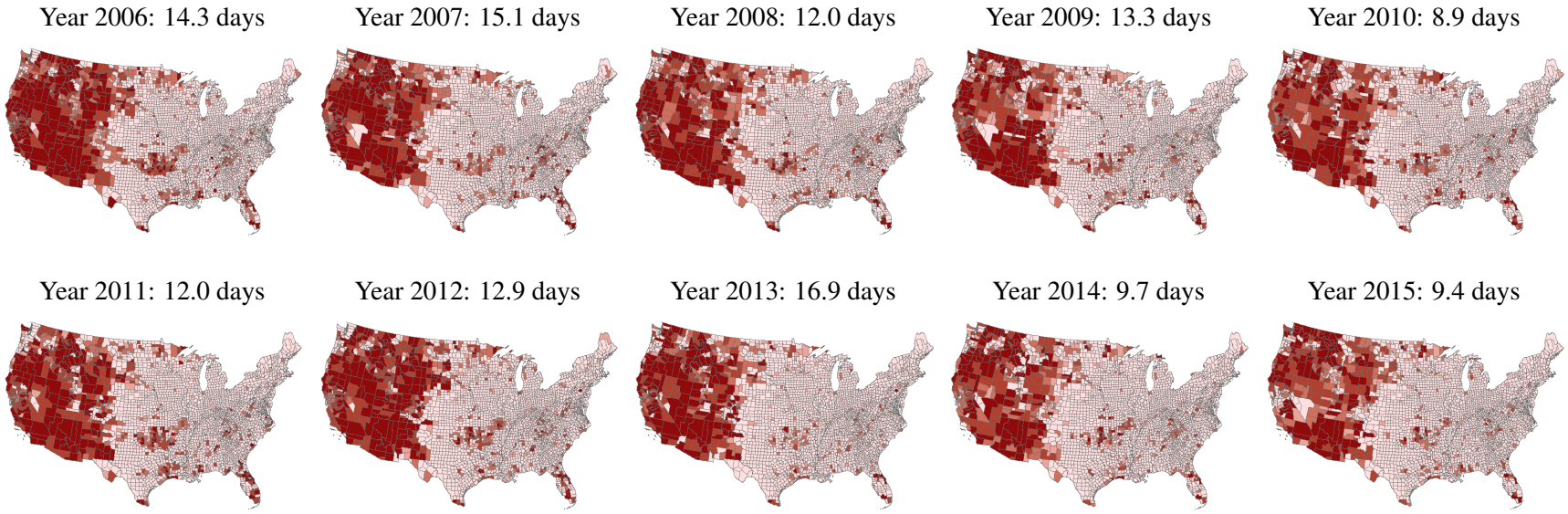
Online Appendix Figures and Tables

Figure A.1: Fire and Smoke on May 7, 2016



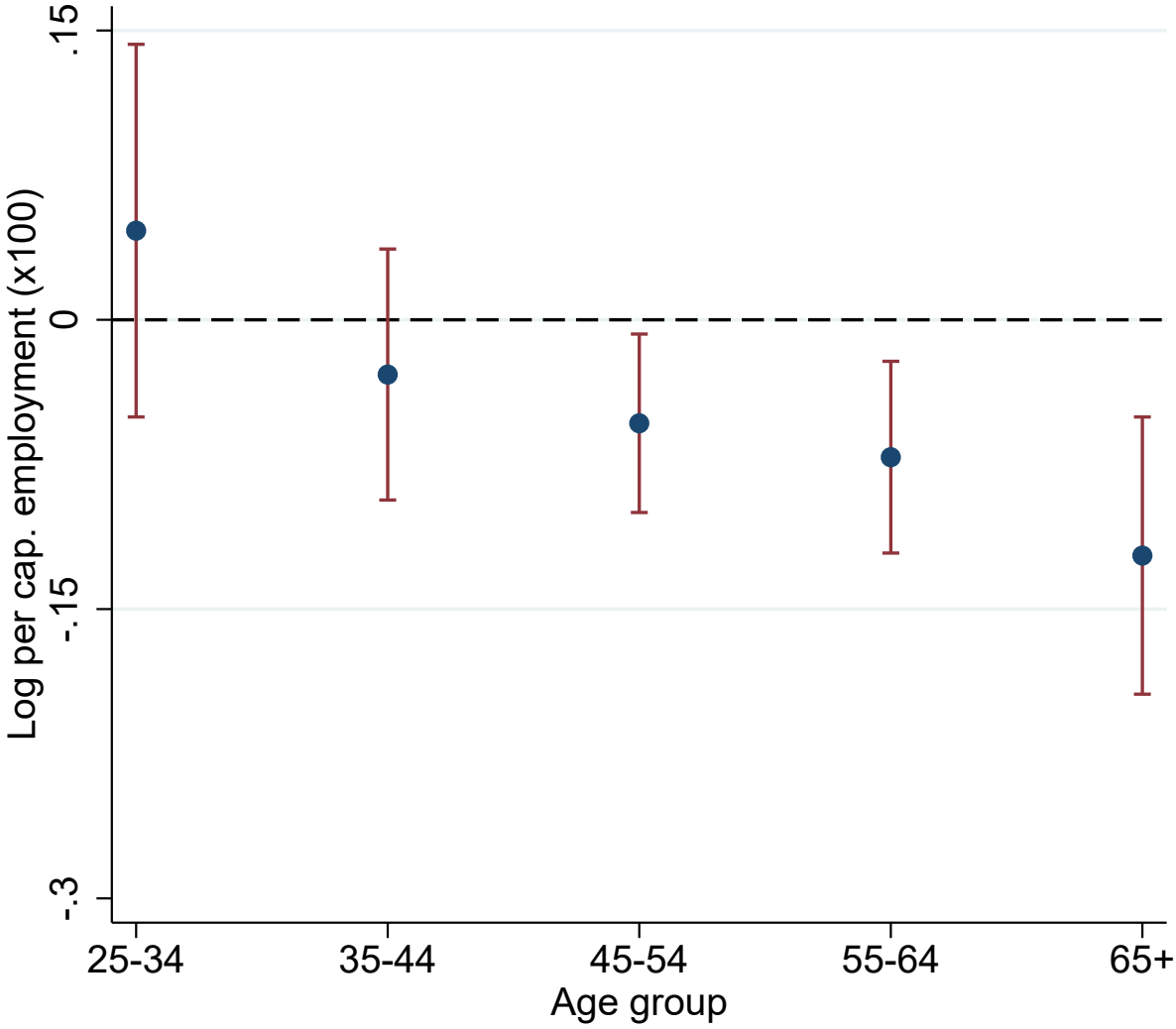
Notes: This map depicts smoke patterns on May 7, 2016 at 9:20 AM. The Fort McMurray fires in Northern Canada can be seen north of Alberta. This large wildfire produces a smoke plume that reaches the upper Midwest. Wildfires in the U.S. Southeast produce plumes reaching Canada. Source: WeatherUnderground.com via WildfireToday.com.

Figure A.2: County Annual Wildfire Exposure 2006 – 2015



Notes: Notes: This figure plots county-level average annual days of wildfire exposure for the lower 48 states for each year from 2006 to 2015. Average exposure during this period was 20.1 days per year. Grayscale shading indicates of smoke exposure: 0 – 1 days (lightest shading), 1 – 3 days, 3 – 10 days, 10 – 33 days, or more than 33 days (darkest shading).

Figure A.3: Annual Employment Effects of Wildfire Smoke: Age Profile



Notes: The figure reports estimated combined effects (current plus lagged effect) of an additional day of wildfire smoke exposure on annual employment per capita separately for different age groups. The dependent variable is the log of per capita employment as measured by QWI, scaled by 100. The focal independent variables capture the number of days in a year on which a county was exposed to wildfire smoke. Coefficients reflect percentage changes in per capita employment per day of smoke in the relevant age group. All regressions include county fixed effects, state-by-year fixed effects, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic daily precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. All regressions exclude observations for years in which a county experienced a wildfire. Standard errors are clustered at both the county and the state-by-year levels.

Table A.1: Annual Migration Effect of Wildfire Smoke

	(1)	(2)	(3)
	Log per capita in-migrants (scaled by 100)	Log per capita out-migrants (scaled by 100)	Log number of tax exemptions (scaled by 100)
Smoke days	0.005	0.003	0.038
	(0.018)	(0.016)	(0.025)
Number of observations	22,829	22,831	22,851

Notes: The table reports estimated effects of an additional day of wildfire smoke exposure on annual migration. Each column corresponds to a separate regression using county-year observations and relevant county population weights. The focal independent variables capture the number of days in a year on which a county was exposed to wildfire smoke. All regressions include county fixed effects, state-by-year fixed effects, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic annual precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. All regressions exclude observations for years in which a county experienced a wildfire. Standard errors are clustered at both the county and the state-by-year levels.

Table A.2: Annual Income Effect of Wildfire Smoke: Alternative Smoke Definitions

	(1)	(2)	(3)
Smoke definition:	Sum of daily coverage	Days with coverage $\geq 75\%$	Days with entire coverage
Smoke days	-0.053**	-0.060***	-0.071***
	(0.022)	(0.023)	(0.024)
Mean annual smoke days	23.8	22.2	20.4
Number of observations	22,851	22,851	22,851

Notes: The table reports estimated effects of an additional day of wildfire smoke exposure on earning. Each column corresponds to a separate regression using a different smoke measure. For example, in column 2, we count days when a county is at least 75% covered in smoke, where 75% is the average coverage rate conditional on any smoke coverage. The focal independent variables capture the number of days in a year on which a county was exposed to wildfire smoke. All regressions include county fixed effects, state-by-year fixed effects, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic annual precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. All regressions exclude observations for years in which a county experienced a wildfire. Standard errors are clustered at both the county and the state-by-year levels.

Table A.3: Annual Wage Income Effect of Wildfire Smoke (including county-years with wildfires)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Dependent variable: log of per capita annual income (scaled by 100)							
Smoke days (year $t+1$)						-0.014	0.001
						(0.015)	(0.015)
Smoke days (current year t)	-0.035	-0.031*	-0.021	-0.040**	-0.036**	-0.036**	-0.033*
	(0.021)	(0.019)	(0.021)	(0.017)	(0.017)	(0.016)	(0.019)
Smoke days (year $t-1$)						-0.038**	-0.031*
						(0.015)	(0.016)
Smoke days (year $t-2$)							-0.015
							(0.012)
Wage data source	QWI	CBP	REIS	IRS	All	All	All
Mean per capita income	19,758	16,525	21,405	21,010	19,829	19,653	19,728
Number of observations	30,615	30,469	30,329	30,691	30,675	24,544	21,472

Notes: The table reports estimated effects of an additional day of wildfire smoke exposure on annual income. Each column corresponds to a separate regression using county-year observations and county population weights. In columns (1)–(4), the dependent variable is the log of per capita annual income as measured by the data source indicated in the bottom panel, scaled by 100. In columns (5)–(7), the dependent variable is the log of the average per capital annual income across all (four) data sources, scaled by 100. The means of per capita income are in 2010 dollars. The focal independent variables capture the number of days in a year on which a county was exposed to wildfire smoke. Coefficients reflect percentage changes in per capita income per day of smoke. All regressions include county fixed effects, state-by-year fixed effects, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic annual precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. Standard errors are clustered at both the county and the state-by-year levels.

Table A.4: Annual Wage Income Effect of Wildfire Smoke: Robustness

	(1)	(2)	(3)	(4)	(5)
Wage data source	QWI	CBP	REIS	IRS	All
Alternative SE clustering	-0.059	-0.080	-0.054	-0.065	-0.071
... county & state×year levels	(0.026)**	(0.029)***	(0.027)***	(0.025)***	(0.024)***
... county & census division×year levels	(0.026)**	(0.031)**	(0.028)*	(0.025)**	(0.025)***
... county level	(0.025)**	(0.026)***	(0.021)**	(0.015)***	(0.017)***
... state level	(0.024)**	(0.038)**	(0.035)	(0.035)*	(0.034)**

Notes: The table reports estimated effects of an additional day of wildfire smoke exposure on annual income. Each column corresponds to a separate regression using county-year observations and county population weights. In columns (1)–(4), the dependent variable is the log of per capita annual income as measured by the data source indicated in the bottom panel, scaled by 100. In column (5), the dependent variable is the log of the average per capital annual income across all (four) data sources, scaled by 100. The focal independent variables capture the number of days in a year on which a county was exposed to wildfire smoke. Coefficients reflect percentage changes in per capita income per day of smoke. All regressions include county fixed effects, state-by-year fixed effects, and weather controls for 10-degree Fahrenheit bins of daily temperature, quadratic annual precipitation, 60-arc degree bins of daily prevailing wind direction, and quintile bins of daily average wind speed. Unless noted otherwise, all regressions exclude observations for years in which a county experienced a wildfire, with standard errors clustered at both the county and the state-by-year levels.

Science of the Total Environment 472 (2014) 421–424

Contents lists available at ScienceDirect

Science of the Total Environment

journal homepage: www.elsevier.com/locate/scitotenv

Exposure to radionuclides in smoke from vegetation fires

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HIGHLIGHTS

- Natural radionuclides in vegetation are in low concentrations.
- Forest fires release natural radionuclides from vegetation and concentrate them in inhalable ash particles.
- Prolonged inhalation of smoke from forest fires gives rise enhanced radiation exposure of lungs especially due to polonium.
- Respiratory protection of fire fighters and members of public is highly recommended for radioprotection reasons.

ARTICLE INFO

Article history:
Received 30 July 2013
Received in revised form 4 November 2013
Accepted 13 November 2013
Available online xxxx

Keywords:
Forest fires
Radioactivity
Polonium
Inhalation
Radiation dose

ABSTRACT

Naturally occurring radionuclides of uranium, thorium, radium, lead and polonium were determined in bushes and trees and in the smoke from summer forest fires. Activity concentrations of radionuclides in smoke particles were much enriched when compared to original vegetation. Polonium-210 (^{210}Po) in smoke was measured in concentrations much higher than all other radionuclides, reaching 7255 ± 285 Bq kg^{-1}, mostly associated with the smaller size smoke particles (<1.0 μm). Depending on smoke particle concentration, ^{210}Po in surface air near forest fires displayed volume concentrations up to 70 mBq m^{-3}, while in smoke-free air ^{210}Po concentration was about 30 μBq m^{-3}. The estimated absorbed radiation dose to an adult member of the public or a firefighter exposed for 24 h to inhalation of smoke near forest fires could exceed 5 μSv per day, i.e, more than 2000 times above the radiation dose from background radioactivity in surface air, and also higher than the radiation dose from ^{210}Po inhalation in a chronic cigarette smoker. It is concluded that prolonged exposure to smoke allows for enhanced inhalation of radionuclides associated with smoke particles. Due to high radiotoxicity of alpha emitting radionuclides, and in particular of ^{210}Po, the protection of respiratory tract of fire fighters is strongly recommended.

1. Introduction

Radionuclides of uranium (^{228}U), thorium (^{232}Th) and actinium (^{235}U) natural radioactive decay series, and non-series radionuclides such as ^{40}K, are present everywhere in the environment (Eisenbud and Gesell, 1997). These radionuclides occur in vegetation together with many other chemical elements absorbed by plants from soils and rocks. In addition to these naturally occurring radionuclides, artificial radionuclides originating in atmospheric nuclear weapon tests and nuclear accidents (UNSCEAR, 2000) are also present. Once released into the atmosphere, such artificial radionuclides were rapidly dispersed by atmospheric circulation and deposited onto vegetation and soils in vast areas around the globe, as recently observed again following the nuclear power plant accident in Fukushima, Japan (Masson et al., 2011; Carvalho et al., 2012). In Europe, this radioactive fallout was particularly noticeable after the nuclear accident of Chernobyl in April 1986, which caused heavy radioactive depositions particularly on forests in Belorussia and Ukraine (Bard et al., 1997; Paatero et al., 2009). Several years later, extensive forest fires in that region resuspended the radioactive cesium (^{137}Cs) from Chernobyl depositions into the atmosphere. This resuspension of ^{137}Cs was detected and measured in aerosols across western European countries and caused some alarm (Paatero et al., 2009).

Much less attention has been paid to the naturally occurring radionuclides present in vegetation, which are released also into the atmosphere by bush and forest fires, although the health impact of fine and ultrafine particle inhalation has been demonstrated (Wichmann and Peters, 2000; Lazaridis et al., 2008; Vos et al., 2009; Carvalho et al., 2011a; Bae et al., 2014). Combustion of plant biomass may be able to enhance the pathways and biochemical availability of those radionuclides to human beings. During the summer period, thousands of hectares are destroyed every year by forest and bushfires around the Mediterranean basin. The concentrations of radionuclides in surface air were determined in the vicinity of vegetation fires to assess their radiological impact on fire fighters and members of the public. A preliminary radiological assessment is presented herein.

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http://dx.doi.org/10.1016/j.scitotenv.2013.11.073

2. Materials and methods

Determination of radionuclides focused on a selection of the more common naturally-occurring alpha and beta emitters such as uranium (^{238}U, ^{235}U, ^{234}U) and thorium (^{232}Th, ^{230}Th) isotopes, radium (^{226}Ra), lead (^{210}Pb) and polonium (^{210}Po) that generally are the main contributors to the absorbed radiation dose in humans (UNSCEAR, 2000). Concentrations of these radionuclides were determined in vegetation samples and in smoke from vegetation fires, which were sampled in close collaboration with fire brigades in the Viseu region, Centre — North of Portugal, during late summer 2012.

Vegetation samples, such as tree trunk wood and leaves, were collected in the absence of fires, samples brought into the laboratory, oven-dried at 60 °C in air, milled, and the dry powder homogenized for analysis.

The sampling of smoke from wild fires was performed in the field, close to bush and forest fires. The temperature of flames, in each locale at the time of sampling, was measured with a hand held infra-red thermometer with laser pointer (Omega, UK). Air sampling was conducted near flames, to obtain freshly produced smoke particles. For this were used portable battery powered aerosol samplers (F&J Specialty, USA), with microfiber glass filters (Whatman, 50 mm in diameter), and a moderate air flux of 60 L min^{-1}. Larger smoke samples, were obtained using stand-alone large volume samplers (F&J Specialty, USA), powered by an electric generator (Yamaha, 10kVA), with microfiber glass filters 110 mm in diameter and an air flux of ca. 1400 L min^{-1}. Other large volume air samplers mounted on a tripod (Andersen), were used with microfiber glass filters 20 cm × 20 cm, and air flux of ca. 1600 L min^{-1}.

The concentration of radionuclides in six size classes of aerosol particles was determined on samples obtained with a Cascade Impactor air sampler (Andersen), filtering 1100 m^3 total volume per sample with an average flow rate of about 1370 L min^{-1}. The Cascade Impactor allowed the collection on Whatman filters of aerosol particles in size classes of] >7.6 μm];]7.6–3.2 μm],]3.2–1.6 μm],]1.6–1.0 μm],]1.0–0.5 μm], and] <0.5 μm].

All filters used for aerosol sampling were weighted before and after filtration, in a humidity and temperature controlled room, to determine the dry load. This was mostly composed of smoke particles from the vegetation fires. Afterwards, filters were used for determination of radionuclides.

The analysis of radionuclides was performed by radiochemistry and alpha spectrometry according to verified procedures (Oliveira and Carvalho, 2006; Carvalho and Oliveira, 2007). Briefly, isotopic tracers (^{232}U, ^{229}Th, ^{224}Ra, ^{209}Po, stable Pb) were added in known amounts to aliquots of samples, in order to be used as internal tracers for the determination of radiochemical yields. Plant and aerosol samples were dissolved in nitric and hydrochloric acids (3:1) and radioelements were separated and purified through ion exchange chromatography columns, both pre-packed (Eichrom) and prepared in the laboratory with Bio-Rad resins. Radioelements were then electroplated on either stainless-steel or silver disks. The alpha particle emission from the disks' surface was measured with ion-implanted silicon detectors in an alpha spectrometer (OctetePlus, EG&G Ortec). Uncertainties associated with the analytical results are the propagated uncertainties for the entire procedure and are given at 1σ significance level.

Quality assurance of the analytical methods was regularly checked by the analysis of IAEA certified reference materials and by participation in international analytical interlaboratory comparison exercises with good results (Carvalho and Oliveira, 2007).

3. Results and discussion

Determination of radionuclide concentrations in bush and tree samples showed that naturally occurring radionuclides are present in low concentrations, generally below 50 Bq kg^{-1} dry weight (dw) and often much lower than 10 Bq kg^{-1} (dw) in trunk wood, in leaves and other aerial plant structures (Table 1). Low radionuclide concentrations are generally expected in plants because these radioactive elements are neither essential elements nor oligo-elements with biochemical functions in plant metabolism and plant growth. Some plants are even classified as excluders due to their ability to prevent root absorption of some radioelements present in soils (Simon and Ibrahim, 1987). Notwithstanding, small amounts of radionuclides can be absorbed by root uptake and accumulated in plant tissues, such as verified particularly in uranium rich areas (Carvalho et al., 2009a, 2009b, 2011b; Gonçalves et al., 2010). Radionuclides formed in the atmosphere from the radioactive decay of atmospheric radon (^{222}Rn), also a member of the uranium series, are brought to the ground with dry and wet atmospheric depositions and thus are intercepted by plant aerial structures. These radionuclides in atmospheric depositions, such as the long lived radon daughters ^{210}Pb and ^{210}Po, can partly be accumulated in plants through foliar uptake (Simon and Ibrahim, 1987; Eisenbud and Gesell, 1997; IAEA, 2010). The low values of ^{210}Po/^{210}Pb activity concentration ratios, as often measured in plant leaves and other aerial plant structures reflect the concentration ratio of these two radon daughters in surface air and atmospheric depositions, typically around 0.1 (Carvalho, 1995; Carvalho et al., 2011a). However, in regions with frequent forest fires the ^{210}Po/^{210}Pb activity ratio in depositions intercepted by the vegetation may be much higher, as observed in results reported herein due to fires that took place in the region before our sampling (Table 1).

The naturally occurring radionuclides contained in vegetation, and in particular those in vegetables and fruits that are part of our diet,

Table 1
Radionuclide concentrations (Bq kg^{-1} dry weight) in plants, ashes from plant combustion, and in smoke free surface air aerosol for comparison, in Viseu district, Portugal, summer 2011.

Samples	^{238}U	^{235}U	^{234}U	^{230}Th	^{226}Ra	^{210}Pb	^{210}Po	^{232}Th
Cistus, bushes	0.56 ± 0.02	0.027 ± 0.003	0.56 ± 0.02	0.57 ± 0.03	2.1 ± 0.2	9.90 ± 0.35	12.0 ± 2.4	0.38 ± 0.02
Oak tree, trunk wood	24.5 ± 0.6	1.18 ± 0.04	24.2 ± 0.6	1.9 ± 0.1	4.9 ± 1.1	–	–	0.112 ± 0.008
Oak tree, trunk wood	22.6 ± 0.8	1.08 ± 0.05	21.8 ± 0.8	1.60 ± 0.07	5.4 ± 0.5	3.27 ± 0.16	5.51 ± 0.02	0.095 ± 0.007
Oak tree, leaves	1.68 ± 0.07	0.08 ± 0.01	1.56 ± 0.07	0.51 ± 0.04	7.4 ± 0.7	17.2 ± 0.4	30.8 ± 1.2	0.29 ± 0.02
Eucalyptus, trunk wood	0.114 ± 0.004	0.0050 ± 0.0006	0.115 ± 0.004	0.023 ± 0.002	0.96 ± 0.13	0.98 ± 0.03	1.68 ± 0.05	0.016 ± 0.001
Eucalyptus, bark	0.29 ± 0.01	0.014 ± 0.003	0.30 ± 0.01	0.089 ± 0.008	26.0 ± 2.4	1.88 ± 0.09	2.60 ± 0.06	0.049 ± 0.007
Eucalyptus, leaves	4.4 ± 0.1	0.21 ± 0.01	4.1 ± 0.1	0.20 ± 0.02	37.3 ± 2.6	10.3 ± 0.4	49.4 ± 2.3	0.08 ± 0.01
Acacia tree, trunk wood	0.020 ± 0.001	0.0014 ± 0.0004	0.018 ± 0.001	0.017 ± 0.001	1.5 ± 0.1	2.04 ± 0.05	4.05 ± 0.15	0.008 ± 0.001
Acacia tree, leaves	13.0 ± 0.3	0.58 ± 0.02	12.8 ± 0.3	5.3 ± 0.3	30.8 ± 1.6	20.27 ± 0.47	8.61 ± 0.33	0.16 ± 0.02
Pine tree, trunk wood	0.105 ± 0.006	0.006 ± 0.002	0.112 ± 0.006	0.067 ± 0.006	1.14 ± 0.06	1.98 ± 0.09	0.97 ± 0.03	0.065 ± 0.006
Pine tree, trunk wood	0.103 ± 0.013	0.0050 ± 0.0047	0.106 ± 0.013	0.014 ± 0.003	0.92 ± 0.09	1.43 ± 0.13	1.53 ± 0.06	0.009 ± 0.003
Pine tree, bark	0.42 ± 0.02	0.019 ± 0.003	0.42 ± 0.02	–	2.7 ± 0.5	2.80 ± 0.08	2.87 ± 0.06	–
Pine tree, leaves (needles)	1.99 ± 0.07	0.13 ± 0.01	1.91 ± 0.07	2.1 ± 0.1	4.9 ± 0.3	10.36 ± 0.31	3.10 ± 0.08	1.10 ± 0.08
Ashes from ground after forest fire	135 ± 4	6.2 ± 0.3	146 ± 4	259 ± 20	477 ± 54	402 ± 6	1115 ± 66	55.7 ± 4.5
Fly ashes (in surface air collected on filter F#6)	347 ± 19	18.0 ± 3.9	372 ± 20	209 ± 13	6144 ± 2908	2070 ± 88	7255 ± 285	203 ± 13
Fly ashes (in surface air collected on filter F#11)	224 ± 12	9.8 ± 2.6	227 ± 12	261 ± 14	5763 ± 1489	923 ± 53	3604 ± 148	95.7 ± 7.1
Aerosol (surface air in absence of fire smoke)	71.0 ± 3.7	2.8 ± 1.0	71.8 ± 3.7	42.4 ± 2.6	–	5895 ± 218	111 ± 7	42.1 ± 2.7

produce an internal radiation dose in humans following ingestion. This radiation dose is part of our natural radiation background, comprising internal plus external radiation, and for the world population it averages about 2.4 mSv per year for members of the public (UNSCEAR, 2000).

Results for radionuclides determined from smoke particles (fly ash) from forest and bushfires sampled on filters, and from bottom ashes collected from the ground after the fire, are shown in Table 1. Measurements revealed relatively high activity concentrations of several radionuclides in the fly ash, in particular ^{226}Ra and ^{210}Po, with concentrations up to 7255 ± 285 Bq kg^{-1} for the latter. Repeated smoke sampling near several wildfires always showed a trend of general increase of radionuclide concentrations in the air with the increase of smoke particle loads in the air. A linear increase was not always observed for the heavier radioactive elements, namely uranium and thorium, but it was consistently observed for ^{210}Po and ^{210}Pb and statistically significant at $p < 0.01$ (Fig. 1). Ash left at the fire ground contained lower activity concentrations of radionuclides than fly ash.

Sampling of surface air aerosols with the Cascade Impactor allowed for the analysis of the radionuclide distribution in several size classes of aerosol particles. The distribution of refractory elements such as uranium (^{238}U) showed a reasonable homogeneity of activity concentration from large particles, >7.6 μm, to small particles]1.0–0.5 μm], but the very fine particles smaller than 0.5 μm displayed the lowest uranium concentrations (Fig. 2). Thorium isotopes showed the same distribution pattern as uranium, with the lowest concentrations in the very fine particles. In contrast to this, polonium activity concentrations were the highest in particles of the smaller size classes]1.0–0.5 μm] and <0.5 μm, and again higher in the largest particles >7.6 μm. On an air volume basis, the largest activity was associated with ^{210}Po in the small size particles, <0.5 μm, while the contribution of ^{210}Po in large particles to total unit volume activity was minor (Fig. 3).

Measurements of the temperature of flames in vegetation fires have shown temperatures up to 700 °C in bush vegetation and above 1000 °C in pine woods. The volatilization point of common polonium compounds is around 390 °C. Some are volatile at even lower temperatures, while for radium compounds volatilization points are likely higher (although not always well known), and certainly are much higher for uranium and thorium compounds (> 800 °C, and even above 1400 °C). Volatilization of uranium and thorium compounds in bush fires, and thus their release into the atmosphere in gaseous forms seems unlikely, but their presences in fly ash particles may occur due to the combustion of organic compounds and reduction of plant mass until only refractory materials are left in the ash. Concentrations of these radionuclides, particularly uranium and thorium, in the air would correspond to the radionuclide content of fly ash particles. In contrast to these elements, polonium compounds and probably some radium compounds, are volatile at lower

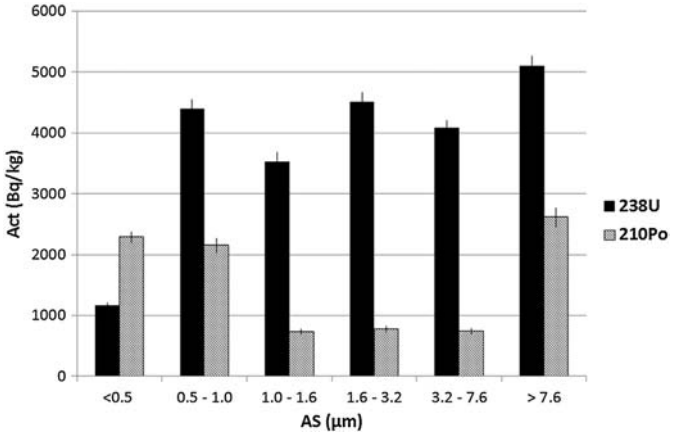

Fig. 2. Radionuclide concentrations as specific activity (Bq kg^{-1}) of several size classes of aerosol particles (AS, aerodynamic size, μm) in the smoke from a forest fire (aerosol load 0.037 mg m^{-3}).

temperatures and may be released by forest fires into the atmosphere in gaseous forms. Polonium atoms in the gas phase are positive ions and can easily be recaptured by electrostatic charges onto aerosol particles (Annunziata, 2007). The capture of gaseous ^{210}Po ions would concentrate this radionuclide preferentially onto the smallest aerosol particles, due to their greater surface area per unit mass.

We hypothesize that the release of radionuclides from plant biomass may occur through two pathways. One is the enrichment of radionuclides in the ash (refractory fraction) with increased reduction of plant material mass due to volatilization of water, organic compounds, and elements of low volatilization point. The other consists in volatilization of radionuclides when flame temperature exceeds their volatilization points. These radionuclides, which include ^{210}Po as the most likely candidate to volatilization, would be released as gas ions and are likely to be captured by electrostatic forces onto aerosol particles, and thus becoming enriched in the smaller particles (<0.5 μm) due to their higher surface/mass ratio.

In the vicinity of vegetation fires, measured activity concentrations were mostly due to ^{210}Po radioactivity associated to the smoke finest particles, averaging 70 mBq m^{-3}. When inhaled, these very fine particles can penetrate deeply into the lung, therefore carrying most of the alpha emitting ^{210}Po, while the larger aerosol particles, >1.0 μm, carrying most of U and Th radioactivity, are largely retained in the upper segments of the respiratory tract.

The radiation dose associated to the deep inhalation of ^{210}Po was calculated assuming an average daily inhalation of 22 m^3 of air and ^{210}Po concentration of 70 mBq m^{-3} leading to a ^{210}Po inhalation of about

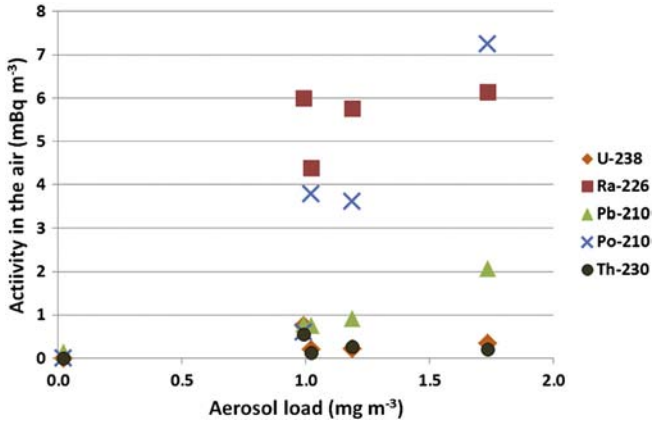

Fig. 1. Radionuclide concentrations (mBq m^{-3}) as a function of aerosol load (mg m^{-3}) in surface air, from an area without smoke to increasing smoke concentrations near forest fires. Linear fits to data points are statistically significant for ^{210}Po and ^{210}Pb at $p < 0.01$, and not significant for other radionuclides.

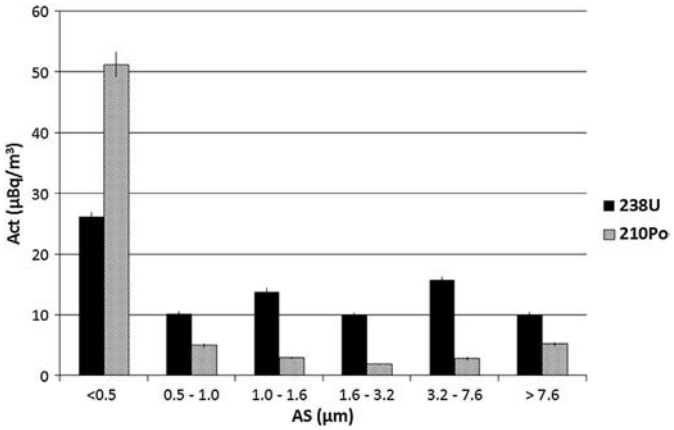

Fig. 3. Radionuclide concentrations per unit air volume (μBq m^{-3}) of several size classes of aerosol particles (AS, aerodynamic size, μm) in the smoke from a forest fire (aerosol load 0.037 mg m^{-3}).

1.5 Bq d^{-1}. Using the dose conversion factor (3.3E-06 Sv Bq^{-1}) recommended by the IAEA (1996) and the EU (EURATOM, 1996), the committed effective dose for an adult member of the public for such ^{210}Po inhalation rate is estimated at about 5.1E-06 Sv d^{-1}.

Similar studies were performed on radioactivity in cigarette smoke (Desideri et al., 2007). It was concluded that ^{210}Po inhaled with the cigarette smoke particles produces exposure of the lung to significantly elevated radioactivity, up to levels able to trigger lung cancer, even without taking into consideration the health effects of other substances present in cigarette smoke. It was computed that the lung of a chronic smoker of one cigarette pack per day, receives from inhaled smoke about 30 mBq of ^{210}Po per day. This activity is about 40 times higher than that for the non-smoker, 0.66 mBq per day from background ^{210}Po in the air, which was measured at about 30 μBq m^{-3} in the Lisbon area (Carvalho, 1995; Carvalho and Oliveira, 2006).

Converting the daily inhalation of ^{210}Po into absorbed radiation dose, the dose from ^{210}Po inhalation in background air is 2.2E-09 Sv d^{-1}, and the dose for the cigarette smoker described above is 9.9E-08 Sv d^{-1}. The radiation dose computed for an adult exposed for 24 h to smoke from vegetation fire, 5E-06 Sv d^{-1}, is about 50 times higher than the effective radiation dose absorbed by the cigarette smoker, and about 2300 times higher than that of the reference person breathing in open air in the Lisbon area.

The relevance of ^{210}Po inhalation at these high concentration levels to human health stems from known facts. Polonium-210 is an alpha emitting radionuclide and it is much more strongly absorbed into the human organism than heavy radioactive elements, such as uranium and thorium (IAEA, 1996). Polonium-210 in man has its main origin in the ingested diet but it is also accumulated from the air through inhalation and lung absorption (Carvalho, 1995). Exposure to high activities of ^{210}Po carried by smoke particles into the lung, due to high ^{210}Po radiotoxicity, may thus represent a serious threat to human health (Harrison et al., 2007; Carvalho and Oliveira, 2009). Another source contributing to enhanced ^{210}Po concentrations in surface air is coal combustion, both in house ovens and fireplaces and in power plants, which may generate relatively low ^{210}Po concentration increase in the air but of a widespread reach, and with potential impact on public health (Gonçalves et al., 2010; Yan et al., 2012).

4. Conclusions

The occurrence of natural radionuclides at very low concentrations in vegetation is part of our natural environment and background radiation exposure. However, this is strongly modified by vegetation and forest fires. The combustion of plants concentrates radionuclides previously contained in a large biomass into the much smaller mass of smoke that is released into the atmosphere and eventually inhaled. Measurements revealed that naturally occurring radionuclides are all in higher concentrations (enriched) in smoke particles compared to the original vegetation. In particular, ^{210}Po concentrations are enriched in the smaller size smoke particles especially in the <0.5 μm particle size fraction, and in concentrations much higher than those of other radionuclides.

Small and inhalable smoke particles become the vehicle for significant radionuclide inhalation. Prolonged breathing of smoke near a vegetation fire, may allow for inhalation of radionuclide activities even higher than in cigarette smoke, and thus cause enhanced lung exposure to radionuclides, especially ^{210}Po. A preliminary assessment of the effective radiation dose indicated that a significant radiation dose exposure may occur, in firemen and members of the population exposed to the smoke plume without protection of the respiratory tract. Even disregarding the potential health effects of other toxic substances present in smoke, and the health effect of fine particles inhalation, radionuclide inhalation alone may be very harmful at the concentrations measured.

From results presented herein, it is concluded that the respiratory tract of fire fighters should be protected, and chronic inhalation of smoke particles avoided. Further investigation is required to better quantify this radiation exposure pathway and to assess its implications for public health.

Acknowledgments

This research was funded by the “Fundação para a Ciência e Tecnologia”, Portugal, Contract Nº. PTDC/AMB/65706/2006. Thanks are due to Christophe I. Burbidge (IST/CTN) for revision of this manuscript.

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Published in final edited form as:
Sci Total Environ. 2018 May 15; 624: 586–595. doi:10.1016/j.scitotenv.2017.12.086.

Wildland Fire Smoke and Human Health

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Abstract

The natural cycle of landscape fire maintains the ecological health of the land, yet adverse health effects associated with exposure to emissions from wildfire produce public health and clinical challenges. Systematic reviews conclude that a positive association exists between exposure to wildfire smoke or wildfire particulate matter ($PM_{2.5}$) and all-cause mortality and respiratory morbidity. Respiratory morbidity includes asthma, chronic obstructive pulmonary disease (COPD), bronchitis and pneumonia. The epidemiological data linking wildfire smoke exposure to cardiovascular mortality and morbidity is mixed, and inconclusive. More studies are needed to define the risk for common and costly clinical cardiovascular outcomes. Susceptible populations include people with respiratory and possibly cardiovascular diseases, middle-aged and older adults, children, pregnant women and the fetus. The increasing frequency of large wildland fires, the expansion of the wildland-urban interface, the area between unoccupied land and human development; and an increasing and aging U.S. population are increasing the number of people at-risk from wildfire smoke, thus highlighting the necessity for broadening stakeholder cooperation to address the health effects of wildfire. While much is known, many questions remain and require further population-based, clinical and occupational health research. Health effects measured over much wider geographical areas and for longer periods time will better define the risk for adverse health outcomes, identify the sensitive populations and assess the influence of social factors on the relationship between exposure and health outcomes. Improving exposure models and access to large clinical databases foreshadow improved risk analysis facilitating more effective risk management. Fuel and smoke management remains an important component for protecting population health. Improved smoke forecasting and translation of environmental health science into communication of actionable information for use by public health officials, healthcare professionals and the public is needed to motivate behaviors that lower exposure and protect public health, particularly among those at high risk.

Graphical Abstract

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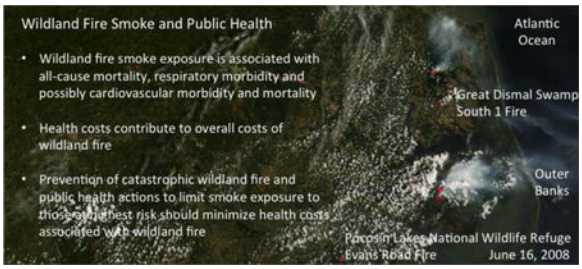
The author declares no competing financial interest.



Keywords

Wildfire emissions; smoke; health effects; air pollution; particulate matter; $PM_{2.5}$

1. Introduction

The natural cycle of wildland fire plays an important role for maintaining the ecological health of the landscape. Yet, the emission and atmospheric transport of combustion products from wildland fire, namely smoke represents a costly and growing global public health problem impacting vulnerable communities, and individuals who are more sensitive to the adverse health effects of smoke exposure. A contemporary estimate of the annual global mortality from landscape fire smoke is 339,000 deaths (Fig. 1, Johnston et al., 2012). Despite this shockingly high estimate, extreme weather events and drought are further increasing the risk of wildland fire and its attendant risks to health (Flannigan, et al. 2009). As the intensity and size of wildland fires increase, so do the associated costs and the vulnerable and at-risk populations adversely impacted by wildland fire smoke. Accordingly, the public health impacts of wildland fire smoke are taking on greater importance and merit the attention of all who have responsibility for land and air quality management decisions and wildland fire policy, who protect the health of the public and at-risk populations, and the stakeholders who are impacted by wildland fire policy. Consequently, the range of decision-makers and stakeholders needed to address this issue is vast and includes local, state, federal and Tribal governments and agencies/offices responsible for land and forest use and fire management, environmental quality and public health. Health care systems, health care professionals, and health insurers as well as city and regional planners should also take an active role in formulating and implementing solutions to mitigate the adverse health impacts of wildland fire emissions.

The review that follows expands on the plenary session titled, “Wildland Fire Smoke and Health: What’s new since the *1st International Smoke Symposium*” presented at the *2nd International Smoke Symposium 2016* in Long Beach, CA on November 15, 2016. The purpose of the plenary session was to communicate the state of our knowledge of the human health effects of wildland fire smoke exposure. The presentation integrated new epidemiological studies and recent systematic reviews of the health effects of wildland fire smoke exposure (Youssouf et al., 2014, Liu et al., 2015, Reid et al., 2016a). This paper also broadens the discussion to consider the health costs of catastrophic wildfire and the role of public health and healthcare professionals for mitigating the adverse health effects of

wildland fire smoke. Such information is critically important for risk assessment, policy analysis, and decision-making regarding air quality, land use and fuel management.

2. Wildland Fire Emissions and Smoke

Wildland fire emissions are complex both physically and chemically, and attendant smoke formation, physiochemical aging and atmospheric transport are influenced by many factors including but not limited to the type of fuel, the type of fire, the characteristics of the landscape, the rate of fuel consumption, and meteorological conditions. The key primary emissions from wildland fires that worsen air quality include ambient air particles such as fine and coarse particulate matter (PM), and gases, carbon monoxide (CO), methane, nitrous oxide (N_2O), nitrogen oxides (NOx), volatile organic carbon (VOC) as well as many other air toxics (Urbanski et al., 2009). Emissions also contain a number of trace metals. Air quality is further affected by the formation of secondary pollutants such as organic aerosols, and ozone generated by the photoreaction of NOx and VOCs in the atmosphere (Jaffe et al. Atmos Environ 2012).

Particulate matter, NO_2, CO, and O_3, are National Ambient Air Quality Standard (NAAQS) criteria pollutants regulated under the Clean Air Act, and the health impacts of these pollutants are well established. Yet, the extent to which air pollutants generated by wildland fires affect health is less well quantified, and the study of such effects represents an emerging and rapidly advancing field. So, while much has been learned over the last decade and will be briefly summarized here, much is still unknown and further research is needed to better define the short-term and long-term impacts of wildfire emissions on health while being mindful of the ecological benefits of wildland fire. Such knowledge is critically important for policy development and decision-making vis-à-vis fuel management that includes prescribed fire, smoke forecasting (Yao et al., 2013), and public health and clinical interventions intended to limit exposure to smoke and protect population health.

3. Health Effects of Wildland Fire Smoke and Wildfire-Related $PM_{2.5}$

Recent systematic reviews (Youssouf et al., 2014; Liu et al., 2015; Reid et al., 2016a) conclude that a strong association exists between exposure to wildland fire smoke or wildfire-$PM_{2.5}$ and all-cause mortality and respiratory morbidity (see Table1). Strong positive associations are present between wildland fire smoke exposure and exacerbations of asthma and COPD, bronchitis and pneumonia (Youssouf et al., 2014; Liu et al., 2015; Reid et al., 2016a). Wildland fire smoke exposure and respiratory mortality do not appear to be associated. The epidemiological data linking wildfire smoke exposure to cardiovascular mortality and morbidity is mixed, and as yet inconclusive. More data is needed to accurately define the risk of cardiovascular health effects including common, life-threatening, disabling and costly clinical outcomes that include myocardial infarction, stroke, heart failure, heart rhythm disturbances, and sudden death. Health data including clinical outcomes collected over much wider geographical areas with larger numbers of exposed people and for longer periods of time would better define the risk of adverse health effects. Additionally, the study of larger and diverse populations will help define the most sensitive populations and the

evaluation of the influence of non-chemical stressors and social factors on the relationship between exposure and the health effects of wildland fire smoke.

Table 1 summarizes the associations between wildfire-PM or smoke exposure and health outcomes based on the most recent critical review (Reid et al., 2016a). Each outcome is listed along with the direction and strength of the association. Recently published studies are provided and are marked as either showing a positive association between wildfire-PM or smoke and the health outcome of interest or no association.

Epidemiological studies have defined populations of individuals who might be at greater risk from the adverse health effects of wildland fire smoke or $PM_{2.5}$. Susceptible populations probably include people with pre-existing respiratory disease, middle-aged and older adults (Lui et al., 2015; Reid et al., 2016a), children, pregnant women and fetuses (Reid et al. 2016a), although not all studies are consistent (Reid et al., 2016b). A recent study provides evidence that risks are greater for older women and African-Americans (Lui et al., 2017b) and those with indicators of lower socio-economic status (Reid et al., 2016b; Lui et al., 2017b).

Since the publication of the most recent systematic review (Reid et al., 2016a) several epidemiological studies have been published further describing the impacts of wildland fire smoke on the health of the exposed population (Table 1, right hand column). Health outcomes associated with wildland fires were described for Colorado in 2012 (Alman et al., 2016), California in 2008 (Reid et al., 2016b), the western U.S. between 2004 and 2009 (Liu et al., 2017b), North Carolina in 2008 (Parthum et al., 2017) and 2011 (Tinling et al., 2016), the northeastern and Mid-Atlantic States of the U.S. (Le et al., 2014) and Boston and New York in 2002 (Zu et al., 2016), Europe in 2005 and 2008 (Kollanus et al., 2017) and Valencia, Spain in 2012 (Vicedo-Cabrera et al., 2016). The findings of these studies further corroborate the conclusions of the previous published systematic reviews. Three recent studies describing the health effects of long-range transport of wildfire related-$PM_{2.5}$ from Quebec, Canada in 2002 (Zu et al., 2016; Le et al., 2014) and the Helsinki metropolitan area, Finland between 2001 and 2010 (Kollanus et al., 2016). No evidence of all-cause mortality was found in either of the studies that examined mortality (Zu et al., 2016, Kollanus et al., 2016). By contrast the study examining the health effects of long-range transported wildfire smoke from the Quebec, Canada wildfires in 2002 (Le et al., 2014) did show positive associations for respiratory and cardiovascular hospitalizations in a Medicare population in the northeastern and Mid-Atlantic States of the United States.

Reid and colleagues (Reid et al., 2016b) studied a long-lived, large wildfire complex in northern California that occurred in the summer of 2008. Daily wildfire-PM exposure was modeled using a data-adaptive machine learning approach with spatiotemporal data sets. The main findings were that for each 5μg/m^3 increase in wildfire-$PM_{2.5,}$ the risk of emergency department visits for asthma [RR=1.06, 95% CI=(1.05, 1.07)] and COPD [RR=1.02 (95% CI=(1.01, 1.04)], and hospitalizations [RR=1.07, 95% CI=(1.05, 1.10)] increased. In this study and in another recent study by Lui and colleagues, effects were more prominent in women, in people living in areas with the lowest median income (Liu et al., 2017b), or among aged adults (Lui et al., 2017a). Rappold and colleagues during the 2008 Pocosin

Lakes National Wildlife Refuge fire in North Carolina also found that poor socioeconomic conditions increased the association between exposure to wildland fire $PM_{2.5}$ and emergency department visits for asthma, and heart failure (Rappold et al., 2012).

Like the previous studies, Tinling and colleagues (Tinling et al., 2016) found positive associations between wildfire-PM and respiratory health effects during the 2012 Pains Bay peat fire in eastern North Carolina, a fire that was located very close to the 2008 Pocosin National Wildlife Refuge Fire. As exposure to wildfire-$PM_{2.5}$ increased, respiratory/other chest symptoms and upper respiratory infections increased in adults and children. The sensitivity of children, particularly those with asthma or rhinitis to the respiratory effects of wildfire-$PM_{2.5}$ were also observed in the Valencia birth cohort when surveyed for health effects after the 2012 wildfire in Valencia Spain (Vicedo-Cabrera et al., 2016). By contrast to the previous studies Tinling and colleagues showed that as daily wildfire-$PM_{2.5}$ increased, hypertension and ‘all-cause’ cardiac outcomes increased on the day of exposure and up to two days after exposure (Tinling et al., 2016). The observation of cardiovascular health effects during the 2012 Pains Bay peat fire in eastern North Carolina largely confirmed the findings reported on the 2008 Pocosin Lakes National Wildlife Refuge peat fire (Rappold et al., 2011; Rappold et al., 2012).

Alman and colleagues (Alman et al., 2016) studied the effect of wildfire smoke on respiratory and cardiovascular emergency department visits and hospitalizations over a 2-day period in Colorado in 2012. Exposure to $PM_{2.5}$ was modeled with a Weather Research and Forecasting Model. $PM_{2.5}$ exposure was associated with asthma and wheeze, and COPD for lag day 0 and with a 2-day or 3-day moving average lag period. Neither the Reid et al. (Reid et al., 2016b) nor the Alman et al. (Alman et al., 2016) study identified an effect of wildfire PM on cardiovascular outcomes.

While the associations between wildfire smoke and cardiovascular effects remain mixed, one study assessing the health impacts of intense wildfires in Victoria, Australia from December 2006 through January 2007 (Haikerwal et al., 2015) is of particular interest. This study showed a positive association between exposure to wildfire-related $PM_{2.5}$ and important clinical cardiovascular events. The major findings included a positive association between wildfire-$PM_{2.5}$ exposure of the total population and out-of-hospital cardiac arrests. Significant positive associations were also observed between wildfire-$PM_{2.5}$ exposure and out-of-hospital cardiac arrests among older adults and men. Increased risk was also observed for hospitalizations for ischemic heart disease and myocardial infarction with statistically significant positive associations for the total population, older adults and women (Haikerwal et al., 2015)(see Fig. 2). Such associations of wildfire $PM_{2.5}$ with health outcomes are very similar to what is known about the health effects of ambient air $PM_{2.5}$ exposure. Another recent study (Kollanus et al. 2016) published after the systematic reviews also provides borderline evidence of cardiovascular morality among the aged, that is those individuals 65 years of age or greater on the day of exposure to smoke related $PM_{2.5}$. The effect estimate was a 13.8% (Confidence interval: −0.6 to 30.4) increase in mortality for each 10$\mu g/m^3$ increase in smoke-affected day $PM_{2.5}$. Wildfire smoke has also been associated with low birth weight (Holstius et al., 2012) when wildfire smoke exposure, as estimated by ambient PM_{10} concentrations occurs during the third trimester.

Many epidemiological studies estimating the health effects of $PM_{2.5}$ (US EPA, 2009) have identified the populations most susceptible to the adverse health effects of $PM_{2.5}$ as individuals with pre-existing respiratory and cardiovascular disease, adults 65 years of age and older, populations with lower socio-economic status, children and developing fetuses. Other populations suspected to be at greater risk include people with chronic inflammatory diseases (e.g., diabetes, obesity) and those with specific genetic polymorphisms (e.g., GSTM1) that mediate physiologic response to air pollution. To date no data has been presented to suggest higher risk among those with diabetes, obesity or specific genetic polymorphisms. Future research should consider such possibilities.

So given that the young, the old and people with respiratory and cardiovascular diseases are possibly at higher risk, just what percentage of the U.S. population do these conditions represent? And how many people might be potentially at-risk in the population. Currently in the United States 14.9 percent of the population is over the age of 65 years (U.S. Census Bureau, 2017a), and 22.8% are children (less than 18 years old)(U.S. Census Bureau, 2017b). Heart disease and lung disease also contribute to the population at-risk with heart disease, accounting for about 28.4 million (11.7%)(US CDC, 2015a) and COPD 12.8 million (5.3%) (CDC, 2015b). Asthma adds an additional 24.6 million (9%) persons (CDC, 2015b). There are about 4 million births in the U.S. each year (Martin et al., 2017). Consequently, even though the relative risks of wildfire-$PM_{2.5}$ and smoke are small, the very large number of at-risk individuals across the U.S. has the potential to produce a large number of serious adverse health outcomes during wildland fire events.

The reader should also keep in mind that wildland fire smoke or wildfire-related $PM_{2.5}$ while serving as a metric of exposure represents only a surrogate of the actual aerosols to which populations are exposed and is not likely to fully characterize the exposure to toxic constituents of the atmosphere unique to each wildland fire. The incomplete characterization of exposure might in part explain the inconsistencies in the epidemiological data particularly for cardiovascular outcomes. Apart from methodological limitations related to study design, exposure misclassification, and statistical power, a real consideration is that differential toxicity of wildland fires emissions based on the multitude of conditions that modify the chemical characteristics of such emission might contribute to these inconsistencies.

4. Mechanisms Proposed to Explain Health Effects

Substantial progress has been made over the last decade identifying the key biological pathways accounting for health effects of inhaled particulate matter and gases (Newby et al., 2015). Currently there are three principal pathways supported by epidemiological, clinical and toxicological data that explain the observed biochemical, physiological and clinical effects of air particulate pollutant exposure. First, inhaled particulates can react with neural receptors in the lung and activate a reflex involving chemical and electrical communication between the lung and the nervous system. Return signals from the brain traveling through the autonomic nervous system result in increases in blood pressure and changes in heart rhythm. Second, air pollutants interact with the alveolar-capillary cells generating oxidative stress reactions and local and systemic inflammatory responses. The consequences of these responses include decreased nitric oxide availability, oxidation and alteration of the function

of blood lipids, platelet activation, and prothrombotic changes in blood proteins that affect the function of blood vessels and increase the likelihood that blood will clot. Third, the smallest fraction of particulate matter, the ultra-fine fraction, defined as particles less that 0.1 μm, can translocate across the alveolar membrane and move systemically acting at a distance from the lung. The biochemical and physiological responses contribute to a number of functional changes including endothelial dysfunction, endothelial activation, and injury. Local changes in the lung promote pulmonary responses affecting airway function, and resistance to viruses and bacteria increasing the risk of infection, for example upper respiratory tract infections, bronchitis, and pneumonia. Systemically, the sequence of biochemical and physiological changes associated with urban-$PM_{2.5}$ increases the risk of cardiac ischemia, and acute coronary syndrome, stroke, arrhythmia and heart failure, yet such outcomes have not been established with certainty after wildland fire smoke exposure suggesting that chemical differences might yield differential health effects. Should cardiovascular health effects ultimately be confirmed, the mechanism of such effects is likely to be similar to that described for urban-PM.

Limited toxicological data exists that can relate directly to epidemiological observations. However, Kim and colleagues (Kim et al., 2014) studied the differential pulmonary and cardiovascular system effects in an *in vivo* study in mice in response to PM collected during the 2008 Pocosin Lakes National Wildlife Refuge fire in eastern North Carolina, a fire extensively studied for its health effects by Rappold and colleagues (Rappold et al., 2011; Rappold et al., 2012). PM was collected during the smoldering and “glowing and nearly extinguished” phase of the fire and the PM was divided into fine and coarse fractions. Lung and systemic markers of injury and inflammation were measured after oropharyngeal aspiration of 100 μg PM/mouse. Fig. 3 shows the toxicological responses of the lung tissue to fine and coarse wildfire-PM, and provides direct evidence of the differential biological effects of the two size fractions of PM in lung tissue. Coarse particle exposure causes inflammatory effects in the lung driven by endotoxin, a constituent of bacterial cell walls and present in coarse PM. Exposure to the fine PM fraction did not produce pulmonary effects in the mouse, but did produce cardiovascular effects (Kim et al., 2014).

The toxicological findings in this study (Kim et al., 2014) provide some biological plausibility for the increase in emergency department visits for pulmonary and cardiovascular health outcomes among those exposed to emissions from the Pocosin Lakes National Wildlife Refuge fire in 2008 (Rappold et al., 2011). The toxicological studies further suggested that cardiovascular effects might be mediated by the wildfire smoke’s fine fraction of PM, while the pulmonary responses appear to be related to coarse PM’s endotoxin content. The possible role of endotoxin as a biologically active component of the coarse fraction of wildfire smoke was strengthened by a controlled human exposure study of 50 healthy adults who were exposed to concentrated ambient fine and coarse PM exposure at 200 $\mu g/m^3$ for 130 minutes (Zhong et al., 2015). Inhalation of coarse PM caused increases in blood pressure and heart rate and the response was associated with the endotoxin and ß-1,3-d-glucan content of the coarse PM. While wildfire smoke was not the specific source of the concentrated air particles used in the controlled human exposure study, the observations taken within the context of the *in vivo* murine study implicates endotoxin as an active component of coarse particles affecting biological responses in man.

5. Hidden Costs of Wildfires

Federal wildfire suppression costs have quadrupled between the late 1980s and the last five years (https://www.fs.fed.us/sites/default/files/2015-Fire-Budget-Report.pdf), because of the location of wildfires, the increasing size and intensity of fires, the expansion of the wildland-urban interface, and base camp and personnel support. Such costs are straining the federal wildfire suppression budget (https://www.usda.gov/media/press-releases/2017/09/14/forest-service-wildland-fire-suppression-costs-exceed-2-billion) and highlight the importance of land use and fuel management decisions. However, these costs only partially account for the total costs of catastrophic wildfires. Wildfires are also associated with many other costs including those associated with premature mortality (Johnston et al., 2012; Rappold et al., 2014; Kollanus et al., 2017), health care utilization, lost productivity, impacts on the quality of life (Jones, 2017b), compromised stream, river and drinking water quality (Hohner et al., 2016; Bladon et al., 2014), and damage to critical infrastructure. Estimates of direct and indirect costs of wildfire to health related costs are emerging as new and important sources of data to be considered when making fuel management and land use decisions. Such estimates also have the potential to be used to estimate health costs associated with fuel management approaches such as prescribed fire that provide ecological benefits and mitigate catastrophic wildfires (Fernandes et al., 2003), yet impair air quality.

The 2008 wildland fire in the Pocosin Lakes National Wildlife Refuge in eastern North Carolina (Fig. 4) offered an opportunity to estimate the total health costs of a wildland fire that burned more than 40,000 acres of peat bogs over 202 days. Suppression costs of this fire totaled $20 million, required 2 billion gallons of water and over 400 people to control the fire (Rappold et al., 2011). Health cost estimates based on excess adverse health impacts and deaths attributable to smoke exposure were $48.4 million. Health cost estimates were based on 4 to 5 premature deaths, 31 non-fatal heart attacks, 41 episodes of bronchitis, 810 asthma attacks, 530 lower respiratory symptoms, 769 upper respiratory symptoms and 3,700 work days lost. The cost of excess emergency department visits for asthma and heart failure alone were estimated to be $1 million (Rappold et al., 2014). Therefore, in this example the estimated costs associated with deaths, lost workdays and healthcare costs were two-fold higher than the costs of suppression. The health costs were also estimated for peat fires in the Great Dismal Swamp that occurred in 2008 in Virginia (Parthum et al., 2017). The clinical observations noted in Table 1 were similar to those observed by Rappold et al. (Rappold et al., 2011). The estimated health costs attributed only to morbidity were $3.69 million (Parthum et al., 2017) with fire suppression costs exceeding $10 million (https://fws.gov/refuge/Great_Dismal_Swamp/what_we_do/firesuppression.html). While the cost attributed to the peat fires of Virginia and North Carolina during 2008 was high, the size of these fires is dwarfed by the catastrophic wildfires of the west. In a third example, Jones and Berrens (Jones et al., 2017a) estimated the health costs of morbidity and morality associated with $PM_{2.5}$ smoke in the Western US between 2005 and 2015. Their analysis suggests that wildfire smoke in the Western US contributes as much as $165 million in annual health costs attributable to morbidity and mortality (Jones et al., 2017a). Fann and colleagues estimated the morbidity, mortality and economic burden of wildland fires for the continental U.S. between the years 2008 and 2012(Fann et al., 2018). The economic burden of these fires

over the five years studied ranged from $63 million (95% CI:$6-$170 million) for short-term exposures, to $450 million (CI: $42 - $1,000 million) for long-term exposures.

6. Population Vulnerabilities

The 24-hour NAAQS for $PM_{2.5}$ and PM_{10} are established to protect the health of the public during short-term exposure to ambient PM, whereas the annual $PM_{2.5}$ standard is intended to provided public health protection against long-term exposures. As has been described, large wildland fires generate massive emissions into the atmosphere over a short period of time and increase local and even distant ambient air PM concentrations that exceed the 24-hour PM standard and are associated with adverse health effects in the most sensitive populations (Navarro et al., 2016).

While it is easy for one to appreciate that wildland fires produce short-term increases in ambient PM, less well appreciated is the fact that wildland fire and prescribe burning of the landscape contribute substantially to the average annual $PM_{2.5}$. Figure 5 was adapted from a recent paper by Rappold and colleagues (Rappold et al., 2017) and illustrates the important point that wildland fires contribute to both short-term and long-term increases in ambient air particle pollution. The annual average daily fire-$PM_{2.5}$ footprint by county in the U.S. between 2008 and 2012 is shown in the left-hand panel of the map in Fig. 5. Large fire perimeters are shown in black. The figure shows that wildland fire smoke contributes substantially to total PM in some areas of the United States. By contrast, the right-hand panel of Fig. 5 shows the number of days by county with wildfire-$PM_{2.5}$ above the 24 hour EPA NAAQS of 35 $\mu g/m^3$. Interestingly, while the southeastern and western US wildland fires produce similar elevations of the annual average $PM_{2.5}$ concentrations (Fig. 5, Left hand panel), the number of days exceeding the 24-hour average $PM_{2.5}$ NAAQS is far fewer in the southeast where prescribed fire is used to a greater extant (Fig. 5, Right hand panel).

The maps depicted in Fig. 5 illustrate the non-uniform spatial distribution of wildland fire smoke exposure across the United States. Likewise, the health characteristics of the population are also distributed unequally across the landscape. For example, Fig. 6 shows the spatial distribution across the U.S. of chronic obstructive pulmonary disease, a population known to be at increased risk for the adverse health effects from wildfire smoke. The prevalence of COPD is greater in the eastern United States. When simultaneously considering the distribution of populations at risk and the distribution of wildfire smoke exposure it becomes apparent that some regions of the U.S. are predicted to have a higher likelihood of adverse health effects during wildland fire events.

The Community health-vulnerability Index (Rappold et al., 2017, see Fig. 7) provides an index at the county-level based on factors that increase risk such as respiratory and vascular disease, age, diabetes and obesity and SES parameters as well as the magnitude of annual exposure from the distribution of wildfire-$PM_{2.5}$ as shown in Fig. 5. The map is instructive because it shows that the county-level risks for adverse health impacts are greatest in the southeastern United States. Even though the western states experience more large fires, the presence of a healthier population appears to lower the overall risk when compared to the counties in the southeastern and mid-Atlantic states.

7. Public Health Guidance and Interventions

One of the potential benefits of the Community Health-Vulnerability Index (Rappold, et al. 2017) is that its use could increase awareness of local wildland fire health risks among public health officials and healthcare professionals responsible for the wellbeing of the affected communities. Researchers in the U.S. Center for Disease Control and Prevention's National Center for Environmental Health are currently developing a similar tool for integration into the Environmental Tracking Network as a national online tool for identifying at-risk populations, and providing public health guidance during wildland fire events (Vaidyanathan et al., 2017). Increased awareness of health risk is more likely to prompt public health officials and health care professionals to advise at-risk individuals to take action to avoid exposures to wildland fire smoke. One source of readily available and up-to-date information to assist public health and health care professionals is the "Wildfire Smoke: Guide for Public Health Officials". The guide is a comprehensive summary of information available at the US EPA's AirNow.gov website (URL: ttps://www3.epa.gov/airnow/wildfire_may2016.pdf) to assist public health officials to prepare for wildland fire smoke, and provides information to share with the public to protect them during such events. The guide offers specific strategies to reduce smoke exposure as well as how to communicate particulate matter concentrations and recommendations for public health action.

While recommending interventions to limit exposure to wildland fire smoke is prudent, particularly among those at highest risk, studies are needed to evaluate the benefits of such interventions. Considering the available data portable air filters have been advocated as a public health response to wildfire smoke (Barn et al., 2016). Fisk and Chan (Fisk and Chan, 2016) further advanced this concept by estimating the health benefits expected of in-home interventions that improve the quality of the indoor air during wildfires. Their analysis indicated that particle filtration in the homes of people at highest risk from the adverse effects of smoke is expected to be economically beneficial (Fisk and Chan, 2016).

A study conducted in Taipei between 2013 and 2014 tested the effect of air home air filtration on biomarkers of inflammation, oxidative stress and blood pressure. The crossover intervention study showed that increased exposure to $PM_{2.5}$ and total VOCs increased inflammation, oxidant stress and blood pressure, and that in-home air filtering decreased biomarkers of inflammation, oxidative stress and the acute phase reactant fibrinogen (Chuang et al., 2017). This study provides biological plausibility for the modeled outcomes predicted by Fisk and Chan (Fisk and Chan, 2016).

It is imperative that effective public health communication strategies be developed in conjunction with communities, public health officials, health care professionals and state officials because the public health impacts of wildland fire smoke will continue to increase (Liu et al., 2016).

8. Knowledge Gaps and Research Opportunities

To effectively study the health effects of wildfire smoke or wildfire-related PM requires three key components: (1) an adequate assessment of exposure, (2) the availability of

reliable data for the health metrics of interest, and (3) an exposed population of sufficient size and exposure gradients to have the statistical power to yield estimates of the association of exposure-health outcome having statistical confidence. Prior to the last decade, epidemiological studies of the health effects of wildland fires were very limited because the occurrences of wildland fires are for the most part unpredictable and occur in sparsely populated wilderness or rural areas that are not typically included in the ambient air pollutant monitoring network.

At a minimum, exposure assessment requires the location, the pollutants present at that location and their concentration, and the duration of exposure. Exposures have been estimated by a number of methods that include comparison of smoky versus non-smoky days, monitored PM, modeled PM, satellite indicators of smoke, temporal and spatial comparisons, and integration of PM monitoring, statistical modeling and satellite imaging (Reid et al., 2016a). Importantly, health effect estimates can be affected by the methods used to estimate exposure (Gan et al., 2017). Thus, the accuracy of the measured relationship between wildfire emissions and health effects depends on our ability to accurately estimate the exposure of an individual to one or more pollutants. More research is needed to integrate measurements and modeling to improve exposure assessments. Ideally, such assessments would also include both indoor and outdoor exposures, the constituents of the emissions and activity.

A more detailed and realistic assessment of the health impacts of wildfire smoke exposure has been made possible by the recent technological advances such as estimates of the temporal and spatial distribution of wildfire-PM concentrations and exposure afforded by satellite imaging of aerosol optical density, and the integration of satellite-based data with other sources of directly measures or modeled air pollution data (van Donkelaar, et al., 2010), GIS coding of residences of exposed individuals, and the availability of large health data sets to test associations. A literature search in Pubmed.gov of the terms “(((wildland fire or wildfire or forest fire or prescribed fire))) AND ((air pollution or particulate matter or smoke or $PM_{2.5}$))) AND health” identified 207 publications between 1990 and November 21, 2017, of which 176 were published during or after 2006. The dramatic increase in epidemiological studies over the last decade highlights just how recent the developments in exposure modeling and epidemiology have allowed the study of the relationship between wildfire smoke and health effects.

Health outcomes have been expressed in many different forms and include: mortality, hospitalizations, emergency department visits, physician office visits, and medication usage (Reid et al., 2016a). Figure 8 illustrates the wide variety of health effects attributed to wildfire smoke density or wildfire-PM. Responses range from asymptomatic subclinical biological and physiological responses affecting a large number of exposed individuals to smaller numbers demonstrating worsened clinical manifestations requiring medication use, healthcare system utilization, and even death. Associations between wildfire smoke density or wildfire-PM and health effects are best defined for mortality, hospitalizations, and emergency department utilization because of the accessibility of such health data. On the other hand an assessment of the full public health impact and societal burden of wildfire

emissions are not known because data corresponding to symptoms contributing to impaired function, discomfort, lost work and productivity is not readily available.

As we look the future, satellite imaging and atmospheric modeling of smoke concentrations are anticipated to improve. Other pollutants are likely to be included offering opportunities to explore co-pollutant interaction. Lower cost, portable PM monitors are expected to be available for deployment to sample real-time ground-level concentrations and increase the spatial resolution of ground-based $PM_{2.5}$ measurements and enhance the value of integrated exposure models. Availability of more health information perhaps through access to electronic health records will further increase the temporal and spatial resolution and statistical power of the epidemiology studies.

More large-scale epidemiology studies will help better define health effect estimates and the public health burden of wildland fires with particular attention to common and costly health conditions such as heart and vascular disease and vulnerable populations. Such large-scale epidemiology studies also have an important role to play when comparing the aggregated health effects of large and less frequent wildland fires to small and more frequent prescribed fires.

Studies are needed to evaluate the health and economic benefit of interventions intended to decrease emissions and exposure. Mitigation of the health effects caused by exposure to smoke includes management of emissions as well as avoidance of exposure. For example does smoke management during prescribed fire mitigate the adverse health effects of wildland fire by optimizing the timing and environmental conditions at the time of the fire? To achieve the best outcomes of interventions, the public and especially those individuals at greatest risk must understand their risk and be willing to take action to avoid smoke exposure. Does increasing awareness among those at greatest risk motivate behavioral actions that limit exposure and mitigate adverse health impacts? Research in the social sciences is needed to better understand the modifying effects of non-chemical and social stressors or conditions and use this information to develop more effective communication strategies (Olsen, et al., 2014) that will increase awareness and willingness to take actions to protect health (Wells et al., 2012). It is important to learn what people are willing to do to decrease their risk from air pollution accompanying wildland fire. Are they willing to stay indoors, use in-home HEPA filtering, and wear an N-95 respirator when outdoors? Will pharmacological or dietary supplements taken before and during exposure curtail the health effects of wildland fire smoke? Health care professionals, hospital systems, and even health insurers also have a role to play in increasing the awareness of their at-risk patients about actions they can take to limit exposure to smoke from landscape fire.

9. Final Comments

In summary, exposure to wildfire emissions is an important and growing public health and clinical problem affecting tens of millions of people in the United States. Changing weather patterns including drought are increasing risks of wildland fire and risks of co-morbidity. Populations at greatest risk include people with chronic lung disease, older individuals, children, pregnant women and fetuses. Individuals with pre-existing heart disease are also

likely to be at increased risk. An expanding wildland-urban interface and an aging U.S. population are increasing the size of vulnerable and sensitive populations. Studies are now providing estimates of health costs associated with wildland fire events and such information will likely be very valuable when considering the relative benefits of various land management policies and practices to prevent catastrophic wildland fires. Yet, knowledge gaps persist and require ongoing research related to population health effects. Effective smoke management is an important component of maintaining population health. Better communication of actionable information by public health officials and health care professionals is needed to more effectively improve the response of the public particularly those at highest risk.

For more information regarding wildfire smoke, wildfire science and health visit the following websites: EPA AirNow (www.airnow.gov) and Fires: Current Conditions webpage (https://airnow.gov/index.cfm?action=topics.smoke_wildfires), U.S. Forest Service, Wildfire (www.fs.fed.us/managing-land/fire), Centers for Disease Control and Prevention, Wildfire Smoke (www.cdc.gov/disasters/wildfires/smoke.html), NASA, Wildfire and Smoke (https://www.nasa.gov/mission_pages/fires/main/index.html), NOAA, Smoke Forecasting System (http://www.arl.noaa.gov/smoke.php), National Interagency Fire Center (www.nifc.gov), and Joint Fire Science Program (www.firescience.gov), and Department of the Interior Office of Wildland Fire (https://www.doi.gov/wildlandfire).

Acknowledgements:

The review was supported by the U.S. Environmental Protection Agency, Office of Research and Development.

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Highlights

1. Wildland fire smoke exposure is an important and growing risk to public health.
2. The size of the population at-risk from wildland fire smoke is increasing.
3. Averting catastrophic wildfire and personal exposure will likely improve health.
4. Stakeholder cooperation is needed to limit the health effects of wildland fire.
5. Research is needed to assess the health benefits of avoiding smoke exposure.

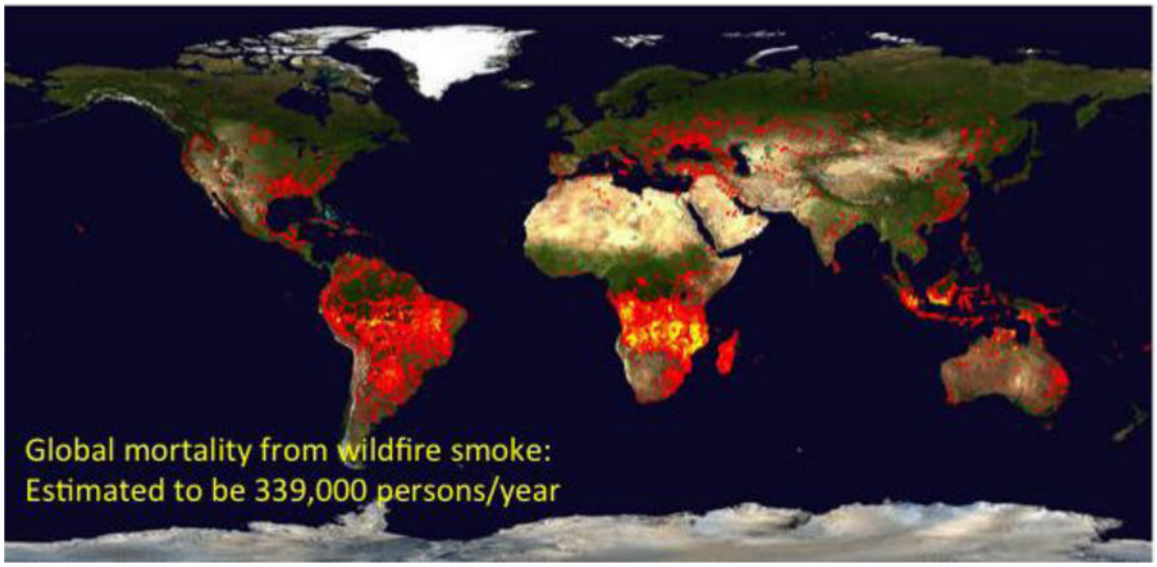

Figure 1. Global fire map corresponding to September 8 to 17, 2015.

The global fire map (https://lance.modaps.eosdis.nasa.gov/cgi-bin/imagery/firemaps.cgi?period=2015251–2015260) reports the location of fires detected by MODIS (Moderate Resolution Image Spectroradiometer) on board NASA's Terra and Aqua research satellites and shows the global expanse of wildfires. Each colored dot indicates a location where MODIS detected a fire during the 10-day period. Red dots indicate low fire counts, whereas yellow dots indicate larger numbers of fires. Global mortality from wildfire smoke is estimated to be 339,000 deaths annually (Johnson et al., 2012).

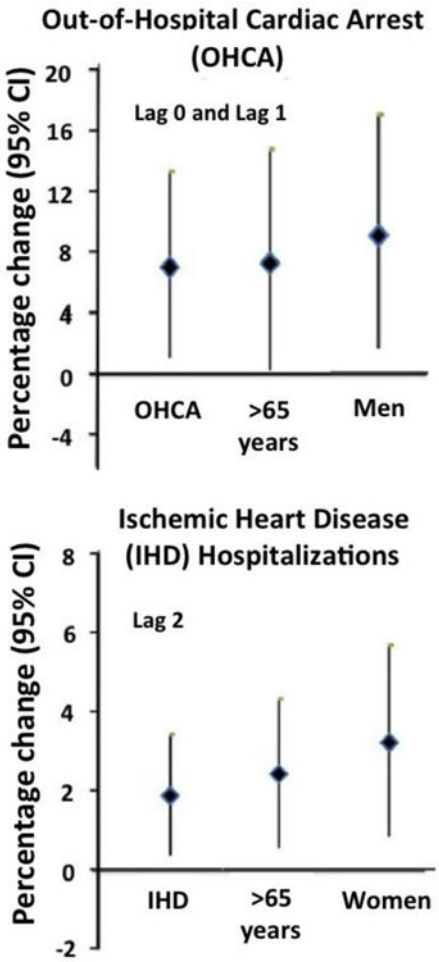

Figure 2. Cardiovascular health effects during wildfires in Victoria, Australia, December 1, 2006 to January 31, 2007.

The upper panel shows the percent change in out-of-hospital cardiac arrest (OHCA) and the lower panel ischemic heart disease (IHD) hospitalizations for a 9μg/m^3 interquartile range increase in wildfire-$PM_{2.5}$ exposure. Lag 0: effect occurs on the day of exposure. Lag 0 to 1: Health effects occur on the day of exposure and a day after exposure. [Adapted from Haikerwal et al., 2015].

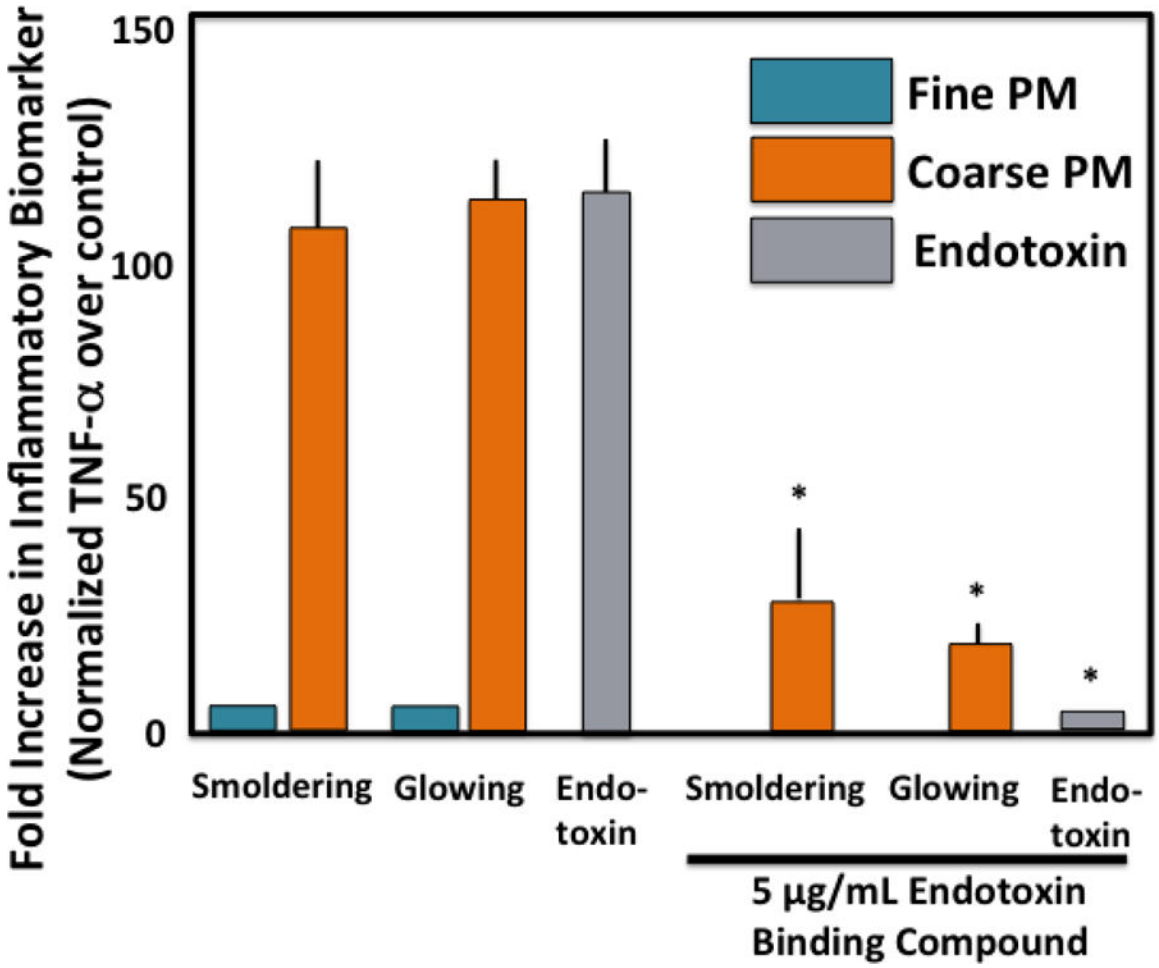

Figure 3. Toxicology of Wildland Fire Emissions.

Murine lungs were exposed to fine (blue bars) and coarse (orange bars) PM collected at the 2008 Pocosin Lakes National Wildlife Refuge fire and endotoxin in the presence and absence of polymixin B (PMB) an antibiotic that binds endotoxin and blocks the effect of endotoxin, a key component of the outer membrane of Gram-negative bacteria. Murine lungs responded with an inflammatory response to exposure to endotoxin (Gray bar) as shown by the increase in the pro-inflammatory cytokine TNF-alpha. The effect of endotoxin was blocked by the addition of polymixin B. Coarse PM (orange bars) induced a pro-inflammatory response as indicated by the increased TNF-alpha, an effect blocked by polymyxin B. Inhibition of the TNF-alpha response by polymyxin B confirms that endotoxin probably plays a role in the inflammatory response induced by the coarse PM fraction. By contrast fine PM (blue bars) did not induce an inflammatory response in this model. (Kim et al., 2014)

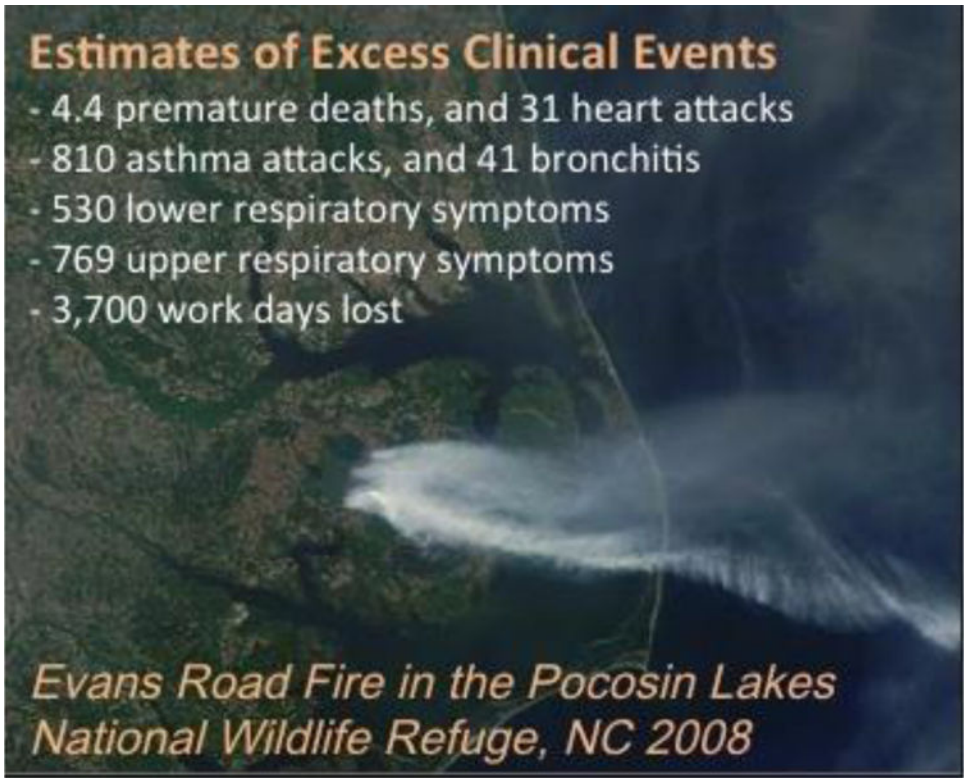

Figure 4. Satellite image showing the location of Evans Road Fire in the Pocosin Lakes National Wildlife Refuge, NC in 2008.

www.fws.gov/pocosinlakes/news/ERF/news-erf-out.html

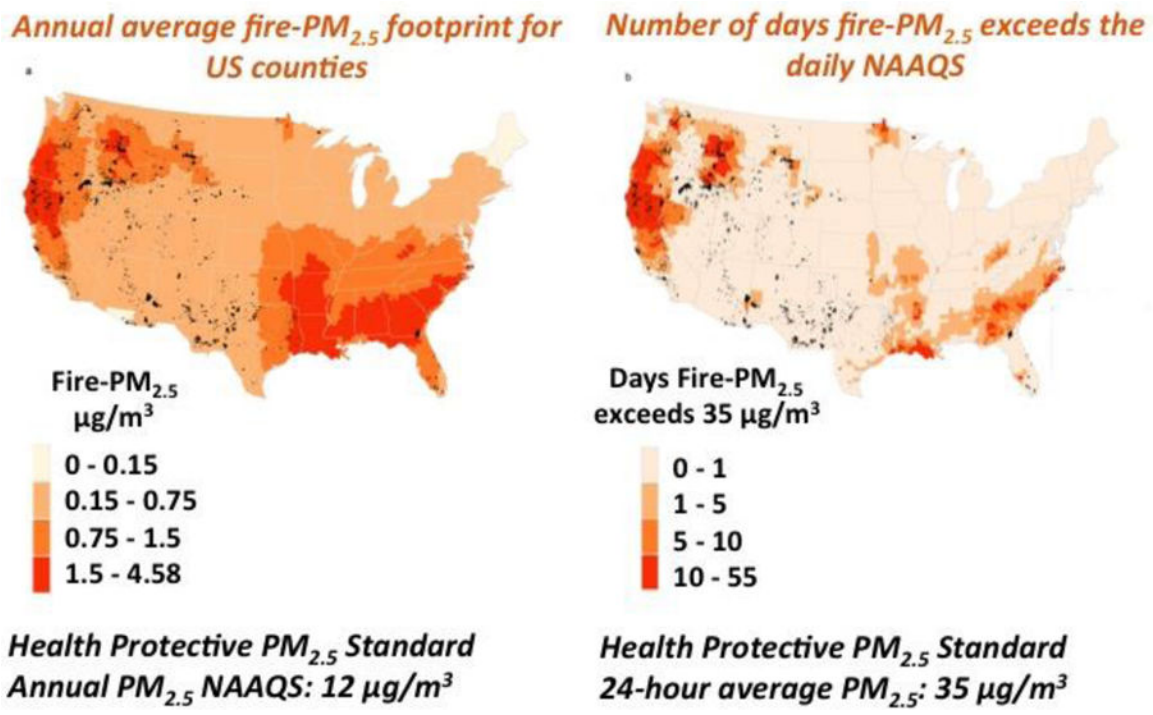

Figure 5.

Left-hand panel. Annual average daily fire- $PM_{2.5}$ footprint by counties of continental U.S. and perimeters of area burned by large fires in black between 2008 and 2012. Right-hand panel. Number of days with fire-$PM_{2.5}$ above 35 µg/m^3 by counties of continental U.S.. Adapted from Rappold et al., 2017.

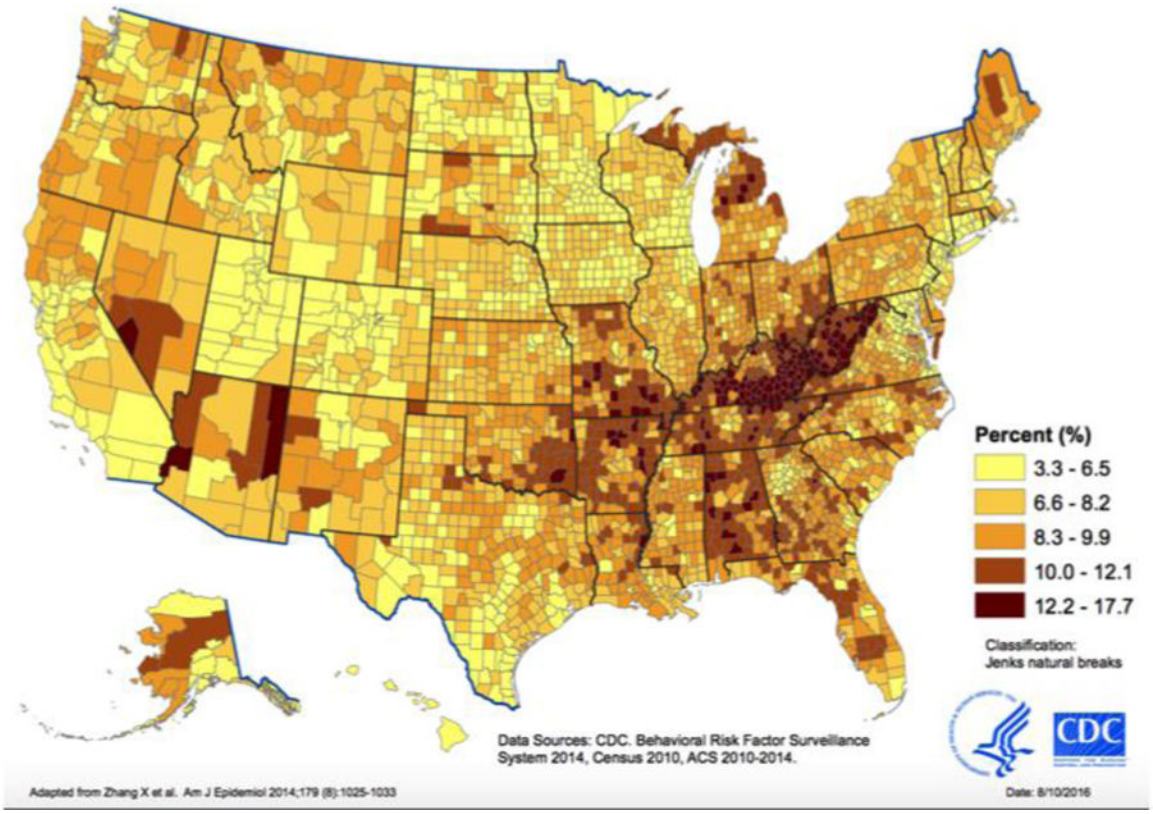

Figure 6. Chronic obstructive pulmonary disease prevalence by county in the U.S. in 2014.
Age-standardized prevalence of chronic obstructive pulmonary disease (COPD) among adults aged ≥ 18 years. Source: CDC, Behavioral Risk Factor Surveillance System, 2014, Census 2010, ACS 2010–2014. www.cdc.gov/copd/pdfs/COPD_cnty2014_saeColor_2.pdf

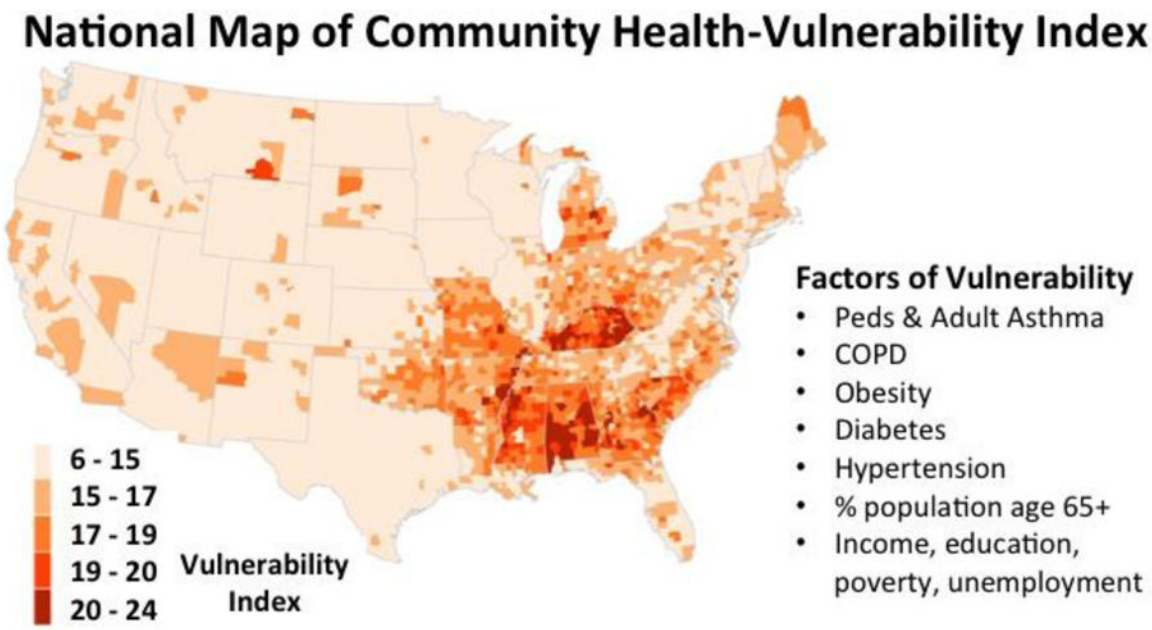

Figure 7. Community Health-Vulnerability Index
Adapted from Rappold et al., 2017.

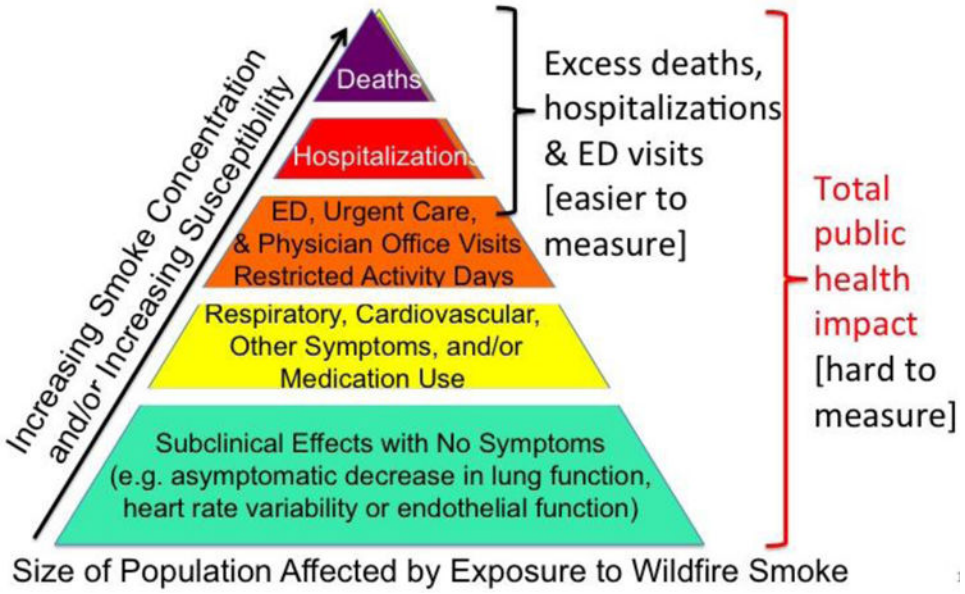

Figure 8.
Clinical and Sub-Clinical Impacts of Wildfire Smoke or $PM_{2.5}$

Table 1.

Associations between Wildfire-PM or Smoke Exposure and Health Outcomes

Outcome	Direction of Association	Strength of Evidence	New Studies Published Since Reid et al., 2016a
Mortality			
All	Increased	Strong	− Zu et al., 2016, − Kollanus et al., 2016
Respiratory	No assoc.		− Zu et al., 2016, − Kollanus et al., 2016
Cardiovascular	Increased	Inconclusive	− Zu et al., 2016, ± Kollanus et al., 2016 *
Morbidity			
Respiratory	Increased	Very Strong	+ Liu et al., 2017a; Lui et al., 2017b; + Tinling et al., 2016; + Reid et al., 2016b; + Parthum et al., 2017; + Le et al., 2014 *
Asthma	Increased	Very Strong	+ Vicedo-Cabrera et al., 2016; + Reid et al., 2016b; + Alman et al., 2016; + Parthum et al., 2017, − Kollanus et al., 2016
COPD	Increased	Very Strong	+ Reid et al., 2016b; Alman et al., 2016; + Parthum et al., 2017, − Kollanus et al., 2016; +Le et al., 2014 *
Infection	Increased	Strong	+ Tinling et al., 2016; + Parthum et al., 2017
Cardiovascular	Increased	Inconclusive	+ Tinling et al., 2016; − Alman et al., 2016; − Reid et al., 2016a; − Kollanus et al., 2016; + Le et al., 2014 *
Acute MI	Mixed	Inconclusive	+ Le et al., 2014 *
Heart Failure	Mixed	Inconclusive	+ Parthum et al., 2017; +Le et al., 2014 *
Cardiac arrest	Mixed	Inconclusive	
Hypertension	Mixed	Inconclusive	+ Tinling et al., 2016; + Reid et al., 2016b (women); + Le et al., 2014 *
Arrhythmia	No assoc.		+ Le et al., 2014 *
IHD	Increased	Inconclusive	+ Alman et al., 2017, + Le et al., 2014 *
Angina	Increased	Inconclusive	
Cerebro-vascular	Mixed	Inconclusive	+ Le et al., 2014 *

COPD = Chronic obstructive pulmonary disease; IHD = Ischemic heart disease; MI=myocardial infarction, No Assoc. = No association; + indicates statistically significant positive association with wildfire-$PM_{2.5}$; - indicates no association with wildfire-$PM_{2.5}$. ± indicates borderline positive association with wildfire-$PM_{2.5}$.

* For individuals ≥65 years.

Published in final edited form as:
Occup Environ Med. 2009 March ; 66(3): 189–197. doi:10.1136/oem.2008.041376.

The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003

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Abstract

Objective—There is limited information on the public health impact of wildfires. The relationship of cardiorespiratory hospital admissions (n = 40 856) to wildfire-related particulate matter ($PM_{2.5}$) during catastrophic wildfires in southern California in October 2003 was evaluated.

Methods—Zip code level $PM_{2.5}$ concentrations were estimated using spatial interpolations from measured $PM_{2.5}$, light extinction, meteorological conditions, and smoke information from MODIS satellite images at 250 m resolution. Generalised estimating equations for Poisson data were used to assess the relationship between daily admissions and $PM_{2.5}$, adjusted for weather, fungal spores (associated with asthma), weekend, zip code-level population and sociodemographics.

Results—Associations of 2-day average $PM_{2.5}$ with respiratory admissions were stronger during than before or after the fires. Average increases of 70 μg/m^3 $PM_{2.5}$ during heavy smoke conditions compared with $PM_{2.5}$ in the pre-wildfire period were associated with 34% increases in asthma admissions. The strongest wildfire-related $PM_{2.5}$ associations were for people ages 65– 99 years (10.1% increase per 10 μg/m^3 $PM_{2.5}$, 95% CI 3.0% to 17.8%) and ages 0–4 years (8.3%, 95% CI 2.2% to 14.9%) followed by ages 20–64 years (4.1%, 95% CI 20.5% to 9.0%). There were no

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Competing interests: None.

$PM_{2.5}$–asthma associations in children ages 5–18 years, although their admission rates significantly increased after the fires. Per 10 μg/m^3 wildfire-related $PM_{2.5}$, acute bronchitis admissions across all ages increased by 9.6% (95% CI 1.8% to 17.9%), chronic obstructive pulmonary disease admissions for ages 20–64 years by 6.9% (95% CI 0.9% to 13.1%), and pneumonia admissions for ages 5–18 years by 6.4% (95% CI 21.0% to 14.2%). Acute bronchitis and pneumonia admissions also increased after the fires. There was limited evidence of a small impact of wildfire-related $PM_{2.5}$ on cardiovascular admissions.

Conclusions—Wildfire-related $PM_{2.5}$ led to increased respiratory hospital admissions, especially asthma, suggesting that better preventive measures are required to reduce morbidity among vulnerable populations.

The numbers of wildfires and their duration in the USA have increased over the past two decades due to warmer temperatures, earlier snowmelts and less rainfall, all of which are expected to worsen because of global warming.[1] These phenomena will likely impact public health. However, although the adverse effects of urban fine particulate air pollution ($PM_{2.5}$ or particles with an aerodynamic diameter of <2.5 μm) on cardiovascular and respiratory health have been well documented,[2] far fewer studies have evaluated the impacts of wildfire-generated $PM_{2.5}$. $PM_{2.5}$ is the air pollutant with the greatest increase in concentrations during fire events,[3] followed by particulate matter with an aerodynamic diameter of <10 μm (PM_{10}).[4] Studies that have evaluated the impacts of wildfire PM on hospital admissions, emergency department visits or clinic visits found associations with respiratory outcomes.[5–11] There is little research on the impact of wildfire smoke on cardiovascular outcomes; two studies have found no significant associations.[89] There have been conflicting reports on wildfire smoke and total mortality.[1213] Several other studies have found adverse impacts of wildfire smoke on respiratory symptoms, medication use and lung function.[1014–16]

We present here the largest study to date evaluating the relationships of hospital admissions for cardiorespiratory outcomes to wildfire-associated $PM_{2.5}$ using data from the catastrophic wildfires that struck southern California in the autumn of 2003. We linked $PM_{2.5}$ concentrations estimated at the zip code level[17] to a population-based dataset of hospital admissions using spatial time series analyses of data before, during and after the fires. Strong, dry winds from inland deserts fanned flames from nine distinct fires, which burned nearly three quarters of a million acres and destroyed approximately 5000 residences and outbuildings. The wildfires generated large amounts of dense smoke that covered much of urban southern California (2003 population of 20.5 million).[18] $PM_{2.5}$ and PM_{10} concentrations far exceeded US federal regulatory standards.[317] The goal of the present study is to assess the impact of this large wildfire event on serious morbidity.

METHODS

Hospital admission data

Hospital admission data for children and adults were obtained from the California State Office of Statewide Health Planning and Development (OSHPD). Specifically, we analysed 40 856 hospital admissions from the period before the wildfire episode (1–20 October), the

episode period across southern California (21–30 October) and the period following the episode (31 October–15 November), for individuals who lived in affected counties and were diagnosed with the respiratory and cardiovascular illnesses listed in table 1. Other variables from OSHPD included in analyses were age, sex, race, ethnicity, five-digit zip code and admission date. Patient zip code data from OSHPD were geocoded to zip code centroids and linked to air monitoring data and U.S. Census 2000 sociodemographic data. Institutional Review Board approvals were obtained from the California State Health and Human Services Agency, Committee for the Protection of Human Subjects, and from the University of California, Irvine Office of Research Administration.

Analyses were stratified by age groups: paediatric (0–4 and 5– 19 years), adult (20–64 years) and elderly (65–99 years), except for chronic obstructive pulmonary disease (COPD, 20–64 and 65–99 years) and cardiovascular outcomes (45–99 years). Census demographic characteristics were missing for 474 admissions due to unmatched zip codes. We also analysed associations for asthma by gender because of differences in the age-dependent prevalence of asthma.

Exposures

We estimated daily PM_{10} and $PM_{2.5}$ concentrations at a zip code level from 1 October through 15 November 2003. These data are presented in more detail in our previous publication.[17] To our knowledge, this was the first study that systematically examined and estimated daily particle concentrations at such a fine spatial resolution over a relatively large study domain for this type of application. Spatially-resolved particle mass data are superior to using only the nearest available monitoring station data because they are expected to better represent personal exposures. We used available air pollution data from governmental network sites to build prediction models. Missing gravimetric PM concentrations from every 3rd or 6th day measurements or due to the incapacitation of monitors by the fires were estimated based on (1) temporal profiles of continuous hourly PM data at co-located or closely located sites and (2) light extinction from visibility data, meteorological conditions and smoke information extracted from moderate resolution imaging spectroradiometer (MODIS) satellite images at a 250 m resolution. Moderately strong prediction equations were developed for gravimetric PM mass at monitoring stations. Light extinction coefficient and MODIS satellite smoke data were the most important predictors of those measurements. Measured $PM_{2.5}$ was more accurately predicted in regression models compared with PM_{10} (R^2 0.78 vs 0.65, respectively). Therefore, the present analysis focuses only on $PM_{2.5}$.

Spatial interpolations of $PM_{2.5}$ concentrations were performed using inverse distance weighting, kriging or cokriging methods for the non-fire periods. Since the fire and smoke created highly heterogeneous pollution surfaces, typical inverse distance weighting and kriging were not suitable during the wildfire period. Therefore, polygons were created based on satellite images to represent each smoke-covered area under different smoke densities. $PM_{2.5}$ concentrations in each smoke-polygon were assigned separately, using measured or estimated concentrations from the predictive models (as described above). For each non-fire and fire day, the spatial $PM_{2.5}$ surfaces and zip code boundary map were overlaid and corresponding $PM_{2.5}$ concentrations were assigned to each zip code centroid (fig 1).

Measurements of daily airborne fungal spores (see online supplement) were carried out in another ongoing study in Riverside County.[19] Pollen concentrations were low and therefore were not included in the analysis. We assumed that Riverside ambient fungal data reflected region-wide trends.

Analysis

Outcomes were the total number of admissions for a diagnostic group within each zip code on each day of the study period. We hypothesised that associations between the wildfires and hospital admission rates would primarily be attributable to an increase in daily zip code-specific levels of $PM_{2.5}$ resulting from the fires. However, it is difficult to separate wildfire-generated PM from other PM sources in this heavily urbanised region. To this end, we constructed a wildfire indicator representing prewildfire, wildfire and post-wildfire periods, and tested the interaction between $PM_{2.5}$ and this indicator. We considered product terms to be significant at the $p<0.1$ level. Because dates of the wildfires varied throughout southern California, dates for the wildfire period indicator were defined to be county-specific based on MODIS satellite images of smoke covering any part of the county's urban areas (table 2).

The choice of adjustment covariates was motivated by biological plausibility that the covariate might confound the relationship between wildfire-related $PM_{2.5}$ and hospital admissions or an a priori belief that the variable could affect both $PM_{2.5}$ and admissions. Meteorological covariates from the National Climatic Data Center (http://www.ncdc.noaa.gov/oa/ncdc.html) included relative humidity, temperature and surface pressure gradient. So-called Santa Ana winds coming off the inland desert regions to the east (a large negative pressure gradient) are a strong determinant of wildfire events. There are few data on the effects of Santa Ana winds on asthma or other outcomes, but it is anticipated that hot dry desert winds associated with this weather pattern bring with them high concentrations of bioaerosols. Therefore, for asthma admissions, we also included fungal spores as a covariate. Deuteromycetes (eg, *Alternaria*) tend to increase during hot, dry windy periods.[20]

In addition, we decided a priori that spatial heterogeneity in census demographic factors at the aggregate zip code level (age, gender, race and income distributions) could confound associations. The distributions of each of these potential confounders were obtained at the zip code level from the 2000 U.S. Census (percentage of non-Caucasians, percentage of females, median household income and age distributions). Income was recoded into discrete variables by quartile. To control for zip code population age distribution, we first calculated the percentage of individuals in a zip code younger than 20 years and older than 65 years. Each zip code was then classified into one of four age categories by cross-classification of young (proportion of individuals <20 years old higher than the median proportion across all zip codes) and old (proportion of individuals>65 years old higher than the median proportion across all zip codes).

We also tested various functions of time including weekend versus weekday, day of the week and a smooth of time. In order to investigate residual confounding by date, we allowed for a flexible functional form (via smoothing splines, with degrees of freedom ranging from 1 to 10) (see online supplement). Controlling for day-of-week trend or the flexible time-

adjusted models showed the $PM_{2.5}$ associations were robust with respect to these adjustments. We also tested various forms of temperature and relative humidity, including raw continuous scales, smoothed and categorical forms. Those models exhibiting the best fit with the fewest assumptions for functional form included weekend versus weekday, and temperature and relative humidity categorised into quartiles. The full set of adjustment covariates included these variables plus local pressure gradient, fungal spores (for asthma), county, and zip code-level distributions of median household income, age, gender and race. Effects of covariates on point estimates of $PM_{2.5}$ were small.

Generalised estimating equations for Poisson data[21] were used to estimate the marginal association of daily hospital admission rates with daily $PM_{2.5}$ levels and presence of the wildfires. Log-transformed zip code-specific population estimates were used as the offset (denominator) term in all models. Age-specific population estimates were used as an offset term in the analysis of age group-specific outcomes. In order to obtain asymptotically valid inferences, covariate estimation was carried out using an independence working correlation structure in combination with empirical variance estimates clustering on zip code.[2223] We note that the use of an independence working correlation structure was motivated by the desire to obtain consistent parameter estimates in the presence of time-varying covariates.[24]

Multiple lag models were considered to investigate associations between $PM_{2.5}$ and hospital admission rates, including a 7-day polynomial distributed lag,[25] and stratified analyses considering different lag associations. We found the 2-day moving average of $PM_{2.5}$ (average of today and yesterday) provided the best fitting model that adequately captured the association between $PM_{2.5}$ and admissions.

RESULTS

PM exposures

During the wildfires, smoke events dramatically increased local PM concentrations and created highly heterogeneous pollution surfaces.[17] For reference, the US National Ambient Air Quality Standard for 24 h average $PM_{2.5}$ is 35 $\mu g/m^3$. The highest 24 h concentrations were ≥240 $\mu g/m^3$ at two sites in San Diego County. Table 2 contains county-level descriptive statistics for $PM_{2.5}$. As expected, average $PM_{2.5}$ concentrations during the wildfire period increased in all counties. Average PM levels during the period following the fires were observed to be lower in all counties relative to the period prior to the fires. This is because of the onshore flow that brought in the cool and moist clean air from the Pacific Ocean that helped end the wildfires.

Spatial time series analysis of hospital admissions

$PM_{2.5}$ associations: interactions with wildfire period—We found that associations of 2-day lagged average of $PM_{2.5}$ with admissions for most respiratory outcomes were stronger during as compared with before or after the wildfires in models including a product term of wildfire period and $PM_{2.5}$, but the interaction was $p<0.1$ primarily for asthma.

Table 3 shows estimates for the relative change in rates for admissions in relation to a 10 $\mu g/m^3$ increase in $PM_{2.5}$. The table includes results for age and sex (asthma only) subgroups

for the entire monitored period, and for wildfire periods. In product term models of $PM_{2.5}$ by wildfire period, $PM_{2.5}$ during the wildfire period was associated with combined respiratory admissions. Asthma admissions across all ages increased by 4.8% (95% CI 2.1% to 7.6%) in relation to $PM_{2.5}$ during the wildfire period, but there was no $PM_{2.5}$ association before or after the fires. The strongest wildfire-related $PM_{2.5}$ associations with asthma admissions were for the elderly, ages 65–99 years (10.1% increase), and children ages 0–4 years (8.3%), followed by adults ages 20–64 years (4.1%). There were no $PM_{2.5}$ associations in school aged children. Among women ages 20– 64 years, the strongest asthma and $PM_{2.5}$ association was during the wildfires, but for men those ages it was after the wildfires. Among women ages 65–99, the strongest $PM_{2.5}$ association was after the wildfires, but for men those ages it was during the wildfires. Fungal spores were also significantly associated with asthma admissions in the adjusted model that included $PM_{2.5}$ (see online supplement).

The wildfires led to notably higher particle concentrations, so that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ used for effect estimates in table 3 represents only a small part of that increase. The overall population-weighted concentrations of predicted 24 h $PM_{2.5}$ at the zip code level were 90 $\mu g/m^3$ and 75 $\mu g/m^3$, under heavy and light smoke conditions, respectively, in contrast to concentrations of 20 $\mu g/m^3$ during the non-fire period.[17] Therefore, we rescaled effect estimates to represent the wildfire-related increases in $PM_{2.5}$. A 55 $\mu g/m^3$ increase in $PM_{2.5}$ during light smoke and a 70 $\mu g/m^3$ increase in $PM_{2.5}$ during heavy smoke conditions are predicted to lead to an adjusted 26% and 34% increase in asthma admissions for all ages, respectively.

For combined ages, acute bronchitis admissions increased more in relation to 10 $\mu g/m^3$ $PM_{2.5}$ during the wildfires (9.6%), but there was no association before or after the fires. In subgroup analyses, this association was still evident in children ages 0–4 years and the elderly.

COPD admissions for people ages 20–64 years significantly increased by 6.8% from 10 $\mu g/m^3$ $PM_{2.5}$ during the wildfires, but there was no association before or after the fires. The COPD increase with $PM_{2.5}$ during the fires was smaller for subjects ages 65–99 years (3.1%).

$PM_{2.5}$ was also associated with increased overall pneumonia admissions, both before (4.5%) and during the fires (2.8%). This was consistent across ages, except children ages 5–19 years showed an association only during the wildfires. There were no associations of $PM_{2.5}$ with admissions for upper respiratory infections (not shown).

There was a small relative increase in admission rates for total cardiovascular outcomes in people ages 45–99 years in relation to $PM_{2.5}$ during the fires. There were suggestions of a small increase in admissions for congestive heart failure in relation to $PM_{2.5}$ during the wildfires ($p<0.1$ compared with the pre-wildfire period), and an even smaller increase in admissions for ischaemic heart disease, but for both outcomes, the 95% confidence intervals crossed 1.0. $PM_{2.5}$ was inversely associated with cardiac dysrhythmia admissions across all periods. Admissions for cerebrovascular disease and stroke were positively associated with $PM_{2.5}$ (1.9%) across all periods.

Associations with wildfire period—In this analysis of the wildfire indicator variable, the prewildfire period is the referent time. Models were adjusted for the same covariates as $PM_{2.5}$ models, and are shown unadjusted and adjusted for $PM_{2.5}$ (table 4). Generally, there was little change in point estimates adjusting for $PM_{2.5}$. There were significantly increased risks for all respiratory hospital admissions after the fires compared with the pre-fire period. Admissions increased for all ages by 17% ($p<0.001$), and in age groups 5–19 years by 37% ($p<0.008$) and 65–99 years by 15% ($p<0.004$). Unexpected decreased risks of respiratory admissions were found during the fires compared with the pre-fire period in 0–4 year olds and elderly adults.

The period following the fires was associated with a 26% increase in the rate of asthma admissions for all ages. Asthma admissions were also increased during the fires among those aged 5–19 years (25%) and 20–64 years (27%), but associations for both groups were stronger after the fires (56% and 36%, respectively).

Increased risk of asthma admissions for the period during the wildfires was stronger in females ages 5–19 years (49%, $p<0.02$) than males (11%, $p = 0.5$) and in females ages 20–64 years (41%, $p<0.001$) than males (27.6%, $p = 0.7$) (not shown). Increased risk of asthma admissions for the period after the wildfires was also stronger in females ages 5–19 years (81%, $p<0.01$) than males (39%, $p<0.11$) and in females ages 20–64 years (47%, $p<0.02$) than males (12%, $p = 0.7$).

Admissions for acute bronchitis and bronchiolitis for combined ages were increased by 48% after the fires. The association for the post-fire period was seen in both ages 0–4 years (51%) and ages 20–64 years (137%). Pneumonia admissions for ages 0– 4, 20–64 and 65–99 years were 46%, 30% and 27% higher during the period after the fires, respectively.

There was a 6.1% increased risk of combined cardiovascular admissions ($p<0.05$), and an 11.3% increased risk of congestive heart failure admissions after the fires ($p<0.06$). However, risk of cardiovascular admissions was lower during the fires by 4.4%. A relative increase in cerebrovascular disease and stroke admissions during the wildfires may have been attributable to a cross-period effect of $PM_{2.5}$ (table 3) because this period association was confounded in the model adjusting for $PM_{2.5}$.

DISCUSSION

This is the first study to systematically examine and estimate the impacts on hospital admissions from wildfire-related $PM_{2.5}$ at such a fine spatial resolution (zip codes) over a large urban region. During the wildfire period, smoke events dramatically increased $PM_{2.5}$ compared to the preceding non-fire period. The wildfires and associated $PM_{2.5}$ were significantly associated with hospital admissions for respiratory illnesses, especially asthma, but also acute bronchitis and COPD. The impact on cardiovascular admissions was weaker.

Although product terms between $PM_{2.5}$ and the wildfire period indicator were not significant at the $p<0.1$ level in many models, we still observed a trend of stronger associations for $PM_{2.5}$ with respiratory admissions during the wildfire period. Some models showed increased admissions in relation to $PM_{2.5}$ before the wildfires, possibly due to the

relatively high concentration of urban PM seen during this hot period (table 2). Some models also showed increased admissions in relation to $PM_{2.5}$ after the wildfires, despite much lower $PM_{2.5}$ concentrations. This may have been attributable to notable increases in respiratory admissions seen then, possibly due to a delayed impact of wildfire smoke.

Models with the wildfire period indicator support this possibility and suggest that some effects of wildfires are not entirely explained by $PM_{2.5}$ exposures. Results yielded inconsistencies for respiratory and cardiovascular admissions when comparing product term models for $PM_{2.5}$ by period to models using the period indicator alone. There were nominal associations of daily $PM_{2.5}$ during the wildfires with cardiovascular admissions, but the period indicator showed associations only after the wildfires. Non-asthma respiratory admission rates were also most strongly increased after the wildfires ended compared with the pre-fire period, while the $PM_{2.5}$ association was generally strongest during the wildfires. We also found the period following the wildfires was significantly associated with higher overall asthma admission rates. These associations were stronger among females. Asthma admissions were increased during the fires as well, but evident only among females ages 5–19 and ages 20–64. Possible reasons for stronger associations among females include the differential impact of hormones and the menstrual cycle, airway function and structure, atopy and perception of symptoms.[26]

Although there was no association of asthma admissions with $PM_{2.5}$ in young people ages 5–19 years, the periods during and after the wildfires were significantly associated with increased admissions in this group. We speculate this may be attributable to unmeasured volatile (non-particulate) toxic air pollutants, including those associated with the more than 5000 buildings that burned. Alternatively, factors associated with the fires, such as psychosocial stress, could have led to effects that were independent of $PM_{2.5}$.

Associations with the post-wildfire period and wildfire-related $PM_{2.5}$ were also found for acute bronchitis and bronchiolitis, and pneumonia. This is the first report of wildfire associations with admissions for acute bronchitis and bronchiolitis, and pneumonia.

We also found a significantly increased risk of admissions for total cardiovascular outcomes and congestive heart failure after the fires. It is possible that systemic inflammation increases more strongly in relation to sustained multiday exposures to air pollutants than with acute single day exposures, as recently shown in our panel study of subjects with coronary artery disease.[27] Analyses of the London ‘‘killer smog’’ of 1952,[28] and recent analyses of particulate air pollution in Dublin, Ireland,[29] suggest that there may be delayed effects for weeks to months. The post-fire increases in cardiorespiratory admissions may be attributed to the following:

1. People may delay deciding to go to hospital until symptoms become too severe[30];
2. Cumulative biological effects of wildfire PM may culminate in severe symptoms many days after the initial cardiorespiratory impact. For example, most subjects with asthma show a progressive clinical and functional deterioration that takes place over hours to weeks[31];

3. Sustained effects of wildfire PM may lead to susceptibility to, or increased severity of, later respiratory infections, possibly through alterations in immune function or respiratory clearance mechanisms.

The strongest evidence for delayed effects in our study was the post-fire increase in asthma admissions combined with the association between asthma admission and $PM_{2.5}$ during the wildfires. However, given past annual trends (see online supplement), it is possible that asthma admissions following the wildfire period would have increased at this time of year anyway. This also applies to the post-fire increases in admissions for acute bronchitis and bronchiolitis, and pneumonia. Other limitations are that the period analysis does not have the temporal resolution of the daily time series analysis of $PM_{2.5}$. Therefore, differences in results of these analyses could result due to imprecision in the estimate for the non-quantitative indicator variable. Furthermore, power may be limited for specific outcomes subdivided by gender and age, which would apply to several nominally significant associations we found.

Our results for respiratory admissions are consistent with two other studies of the 2003 southern California wildfires using other less severe outcomes and focusing on particular regions, including emergency department visits in San Diego county[1132] and respiratory symptoms in 16 towns in southern California.[16] Kunzli et al[16] reported results for school children in an ongoing cohort study who were potentially affected by the wildfires. They found parental self-reports of the smell of fire smoke indoors were associated with reported asthma attacks, wheezing, cough, bronchitis, colds, upper respiratory symptoms, medication usage and physician visits. Authors also analysed the impacts of between-community differences in PM_{10} using data from our study.[17] Changes in PM_{10} were associated with upper respiratory symptoms, cough and unspecified medication use.

Several investigations of wildfires have identified people with asthma as an especially sensitive subpopulation, using analyses of emergency department visits in California mountain counties during wildfires in 1987,[6] emergency department visits in eight Florida hospitals during wildfires in 1998,[5] and hospital admissions during the 1997 Indonesian wildfires.[9] A report from Australia examining smoke from bushfires and asthma emergency department visits found no association.[33]

Other time series studies have shown associations of asthma hospital admissions with urban air pollution.[34] However, the period of observation in our investigation is far shorter than most time series investigations, and thus statistical power is lower. Despite this, we found strong associations between $PM_{2.5}$ and hospital admissions. We attribute this to the large increase in wildfire-related PM, and the spatial time series approach, which likely reduced exposure error compared with the typical use of widely-dispersed regional PM data. Nevertheless, we are still limited by aggregate (not personal) exposure data.

This is the first report of associations of wildfire-related $PM_{2.5}$ with admissions for acute bronchitis and bronchiolitis, and for pneumonia. Our results showing increased COPD admissions in relation to $PM_{2.5}$ during the wildfires are consistent with a study of increased COPD hospital admissions during the 1997 Indonesian wildfires,[9] increased COPD emergency department visits during the 1987 wildfires in California mountain counties,[6] and

respiratory symptoms in a panel of 21 patients with COPD associated with a forest fire near Denver, Colorado in June 2002.[35]

Total cardiovascular and congestive heart failure admissions increased only in the period following the wildfires. However, there was a small relative increase in admission rates for total cardiovascular outcomes in relation to $PM_{2.5}$ during the fires.

Cerebrovascular disease and stroke were significantly increased in relation to $PM_{2.5}$ across the entire study period. Unexpected findings were the inverse associations for cardiac dysrhythmias and $PM_{2.5}$ across the whole period. While urban particles generally have been associated with a variety of adverse cardiovascular outcomes,[2] including stroke,[36] there is little research investigating the effects of smoke from wildfires or wood combustion on circulatory disease.[4] Our results can only be compared to null associations for cardiovascular hospital admissions during the 1997 Indonesian wildfires.[9] Moore et al[8] found that, although there was an excess of respiratory complaints, physician visits for cardiovascular illnesses in regions of British Columbia, Canada were not associated with wildfires.

The mechanisms explaining our findings for wildfire smoke are likely somewhat similar to those found for pollutant components from fossil fuel combustion. Evidence is mounting that urban air pollution triggers oxidative stress and inflammation.[2] A study of people exposed to forest fire smoke in Indonesia in 1997 showed increased circulating levels of interleukin-1b and interleukin-6 during the smoke period.[37] An experimental study of subjects exposed to clean air versus wood smoke in a chamber showed increased airway inflammatory responses (exhaled alveolar NO) and evidence of increased oxidative stress (malonadehyde in breath condensates).[38] An in vitro study using mouse alveolar macrophages tested the effects of size-segregated PM from transported wildfire smoke collected in Helsinki, Finland.[39] Investigators showed that although the transported particles induced less cytokine production per unit mass compared with urban particles, they found enhanced inflammatory and cytotoxic activities per cubic meter of air due to the increased particulate mass concentration in the accumulation mode size range (0.1–2.5 mm in diameter). This might explain our finding of a larger asthma association per 10 $\mu g/m^3$ $PM_{2.5}$ during the wildfires as compared with the pre-wildfire period as simply due to the considerably higher concentrations rather than higher toxicity of wildfire smoke.

It is also possible that unmeasured volatile and semivolatile organic compound components are important in the effects of wildfire smoke, but such data are rarely available. In the present study, these include toxic gases emitted from synthetic materials in the approximately 5000 residences and outbuildings that burned.

Conclusions

We conclude the catastrophic wildfires that struck southern California in October of 2003 led to significantly increased hospital admissions for respiratory illnesses, especially asthma. Southern California experienced a second similar wildfire disaster in October 2007, yielding the two largest wildfire disasters in California's history within this recent 4-year period. A

concern is that growing impacts of global warming on wildfire risk will continue to impact public health in similar regions across the globe.[1]

Given there were significant morbidity impacts associated with wildfire-related $PM_{2.5}$, we recommend that in addition to advisories to avoid outdoor activities that increase exposure during wildfires, preventive measures need to be taken where possible to reduce exacerbations of asthma. This may include the early use of anti-inflammatory medications at the first sign of increasing asthma symptoms. All of the health impacts identified in this study occurred in the face of numerous advisories by public health agencies and the media to avoid outdoor activities and to use air conditioning. Additional preventive measures in susceptible people including those with persistent asthma, such as the use of indoor air filters,[1040] should be considered and then systematically evaluated in future wildfires.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

We thank Joe Cassmassi and others at the South Coast Air Quality Management District for assistance with the meteorological and air pollutant data.

Funding: This study was funded by the South Coast Air Quality Management District contract no. 04182, and the National Institutes of Health, National Institute of Environmental Health Sciences grant no. ES-11615.

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Main messages

▶ Wildfire-related $PM_{2.5}$ led to significantly increased asthma, bronchitis and COPD hospital admissions.

▶ Sensitive subgroups included young children and the elderly.

Policy implications

▶ In addition to advisories to avoid outdoor activities that increase exposure during wildfires, preventive measures need to be taken where possible to reduce exacerbations of asthma

▶ Preventive measures may include advisories for the early use of anti-inflammatory medications at the first sign of increasing asthma symptoms.

▶ The health impacts of wildfires reported here are anticipated to increase worldwide due to global warming, which has broad policy implications.

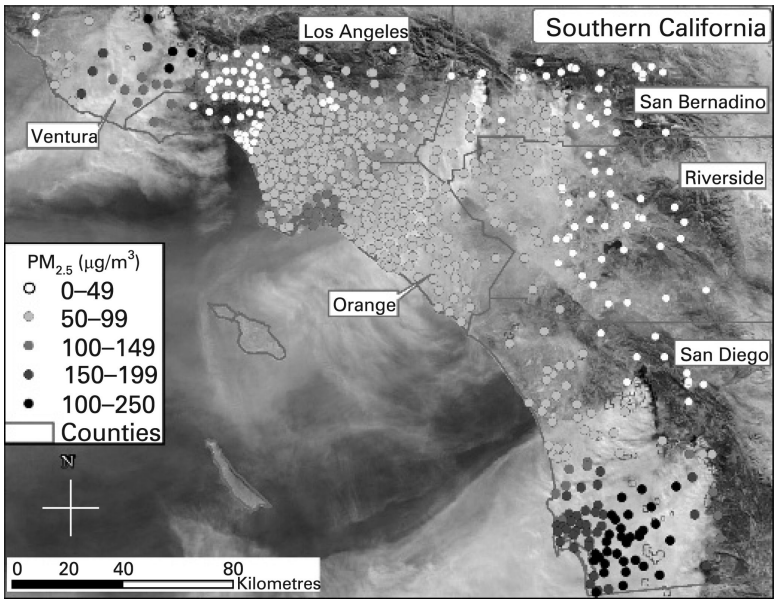

Figure 1.
Interpolated $PM_{2.5}$ concentrations ($\mu g/m^3$) at zip code centroids on 27 October 2003.

Table 1

Number of hospital admission by diagnostic* and age groups

Diagnosis	Total events	Events with U.S. Census 2000 defined population†
All respiratory‡		
Ages 0–4	2158	2143
Ages 5–19	1216	1205
Ages 20–64	8480	8314
Ages 65–99	9456	9357
Total	21 310	21 019
Asthma (ICD-9 493), primary		
Ages 0–4	606	600
Ages 5–19	739	733
Ages 20–64	1165	1151
Ages 65–99	543	538
Total	3053	3022
Acute bronchitis and bronchiolitis (ICD-9 466)		
Ages 0–4	354	353
Ages 5–19	23	23
Ages 20–64	108	106
Ages 65–99	137	136
Total	622	618
Chronic obstructive pulmonary disease (ICD-9 491, 492 and 496)		
Ages 20–64	927	910
Ages 65–99	1973	1950
Total	2900	2860
Pneumonia (ICD-9 480-87)		
Ages 0–4	542	537
Ages 5–19	298	293
Ages 20–64	1721	1686
Ages 65–99	3957	3924
Total	6518	6440
Upper respiratory infections (ICD-9 460–65)		
Ages 0–4	522	518
Ages 5–19	77	77
Ages 20–64	108	104
Ages 65–99	47	47
Total	754	746
All cardiovascular§		
Ages 45–99	27 486	27 170
Ages 65–99	19 380	19 197
Ischaemic heart disease (ICD-9 410–414)		

Diagnosis	Total events	Events with U.S. Census 2000 defined population†
Ages 45–99	10 448	10 319
Ages 65–99	6491	6430
Cardiac dysrhythmias (ICD-9 426, 427)		
Ages 45–99	4051	4004
Ages 65–99	3048	3018
Congestive heart failure (ICD-9 402, 428)		
Ages 45–99	6202	6144
Ages 65–99	4750	4712
Cerebrovascular disease and stroke (ICD-9 430–438)		
Ages 45–99	5973	5908
Ages 65–99	4465	4422

*Principal cause of admission was coded by version 9 of the International Classification of Diseases (ICD-9)

†population with available covariates for census population and census distribution of demographic characteristics used in the multivariate analysis. This excludes subjects aged ≥100 years (48 (0.23%) respiratory and 51 (0.18%) cardiovascular admissions) because 2000 census age categories needed in the analysis stopped at 99 years

‡includes all listed specific respiratory ICD-9 plus 7463 additional admissions for the following ICD-9 codes: 277 (cystic fibrosis), 490 (bronchitis NOS), 494 (bronchiectasis), 495 (extrinsic allergic alveolitis), 506 and 508 (other acute/subacute respiratory conditions due to fumes/vapours, or external agents, not separately analysed because n = 44), 786 (symptoms involving the respiratory system/other chest symptoms).

§includes all listed specific cardiovascular ICD-9 codes plus 812 additional admissions for ICD-9 codes 440–459 (diseases of the peripheral circulation).

Table 2

County-level mean particulate matter (PM2.5) levels,* Southern California, 1 October–15 November 2003

Daily PM_{25} levels (mg/m^3)	County					
	Los Angeles	Orange	Riverside	San Bernardino	San Diego	Ventura
Before fires						
Dates	01/10–23/10	01/10–23/10	01/10–20/10	01/10–20/10	01/10–24/10	01/10–22/10
Concentration (SD)	27.2 (12.4)	23.3 (9.6)	32.7 (14.7)	35.7 (16.6)	18.5 (6.7)	18.4 (8.3)
During fires						
Dates	24/10–29/10	24/10–28/10	21/10–29/10	21/10–30/10	25/10–30/10	23/10–30/10
Concentration (SD)	54.1 (21)	64.3 (26.5)	42.1 (25.5)	45.3 (28.7)	76.1 (66.6)	50.1 (50.5)
After fires						
Dates	30/10–15/11	29/10–15/11	30/10–15/11	31/10–15/11	31/10–15/11	31/10–15/11
Concentration (SD)	15.9 (5.5)	15.5 (10.2)	16.9 (8.6)	18.4 (8.3)	14.2 (7.2)	12.9 (4.3)

* PM_{25} concentrations are calculated with equal weighting per zip code.

Table 3

Relative rate of asthma admissions in relation to a 10 μmg/m^3 increase in 2-day moving average particulate matter ($PM_{2.5}$)

Hospital admissions outcome	All periods RR (95% CI)*	Pre-wildfire period RR (95% CI)	Wildfire period RR (95% CI)	p Value†	Post-wildfire period RR (95% CI)	p Value
All respiratory						
All ages	1.009 (0.999 to 1.018)	1.022 (1.004 to 1.040)	1.028 (1.014 to 1.041)	0.639	0.999 (0.968 to 1.031)	0.198
Ages 0–4	0.994 (0.967 to 1.021)	0.982 (0.921 to 1.046)	1.045 (1.010 to 1.082)	0.103	0.894 (0.807 to 0.991)	0.126
Ages 5–19	1.014 (0.983 to 1.046)	1.026 (0.946 to 1.113)	1.027 (0.984 to 1.076)	0.990	0.958 (0.852 to 1.077)	0.354
Ages 20–64	1.015 (1.002 to 1.029)	1.036 (1.007 to 1.066)	1.024 (1.005 to 1.044)	0.534	1.007 (0.960 to 1.056)	0.315
Ages 65–99	1.009 (0.996 to 1.022)	1.022 (0.994 to 1.050)	1.030 (1.011 to 1.049)	0.649	1.024 (0.976 to 1.074)	0.932
Asthma						
All ages						
Males and females	1.022 (1.001 to 1.042)	0.998 (0.949 to 1.050)	1.048 (1.021 to 1.076)	0.097	0.986 (0.910 to 1.068)	0.792
Males	1.010 (0.980 to 1.040)	1.021 (0.944 to 1.106)	1.031 (0.990 to 1.073)	0.848	1.063 (0.948 to 1.192)	0.553
Females	1.029 (1.001 to 1.058)	0.979 (0.913 to 1.050)	1.059 (1.022 to 1.097)	0.056	0.928 (0.829 to 1.037)	0.412
Ages 0–4						
Males and females	0.996 (0.947 to 1.048)	0.924 (0.824 to 1.035)	1.083 (1.021 to 1.149)	0.017	0.924 (0.767 to 1.113)	0.999
Males	1.018 (0.963 to 1.076)	0.942 (0.815 to 1.089)	1.086 (1.016 to 1.162)	0.101	1.057 (0.839 to 1.332)	0.380
Females	0.937 (0.845 to 1.040)	0.880 (0.706 to 1.099)	1.073 (0.965 to 1.194)	0.116	0.699 (0.515 to 0.949)	0.214
Ages 5–19						
Males and females	1.006 (0.966 to 1.048)	1.045 (0.936 to 1.167)	0.999 (0.935 to 1.068)	0.492	0.918 (0.788 to 1.069)	0.198
Males	0.991 (0.935 to 1.051)	1.034 (0.892 to 1.198)	0.969 (0.883 to 1.064)	0.462	0.979 (0.806 to 1.189)	0.671
Females	1.026 (0.964 to 1.092)	1.065 (0.901 to 1.260)	1.033 (0.943 to 1.132)	0.768	0.831 (0.640 to 1.079)	0.136
Ages 20–64						
Males and females	1.043 (1.012 to 1.076)	1.037 (0.957 to 1.123)	1.041 (0.995 to 1.090)	0.931	1.000 (0.882 to 1.132)	0.624
Males	1.013 (0.954 to 1.077)	1.159 (0.996 to 1.349)	0.939 (0.837 to 1.053)	0.026	1.275 (1.020 to 1.595)	0.486
Females	1.052 (1.015 to 1.090)	0.995 (0.904 to 1.096)	1.064 (1.014 to 1.116)	0.247	0.908 (0.780 to 1.056)	0.310
Ages 65–99						
Males and females	1.027 (0.974 to 1.082)	0.951 (0.849 to 1.064)	1.101 (1.030 to 1.178)	0.032	1.168 (0.967 to 1.412)	0.072
Males	1.046 (0.957 to 1.142)	0.948 (0.804 to 1.116)	1.185 (1.077 to 1.305)	0.029	0.902 (0.629 to 1.294)	0.804

Hospital admissions outcome	All periods RR (95% CI)*	Pre-wildfire period RR (95% CI)	Wildfire period RR (95% CI)	p Value†	Post-wildfire period RR (95% CI)	p Value
Females	1.018 (0.958 to 1.081)	0.947 (0.813 to 1.102)	1.065 (0.977 to 1.162)	0.195	1.263 (1.024 to 1.557)	0.032
Acute bronchitis and bronchiolitis						
All ages	1.044 (0.990 to 1.102)	1.001 (0.890 to 1.126)	1.096 (1.018 to 1.179)	0.223	1.031 (0.870 to 1.222)	0.779
Ages 0–4	1.017 (0.949 to 1.089)	0.987 (0.847 to 1.149)	1.092 (0.997 to 1.195)	0.276	0.910 (0.700 to 1.183)	0.588
Ages 5–19	No convergence					
Ages 20–64	1.039 (0.912 to 1.183)	1.001 (0.792 to 1.266)	1.044 (0.872 to 1.252)	0.778	1.259 (0.921 to 1.722)	0.275
Ages 65–99	1.134 (1.039 to 1.238)	1.073 (0.764 to 1.505)	1.143 (1.032 to 1.265)	0.730	1.190 (0.865 to 1.638)	0.652
Chronic obstructive pulmonary disease						
Ages 20–99	1.018 (0.994 to 1.042)	1.007 (0.958 to 1.058)	1.038 (1.004 to 1.075)	0.320	1.024 (0.943 to 1.112)	0.728
Ages 20–64	1.022 (0.980 to 1.066)	0.995 (0.916 to 1.081)	1.068 (1.009 to 1.131)	0.161	1.015 (0.893 to 1.153)	0.781
Ages 65–99	1.019 (0.992 to 1.048)	1.014 (0.955 to 1.077)	1.031 (0.990 to 1.074)	0.660	1.023 (0.928 to 1.128)	0.878
Pneumonia						
All ages	1.009 (0.994 to 1.024)	1.045 (1.012 to 1.078)	1.028 (1.007 to 1.050)	0.420	0.980 (0.927 to 1.035)	0.045
Ages 0–4	0.995 (0.944 to 1.049)	1.048 (0.931 to 1.180)	1.018 (0.948 to 1.092)	0.691	0.823 (0.649 to 1.044)	0.089
Ages 5–19	1.030 (0.966 to 1.098)	1.017 (0.882 to 1.172)	1.064 (0.990 to 1.142)	0.586	1.017 (0.767 to 1.349)	0.998
Ages 20–64	1.008 (0.982 to 1.035)	1.041 (0.982 to 1.104)	1.032 (0.994 to 1.072)	0.823	1.013 (0.913 to 1.124)	0.633
Ages 65–99	1.011 (0.993 to 1.030)	1.050 (1.006 to 1.097)	1.029 (1.002 to 1.057)	0.445	0.985 (0.920 to 1.055)	0.127
All cardiovascular	0.996 (0.989 to 1.003)	0.992 (0.976 to 1.009)	1.008 (0.999 to 1.018)	0.104	0.991 (0.964 to 1.019)	0.955
Ischaemic heart disease	0.991 (0.980 to 1.003)	0.990 (0.963 to 1.017)	1.007 (0.990 to 1.024)	0.313	0.989 (0.950 to 1.030)	0.976
Congestive heart failure	0.989 (0.974 to 1.004)	0.978 (0.942 to 1.015)	1.016 (0.993 to 1.039)	0.096	0.969 (0.914 to 1.027)	0.791
Cardiac dysrhythmia	0.980 (0.962 to 0.998)	0.979 (0.935 to 1.025)	0.989 (0.961 to 1.017)	0.721	0.976 (0.912 to 1.044)	0.934
Cerebrovascular disease and stroke	1.019 (1.004 to 1.035)	1.015 (0.980 to 1.052)	1.016 (0.997 to 1.036)	0.971	1.044 (0.987 to 1.104)	0.379

*Rate ratio and 95% confidence interval per 10 μg/m^3 increase in 2-day moving average $PM_{2.5}$, adjusted for fungal spore counts (asthma only), race, gender, county, median income, weekend, relative humidity, temperature, age and pressure gradient. RR×100 is the percentage increase in hospital admissions. Estimates for the three strata are derived from the product term models, while estimates for the full period are from a model without interaction terms

†the product term p value for the difference with the pre-fire period.

Table 4

Relative rate of respiratory admissions in relation to wildfire period

Hospital admissions outcome	n*	Pre-wildfire period (referent)	Wildfire period RR (95% CI)†		Post-wildfire period RR (95% CI)	
			Unadjusted for $PM_{2.5}$	Adjusted for $PM_{2.5}$	Unadjusted for $PM_{2.5}$	Adjusted for $PM_{2.5}$
All respiratory						
All ages	21 019	1.00	0.961 (0.916 to 1.008)	0.903 (0.850 to 0.960)	1.143 (1.072 to 1.219)	1.173 (1.097 to 1.253)
Ages 0–4	2143	1.00	0.865 (0.757 to 0.989)	0.842 (0.717 to 0.988)	1.152 (0.957 to 1.388)	1.162 (0.954 to 1.415)
Ages 5–19	1205	1.00	1.098 (0.910 to 1.324)	1.087 (0.863 to 1.370)	1.373 (1.089 to 1.732)	1.467 (1.142 to 1.883)
Ages 20–64	8314	1.00	0.991 (0.922 to 1.066)	0.923 (0.843 to 1.012)	1.074 (0.971 to 1.188)	1.104 (0.992 to 1.228)
Ages 65–99	9357	1.00	0.932 (0.867 to 1.003)	0.874 (0.795 to 0.959)	1.147 (1.045 to 1.259)	1.193 (1.084 to 1.313)
Asthma						
All ages	3022	1.00	1.088 (0.965 to 1.227)	0.992 (0.856 to 1.149)	1.264 (1.085 to 1.473)	1.336 (1.134 to 1.573)
Ages 0–4	600	1.00	0.806 (0.632 to 1.029)	0.714 (0.515 to 0.990)	1.092 (0.759 to 1.572)	1.133 (0.777 to 1.654)
Ages 5–19	733	1.00	1.254 (0.999 to 1.575)	1.282 (0.958 to 1.716)	1.564 (1.160 to 2.109)	1.629 (1.184 to 2.243)
Ages 20–64	1151	1.00	1.273 (1.067 to 1.518)	1.221 (0.979 to 1.524)	1.362 (1.043 to 1.779)	1.486 (1.111 to 1.987)
Ages 65–99	538	1.00	0.869 (0.657 to 1.151)	0.645 (0.450 to 0.925)	0.924 (0.606 to 1.408)	1.005 (0.650 to 1.552)
Acute bronchitis/bronchiolitis						
All ages	618	1.00	1.143 (0.878 to 1.490)	0.959 (0.696 to 1.321)	1.482 (1.042 to 2.109)	1.580 (1.089 to 2.291)
Ages 0–4	353	1.00	1.128 (0.819 to 1.555)	0.899 (0.607 to 1.333)	1.520 (0.947 to 2.440)	1.547 (0.954 to 2.507)
Ages 5–19	23	1.00				
Ages 20–64	106	1.00	1.350 (0.688 to 2.648)	1.320 (0.608 to 2.863)	2.454 (1.068 to 5.640)	2.515 (1.055 to 5.998)
Ages 65–99	136	1.00	1.166 (0.643 to 2.115)	0.934 (0.422 to 2.066)	0.911 (0.428 to 1.942)	0.997 (0.439 to 2.262)
Chronic obstructive pulmonary disease						
Ages 20–99	2860	1.00	0.988 (0.875 to 1.115)	0.913 (0.779 to 1.069)	1.043 (0.885 to 1.228)	1.064 (0.897 to 1.262)
Ages 20–64	910	1.00	0.967 (0.779 to 1.201)	0.873 (0.660 to 1.156)	1.175 (0.862 to 1.601)	1.311 (0.954 to 1.802)
Ages 65–99	1950	1.00	1.002 (0.869 to 1.156)	0.926 (0.767 to 1.117)	0.985 (0.811 to 1.196)	0.981 (0.798 to 1.206)
Pneumonia						
All ages	6440	1.00	0.943 (0.868 to 1.025)	0.888 (0.799 to 0.986)	1.294 (1.158 to 1.446)	1.318 (1.174 to 1.479)
Ages 0–4	537	1.00	0.938 (0.705 to 1.247)	0.951 (0.678 to 1.333)	1.458 (0.974 to 2.182)	1.374 (0.885 to 2.133)
Ages 5–19	293	1.00	0.891 (0.604 to 1.312)	0.830 (0.541 to 1.272)	0.960 (0.588 to 1.569)	0.969 (0.578 to 1.624)

Hospital admissions outcome	n*	Pre-wildfire period (referent)	Wildfire period RR (95% CI)†		Post-wildfire period RR (95% CI)	
			Unadjusted for $PM_{2.5}$	Adjusted for $PM_{2.5}$	Unadjusted for $PM_{2.5}$	Adjusted for $PM_{2.5}$
Ages 20–64	1686	1.00	0.927 (0.795 to 1.081)	0.837 (0.690 to 1.016)	1.314 (1.064 to 1.622)	1.300 (1.047 to 1.615)
Ages 65–99	3924	1.00	0.959 (0.861 to 1.068)	0.899 (0.782 to 1.033)	1.277 (1.102 to 1.481)	1.331 (1.142 to 1.552)
			Unadjusted for $PM_{2.5}$	Adjusted for $PM_{2.5}$	Unadjusted for $PM_{2.5}$	Adjusted for $PM_{2.5}$
All cardiovascular‡	27 170	1.00	0.958 (0.920 to 0.997)	0.947 (0.902 to 0.994)	1.061 (1.006 to 1.119)	1.053 (0.994 to 1.114)
Ischaemic heart disease	10319	1.00	0.913 (0.852 to 0.978)	0.905 (0.832 to 0.985)	1.029 (0.943 to 1.123)	1.029 (0.936 to 1.131)
Congestive heart failure	6144	1.00	0.891 (0.817 to 0.972)	0.911 (0.819 to 1.014)	1.113 (0.997 to 1.242)	1.105 (0.982 to 1.244)
Cardiac dysrhythmia	4004	1.00	0.968 (0.874 to 1.072)	0.964 (0.851 to 1.093)	1.089 (0.949 to 1.251)	1.057 (0.914 to 1.223)
Cerebrovascular disease and stroke	5908	1.00	1.066 (0.981 to 1.159)	1.017 (0.922 to 1.123)	1.013 (0.907 to 1.132)	1.013 (0.902 to 1.138)

*Number of hospital admissions for zip codes with defined populations

†adjusted for race, gender, county, median income, weekend, relative humidity, temperature, age and pressure gradient

‡cardiovascular admissions were for subjects ages 45-99 years. $PM_{2.5}$, particulate matter.

A Section 508–conformant HTML version of this article is available at http://dx.doi.org/10.1289/ehp.1408436.

Forest Fire Smoke Exposures and Out-of-Hospital Cardiac Arrests in Melbourne, Australia: A Case-Crossover Study

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Background: Millions of people can potentially be exposed to smoke from forest fires, making this an important public health problem in many countries.

Objective: In this study we aimed to measure the association between out-of-hospital cardiac arrest (OHCA) and forest fire smoke exposures in a large city during a severe forest fire season, and estimate the number of excess OHCAs due to the fire smoke.

Methods: We investigated the association between particulate matter (PM) and other air pollutants and OHCA using a case-crossover study of adults (≥ 35 years of age) in Melbourne, Australia. Conditional logistic regression models were used to derive estimates of the percent change in the rate of OHCA associated with an interquartile range (IQR) increase in exposure. From July 2006 through June 2007, OHCA data were collected from the Victorian Ambulance Cardiac Arrest Registry. Hourly air pollution concentrations and meteorological data were obtained from a central monitoring site.

Results: There were 2,046 OHCAs with presumed cardiac etiology during our study period. Among men during the fire season, greater increases in OHCA were observed with IQR increases in the 48-hr lagged PM with diameter ≤ 2.5 μm ($PM_{2.5}$) (8.05%; 95% CI: 2.30, 14.13%; IQR = 6.1 μg/m^3) or ≤ 10 μm (PM_{10}) (11.1%; 95% CI: 1.55, 21.48%; IQR = 13.7 μg/m^3) and carbon monoxide (35.7%; 95% CI: 8.98, 68.92%; IQR = 0.3 ppm). There was no significant association between the rate of OHCA and air pollutants among women. One hundred seventy-four "fire-hours" (i.e., hours in which Melbourne's air quality was affected by forest fire smoke) were identified during 12 days of the 2006/2007 fire season, and 23.9 (95% CI: 3.1, 40.2) excess OHCAs were estimated to occur due to elevations in $PM_{2.5}$ during these fire-hours.

Conclusions: This study found an association between exposure to forest fire smoke and an increase in the rate of OHCA. These findings have implications for public health messages to raise community awareness and for planning of emergency services during forest fire seasons.

Citation: Dennekamp M, Straney LD, Erbas B, Abramson MJ, Keywood M, Smith K, Sim MR, Glass DC, Del Monaco A, Haikerwal A, Tonkin AM. 2015. Forest fire smoke exposures and out-of-hospital cardiac arrests in Melbourne, Australia: a case-crossover study. Environ Health Perspect 123:959–964; http://dx.doi.org/10.1289/ehp.1408436

Introduction

Millions of people worldwide can potentially be exposed to seasonal high levels of smoke from forest (bush or wild) fires, making this an important public health problem. Because forest fires are predicted to increase in frequency and severity (Confalonieri et al. 2007) and smoke from these fires can travel long distances, it is important to understand the impact of these seasonal high peak smoke concentrations.

The smoke from forest fires consists of many different constituents, but the pollutant most significantly increased during smoke episodes is $PM_{2.5}$ (particles with an aerodynamic diameter ≤ 2.5 μm) (Reisen et al. 2011). On days without forest fire smoke, $PM_{2.5}$ makes up approximately 40% of PM_{10} (particles with an aerodynamic diameter ≤ 10 μm) (Chan et al. 2008). During forest fires, this proportion increases dramatically (Reisen et al. 2013). Studies around the world have observed particulate matter (PM) concentrations during forest fires well above the recommended air quality standards (Johnston et al. 2011; Morgan et al. 2010; Reisen et al. 2011, 2013; Sapkota et al. 2005; Schranz et al. 2010). The World Health Organization (2006) 24-hr average air quality guideline for PM_{10} is 50 μg/m^3 and for $PM_{2.5}$ 25 μg/m^3. Other pollutants that are increased during forest fire smoke episodes, but not to the extent of $PM_{2.5}$, are ozone (O_3) and carbon monoxide (CO) (Dutkiewicz et al. 2011; Reisen et al. 2011).

A recent review concluded that several studies have found associations between forest fire smoke and respiratory morbidity (Dennekamp and Abramson 2011); however, only a few studies have investigated cardiovascular health outcomes. Of those studies investigating cardiovascular outcomes and forest fire smoke, most of them investigated hospital admissions (Delfino et al. 2009; Hanigan et al. 2008; Henderson et al. 2011; Johnston et al. 2007; Martin et al. 2013; Morgan et al. 2010; Mott et al. 2005). These studies either showed no association or inconsistent results. Two of the studies have found a weak association with hospital admissions, but only for indigenous people (Hanigan et al. 2008; Johnston et al. 2007). A comprehensive study from Sydney, Australia, found a small increase in nonaccidental mortality at a lag of 1 day after exposure to forest fire smoke [odds ratio (OR) = 1.05; 95% confidence interval (CI): 1.00, 1.10] (Johnston et al. 2011).

We have previously shown an association between urban $PM_{2.5}$ concentrations and out-of-hospital cardiac arrest (OHCA) (Dennekamp et al. 2010; Straney et al. 2014). This poses the question: What are the health effects of exposure to episodes of forest fire smoke, where the $PM_{2.5}$ concentrations may be many times higher than urban background concentrations and when air quality standards are regularly exceeded?

To our knowledge, this is the first study to investigate the association between OHCA and forest fire smoke exposure. OHCA is potentially a better outcome to investigate than hospital admissions, because most patients who present with a cardiac arrest with presumed cardiac etiology die before reaching a hospital (Stub et al. 2011). As a result, it is likely that hospital studies will miss a substantial number of relevant cases of acute cardiac events.

The southeast of Australia experienced a very severe forest fire season in the summer of 2006–2007, and > 1 million hectares

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Supplemental Material is available online (http://dx.doi.org/10.1289/ehp.1408436).

We thank Ambulance Victoria, Environment Protection Authority Victoria, CSIRO Marine and Atmospheric Sciences, and the Bureau of Meteorology for providing data.

The study was funded by the National Health & Medical Research Council. M.D. is supported by the Centre for Air quality and health Research and evaluation. M.J.A. holds unrelated research grants from Pfizer and Boehringer Ingelheim.

The authors declare they have no actual or potential competing financial interests.

Received: 17 March 2014; Accepted: 17 March 2015; Advance Publication: 20 March 2015; Final Publication: 1 October 2015.

of land were burnt. Smoke from the fires traveled long distances and covered the city of Melbourne on several days. This, together with a detailed ambulance registry on OHCAs in Melbourne, provided a unique opportunity to investigate the association between forest fire smoke and cardiac arrests in a large urban population during a severe forest fire season.

Methods

Study population and outcome data. OHCA data covering July 2006 through June 2007 were drawn from Ambulance Victoria's Victorian Ambulance Cardiac Arrest Registry (VACAR) (http://www.ambulance.vic.gov.au/Research/Latest-Research.html). Ambulance Victoria follows the Utstein style criteria, an established set of common definitions for cardiac arrest (Cummins et al. 1991; Jacobs et al. 2004). The VACAR captures all cardiac arrests attended by the ambulance service (Fridman et al. 2007); it is one of the largest and most comprehensive cardiac registries in the world and includes data on age, sex, and exact time of the emergency call on an individual basis.

OHCAs were included if they occurred in metropolitan Melbourne, had presumed cardiac etiology, and occurred in those ≥ 35 years of age. We excluded those < 35 years of age because it was more difficult to determine possible cardiac etiology (e.g., due to genetic diseases) (Deasy et al. 2011). Of the total number of OHCAs attended by ambulance personnel, about 80% had presumed cardiac etiology and were included in the analysis (Dennekamp et al. 2010). Reasons for exclusions have been detailed elsewhere (Dennekamp et al. 2010), but included OHCAs due to road traffic accidents or other trauma, overdoses, terminal illness, or an underlying respiratory cause.

Ambient air pollution and meteorology data. Hourly average $PM_{2.5}$, PM_{10}, CO, O_3, nitrogen dioxide (NO_2), and sulfur dioxide (SO_2) were obtained from the Environment Protection Authority (EPA) Victoria (http://www.epa.vic.gov.au) using a central monitoring station in inner suburban Melbourne. Hourly average observations of temperature and relative humidity were obtained from the Bureau of Meteorology monitoring site at Melbourne Airport.

Fire season and fire-hours. The fire season for the purposes of this paper was defined as the period from November 2006 through March 2007 because this is the annual fire danger season in Victoria. This period has the highest "fire danger ratings" (which is an indication of how dangerous a fire would be if it started), and the vast majority of days on which total fire bans were declared in Victoria occurred within this period (30 total fire ban days were declared in 2006 and 2007, and only 3 fell outside this period).

"Fire-hours" are defined as the periods when the Melbourne population was most likely to have been affected by forest fire smoke, and they can be identified by a combination of chemical transport modeling and observed increases in particle and gas concentrations during forest fires. Details of the criteria for the identification of fire-hours are presented in Supplemental Material (see Supplemental Material, "Criteria for the identification of fire-hours," and Supplemental Material, Figures S1 and S2 and Table S1). In summary, fire-hours were identified as those when the hourly $PM_{2.5}$ concentration was > 50 μg/m^3, the hourly carbon monoxide concentration was > 50 ppm, and the back trajectories for air masses at 1,000 m elevation were in the northwest to northeast sector (315° to 45°) where the forest fires were occurring.

Statistical analysis. **Case-crossover analysis.** A case-crossover analysis was conducted using a time-stratified referent period to select control exposures associated with each index case, where case exposure was the exposure in the hour the OHCA occurred. The reference exposures were the exposures in the day and hour of the case on all days falling within the same month and on the same day of the week as the case. This approach eliminated confounding by hour of the day, day of the week, and monthly trends and also seasonal and long-term trends in the exposure variables (Bateson and Schwartz 1999, 2001; Maclure 1991).

OHCA was the binary outcome (dependent) variable in the analysis. The exposure variables were hourly average pollutant concentrations. Analyses were done for lag 0 (hour of arrest), lag 1 (hour before arrest), lag 2, and so on, and average concentrations of lag 0–2 (average of hour of arrest, lag 1, and lag 2), lag 0–3, lag 0–4, lag 0–8, lag 0–12, lag 0–24, and lag 0–48. In addition, analyses were done using the whole year (from 1 July 2006 through 30 June 2007) and for the fire season only (1 November 2006 through 31 March 2007). Stratification was done by sex, age group (35–64, 65–74, ≥ 75 years), and both age and sex.

Temperature and relative humidity were included as potential confounders. Conditional logistic regression models were used to evaluate the association between the pollutants and OHCA. The parameter estimates from these models may be interpreted as proportional changes in the odds (also referred to as rate in this manuscript), calculated from the odds ratios for the interquartile range (IQR) of the pollutant. The percentage difference in the odds (rate) was calculated from the OR using the formula: $(OR - 1) \times 100$.

As a first step, single-pollutant models were developed, followed by multi-pollutant models, which included those pollutants that showed associations.

Calculation of excess OHCAs attributable to exposure to $PM_{2.5}$ due to forest fire smoke. Using the fire-hour data, we merged the hourly pollution data such that each pollutant recording had a binary variable indicating whether that hour was associated with a forest fire period. We constructed a second binary variable with a default value of 0, but equal to 1 where the current or any of the preceding 47 hr included fire-hours periods. We calculated the total number of hours where at least 1 hr in the preceding 48 hr was associated with forest fire smoke. This represented the risk period.

We used two approaches for estimating the number of OHCA attributable to the forest fires: *a*) a model-derived estimate, using the OR for and IQR increase of $PM_{2.5}$ levels in the 48 hr preceding the arrest, and *b*) a direct calculation based on the difference in the rates between the fire-hours and non–fire-hours. For details of these two approaches, see Supplemental Material, "Methodology: calculation of excess out-of-hospital cardiac arrests."

All analyses were conducted using Stata (version 12.1; StataCorp, College Station, TX, USA). *p*-Values < 0.05 were considered statistically significant.

Results

Study population and exposure description. A total of 2,046 OHCAs occurred during the study period (July 2006 through June 2007) in metropolitan Melbourne; 64% were men, and the mean age was 71.8 ± 14.2 years. Of these, 783 (38%) occurred during the fire season (November 2006 through March 2007). Men were significantly younger than women when an OHCA occurred, 69.6 versus 75.8 years, respectively ($p < 0.01$).

Average hourly concentrations of air pollutants and weather data for the fire season and for the whole year are displayed in Table 1. $PM_{2.5}$ was the pollutant that showed the greatest increase when comparing concentrations between the fire season and non-fire season. Figure 1 presents the hourly $PM_{2.5}$ concentrations in December 2006 and January 2007 and clearly shows the high peak exposures that occurred during exposure to forest fire smoke. The highest hourly $PM_{2.5}$ exposure was 247.2 μg/m^3, which occurred at 1500 hours on 20 December 2006.

A total of 174 fire-hours were identified during the 2006–2007 fire season, over 12 days (Figure 1). The number of fire-hours per day ranged from 4 to 21 hr. The 12 days fell in the period between 9 December 2006 and 10 January 2007. The average

$PM_{2.5}$ concentration during the fire-hours was 106 μg/m^3.

Association between air pollution exposure and OHCA. Table 2 displays the results of a percentage increase in the rate of OHCA for an IQR increase in airborne PM and CO, both for the entire study year (June 2006–July 2007) and for the fire season (November 2006–March 2007). The results for all analyses can be found in Supplemental Material, Table S2. An increased risk of OHCA was observed for an IQR increase in the 48-hr lagged $PM_{2.5}$, both overall (4.4%; 95% CI: 0.2, 8.7%) and among men (7.8%; 95% CI: 2.5, 13.3%). IQR increases in 24-hr and 48-hr CO levels were also associated with increased risks of OHCA among men. During the fire season, the 48-hr estimated effects of $PM_{2.5}$ remained significant, at 5.4% (95% CI: 0.9, 10.1%). In addition, the 48-hr IQR increase in CO became significantly associated with the risk of OHCA (10.0%; 95% CI: 0.6, 20.2%). Among men in the fire season, greater increases in OHCA were observed with IQR increases in the 48-hr lagged $PM_{2.5}$, PM_{10}, and CO. There was no significant association between the risk of OHCA and any of the air pollutants among women.

The hour of the reported arrest and the IQR change in O_3 for the whole year lagged 2 and 4 hr were associated with increases in OHCA in 65- to 74-year-olds for the entire duration of the study (Table 3). During the fire season period, similar results for O_3 and OHCA were observed among 65- to 74-year-olds, although the confidence intervals were much wider. In addition, among those > 75 years old, 8- and 12-hr lags were associated with an increased risk. For women, 8- and 12-hr lags of an IQR change in O_3 were associated with OHCA during the fire season.

Two-pollutant models were developed for 48-hr $PM_{2.5}$ with O_3 and CO. For O_3, both the 48-hr lag and the 2-hr lags were investigated (because significant associations were seen in the O_3 single-pollutant model for the 2-hr lag), and $PM_{2.5}$ remained significant (4.7%; 95% CI: 0.4, 9.3% and 7.5%; 95% CI: 2.0, 13.3, respectively). However, O_3 became nonsignificant (–4.7%; 95% CI: –18.4, 11.3% and 2.5%; 95% CI: –10.2, 17.0%, respectively). The two-pollutant models for $PM_{2.5}$ and CO (48-hr average) resulted in both associations becoming nonsignificant (3.71%; 95% CI: –1.2, 8.9% and 2.0%; 95% CI –6.3, 10.9%, respectively). However, restricting this analysis to males only resulted in a significant association with $PM_{2.5}$ but not CO (6.7%; 95% CI: 0.5, 13.3% and 3.3%; 95% CI: –7.11, 14.9, respectively). The correlation between $PM_{2.5}$ and O_3 was 0.24, and with $PM_{2.5}$ and CO was 0.37.

Excess arrests attributable to forest fire smoke. Two methods were used to estimate the number of excess arrests that were attributable to forest fire smoke (see Supplemental Material, "Methodology: calculation of excess out-of-hospital cardiac arrests"). Using the model-derived calculation, we estimated that 23.9 (95% CI: 3.1, 40.2) excess arrests were associated with the fire-hours; and using the direct calculation, we estimated that 28.9 (95% CI: 3.8, 52.9) excess arrests were associated with the fire-hours.

The mean temperature was higher in the hours that were identified as being affected

Table 1. Average of hourly air pollution and meteorological data for the whole year (1 July 2006–30 June 2007), for the risk period,[a] and for the nonrisk period.[b]

Exposure	*n*, whole year	Mean, whole year	Mean, risk period	Mean, nonrisk period	Ratio risk/ nonrisk period	Percentile cut point, whole year			
						25th	50th	75th	IQR
$PM_{2.5}$ (μg/m^3)	8,590	7.6	32.4	6.3	5.2	2.4	4.8	8.5	6.1
PM_{10} (μg/m^3)	8,618	21.0	55.2	19.2	2.9	11.3	16.9	25.0	13.7
CO (ppm)	8,200	0.42	0.51	0.42	1.2	0.2	0.3	0.5	0.3
O_3 (ppb)	8,201	17.1	33.3	16.2	2.1	6	16	23	17
NO_2 (ppb)	8,226	11.3	8.8	11.4	0.8	5	9	16	11
SO_2 (ppb)	8,177	0.84	0.6	0.86	0.7	0	1	1	1
Temperature (°C)	8,708	14.9	21.6	14.5	1.5	10.2	13.9	18.3	8.1
Relative Humidity (%)	8,708	64.8	45.6	65.8	0.7	50	68	82	32

[a]Those hours where at least 1 "fire-hour" occurred in the previous 48 hr. [b]Those hours in the whole year except for the risk period.

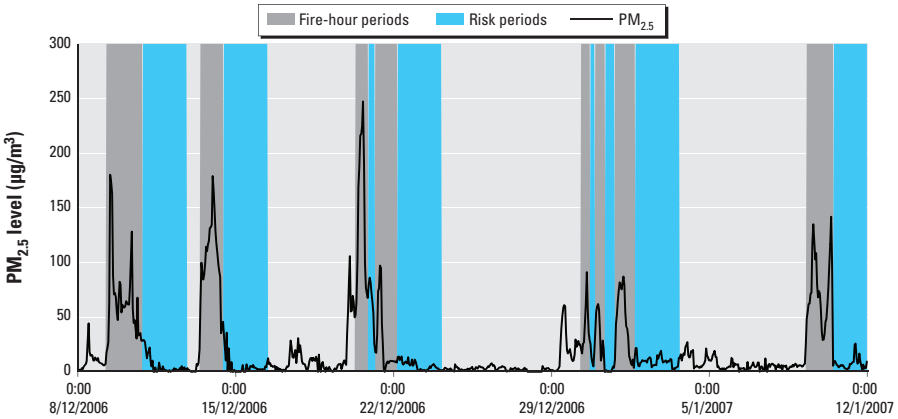

Figure 1. Hourly average $PM_{2.5}$ concentration in Melbourne from 8 December 2006 through 12 January 2007 (0:00, midnight). The dark gray areas represent the "fire-hours" (periods with forest fire smoke), and the blue areas represent the "risk period" (at least 1 fire-hour in the previous 48 hr).

Table 2. Estimated percentage difference (95% CI) in the rate of out-of-hospital cardiac arrest for an IQR increase in each air pollutant[a] using conditional logistic regression models.[b,c]

Study population	Hourly lags	$PM_{2.5}$	PM_{10}	CO
Whole year (July 2006–June 2007)				
Total (*n* = 2,046)	0	1.3 (–1.0, 3.8)	–0.2 (–4.1, 3.8)	–0.6 (–4.8, 3.9)
	0–24	3.0 (–0.3, 6.5)	3.9 (–1.5, 9.6)	2.7 (–3.3, 9.2)
	0–48	4.4 (0.2, 8.7)*	4.0 (–2.4, 10.8)	5.6 (–1.6, 13.2)
Men (*n* = 1,311)	0	2.2 (–0.7, 5.3)	1.4 (–3.5, 6.5)	1.1 (–4.3, 6.9)
	0–24	4.9 (0.7, 9.3)*	6.6 (–0.2, 13.9)	8.0 (0.1, 16.6)*
	0–48	7.8 (2.5, 13.3)**	8.4 (0.1, 17.3)*	10.0 (0.6, 20.2)*
Women (*n* = 735)	0	–0.2 (–4.2, 3.9)	–2.9 (–9.3, 3.9)	–4.1 (0.6, 20.2)
	0–24	–0.4 (–6.0, 5.5)	–0.9 (–9.6, 8.6)	–6.0 (–15.1, 4.1)
	0–48	–1.8 (–8.6, 5.4)	–3.4 (–13.3, 7.7)	–1.4 (–15.1, 4.1)
Fire season (November 2006–March 2007)				
Total (*n* = 783)	0	1.9 (–0.6, 4.5)	3.0 (–1.4, 7.5)	3.9 (–6.0, 14.8)
	0–24	3.5 (–0.1, 7.3)	7.0 (0.8, 13.6)*	16.5 (–0.1, 35.8)
	0–48	5.4 (0.9, 10.2)*	7.7 (0.3, 15.8)*	24.6 (4.5, 48.0)*
Men (*n* = 500)	0	2.5 (–0.7, 5.7)	4.5 (–1.0, 10.3)	6.9 (–5.8, 21.3)
	0–24	4.7 (0.1, 9.4)*	8.3 (0.6, 16.6)*	24.6 (2.9, 50.8)*
	0–48	8.1 (2.3, 14.1)**	11.1 (1.6, 21.5)*	35.7 (9.0, 68.9)**
Women (*n* = 283)	0	0.9 (–3.3, 5.2)	0.4 (1.5, 21.5)	–1.3 (–16.2, 16.2)
	0–24	1.1 (–4.9, 7.6)	4.1 (–6.0, 15.3)	0.9 (–22.4, 31.4)
	0–48	0.2 (–7.2, 8.2)	1.3 (–10.4, 14.4)	4.3 (–22.1, 39.8)

[a]IQR is based on the distribution of the whole year. IQRs are as follows: 6.1 μg/m^3 ($PM_{2.5}$), 13.7 μg/m^3 (PM_{10}), and 0.3 ppm for CO. [b]No significant results for less than 24-hr rolling average. [c]Adjusted for temperature and relative humidity. *$p < 0.05$. **$p < 0.01$.

by forest fire smoke when compared with the other hours throughout the year (21.65 vs. 14.53°C). However, each degree increase in temperature was associated with only a 0.02% increase in risk of OHCA, and after adjusting for this there was no change in the estimated number of excess arrests.

Discussion

This study shows that exposure to forest fire smoke in the 2006–2007 Victoria fire season was associated with an increased risk of having an OHCA in Melbourne, its capital city with a population of about 4 million. In addition, 24–29 excess OHCAs were estimated to have occurred in Melbourne because the air quality was affected by smoke from the forest fires.

Although hourly air quality data were available, and several short-term averages (including 1, 2, 4, 8, and 12 hr) were investigated, the strongest association was found with the 24-hr and 48-hr rolling averages for $PM_{2.5}$ and CO before the OHCA occurred, suggesting that there was a delayed or cumulative association. This association was seen predominantly in men. We are not aware of any plausible reasons why the associations were seen particularly in men, and did not have data concerning recognized cardiac risk factors for coronary artery disease. However, almost two-thirds of the OHCA during the 2006–2007 fire season occurred in men, and men having OHCAs were significantly younger than women. Our observations could possibly reflect only the higher age-related incidence of coronary artery disease and OHCA and effects on the background of greater abnormalities of other risk factors in men. Perhaps it is related to confounding factors such as smoking or blood pressure, or even other factors such as behavioral differences (e.g., time spent outside could play a role). We are aware of no other studies to date that have investigated OHCA and $PM_{2.5}$ during forest fire smoke events. However, of the studies investigating urban $PM_{2.5}$ and OHCA, some have reported a strong association for men (Dennekamp et al. 2010; Ensor et al. 2013) whereas others did not find this (Rosenthal et al. 2008; Silverman et al. 2010; Sullivan et al. 2003). A study from Launceston, Australia, looking at cardiovascular mortality found similar sex-specific observations. The air pollution there derived mainly from biomass combustion (residential woodsmoke), but the authors found a significant improvement in cardiovascular mortality when air pollution decreased, though this was not found in women (Johnston et al. 2013).

For O_3, when we analyzed all OHCAs, the coefficients were not significantly elevated. Only when we stratified by age and sex were significant positive associations observed, and only at rolling averages of ≤ 12 hr. In the literature, the studies investigating the association between urban O_3 and OHCA show inconsistent results. Some did not find associations (Dennekamp and Abramson 2011; Silverman et al. 2010) and some very recent studies did (Ensor et al. 2013; Raza et al. 2014; Rosenthal et al. 2013). Of the latter, one study found statistical associations with O_3 exposure 2 hr, 24 hr, and 72 hr before an OHCA (Raza et al. 2014); another found significant associations only with lag 2 days exposures and not hourly lagged exposures (Rosenthal et al. 2013); and another found associations with both hourly and daily (maximum 8-hr average) exposure (Ensor et al. 2013; Raza et al. 2014; Rosenthal et al. 2013).

The $PM_{2.5}$ associations found here are relatively similar to those of our previous study, in which we investigated the association between urban air pollution and OHCA in Melbourne (Dennekamp et al. 2010). A 4.25-μg/m^3 increase in $PM_{2.5}$ was associated with a a 3.61% increase in risk of OHCA (95% CI: 1.29, 5.99%) in our urban air pollution study, and of 3.75% (95% CI: 0.60, 7.00%) during the fire season in the present study. However, even though the estimated effects may be similar, the change in air quality concentrations is much larger during forest fire episodes compared with non–forest fire episodes, hence resulting in a measureable excess in OHCAs during relatively short smoke episodes.

The estimated effects for $PM_{2.5}$, PM_{10}, O_3, and CO were almost all larger and stronger in the fire season despite the smaller sample size. This association was confirmed by the analysis using fire-hours, which showed a significant increase in the number of OHCAs.

Our findings suggest that $PM_{2.5}$ seems to be the key pollutant associated with excess OHCA during forest fires. First of all, in our study $PM_{2.5}$ increases the most during a forest fire season compared with any of the other pollutants that were monitored (including PM_{10}). And even with introduction of O_3 and CO, the $PM_{2.5}$ association remained consistent. However, this should be interpreted with caution because of the high correlation between the pollutants, particularly between $PM_{2.5}$ and CO (r = 0.37).

Previous studies have not shown consistent associations between exposure to forest fire smoke and cardiovascular outcomes (Delfino et al. 2009; Hanigan et al. 2008; Henderson et al. 2011; Johnston et al. 2007, 2011; Martin et al. 2013; Morgan et al. 2010; Mott et al. 2005; Schranz et al. 2010). Most of these studies investigated hospital admissions for cardiovascular disease in general or for a specific disease type (e.g., myocardial infarction), whereas ours, to our knowledge, is the first study to clearly show an association between ambulance data for OHCA and forest fire smoke. Perhaps we find an association here because the vast majority of OHCAs are fatal, and therefore these cases do not appear in hospital emergency presentations or hospital

Table 3. Estimated percentage difference in the rate of out-of-hospital cardiac arrest for an IQR increase in O_3[a] of 17 ppb using conditional logistic regression models.

	Age group (years)				Sex	
Hourly lags	≥ 35	35–64	65–74	≥ 75	Men	Women
Whole year (1 July 2006–30 June 2007)						
0	5.9 (–4.6, 17.5)	–7.9 (–23.2, 10.3)	43.8 (10.8, 86.5)**	7.5 (–7.6, 25.0)	6.5 (–6.6, 21.3)	6.8 (–10.3, 27.0)
0–2	4.3 (–6.1, 15.9)	–10.5 (–25.6, 7.7)	42.8 (10.4, 84.8)**	5.6 (–9.3, 23.0)	4.4 (–8.5, 19.2)	5.7 (–11.1, 25.7)
0–4	3.4 (–7.0, 15.1)	–10.7 (–26.2, 8.2)	36.1 (5.2, 76.0)*	4.9 (–10.0, 22.2)	3.9 (–9.1, 18.8)	4.2 (–12.8, 24.5)
0–8	6.9 (–4.2, 19.2)	–5.4 (–22.3, 15.2)	25.2 (–3.6, 62.6)	10.5 (–5.2, 28.8)	3.8 (–9.4, 19.0)	13.9 (–5.2, 36.8)
0–12	7.9 (–4.1, 21.3)	–5.5 (–23.6, 16.9)	20.0 (–8.5, 57.1)	13.9 (–3.5, 34.4)	3.6 (–10.5, 19.9)	17.1 (–3.8, 42.4)
0–24	4.3 (–8.7, 19.2)	–6.8 (–26.9, 18.9)	23.7 (–9.1, 68.4)	7.3 (–11.0, 29.3)	1.8 (–13.8, 20.2)	9.6 (–12.3, 36.9)
0–48	–0.3 (–14.2, 15.9)	–1.8 (–25.0, 28.5)	2.4 (–28.2, 46.2)	0.6 (–18.6, 24.3)	0.7 (–16.5, 21.5)	–2.5 (–24.2, 25.4)
Fire season (1 November 2006–31 March 2007)						
0	9.1 (–4.7, 25.0)	2.4 (–17.6, 27.3)	48.9 (2.4, 116.6)*	8.5 (–11.4, 33.0)	5.4 (–11.2, 25.2)	17.8 (–5.8, 47.3)
0–2	8.6 (–5.3, 24.5)	–2.5 (–22.3, 22.4)	57.0 (8.3, 127.6)*	9.3 (–10.8, 33.9)	4.7 (–12.1, 24.7)	17.3 (–5.9, 46.3)
0–4	11.5 (–3.0, 28.0)	0.1 (–20.9, 26.8)	56.2 (8.3, 125.3)*	11.8 (–8.6, 36.9)	7.9 (–9.6, 28.7)	19.4 (–4.7, 49.7)
0–8	20.0 (3.8, 38.6)	9.6 (–14.6, 40.5)	45.5 (0.7, 110.3)*	23.4 (0.5, 51.5)*	11.2 (–7.2, 33.4)	38.8 (9.0, 76.7)**
0–12	24.1 (5.4, 46.2)	12.0 (–15.5, 48.4)	20.9 (–18.1, 78.5)	38.1 (9.0, 75.1)**	12.4 (–8.5, 38.1)	49.0 (13.4, 95.7)**
0–24	15.2 (–5.2, 40.1)	14.8 (–18.2, 61.1)	–8.7 (–43.2, 46.8)	30.3 (–1.4, 72.2)	5.9 (–17.2, 35.5)	33.7 (–3.2, 84.6)
0–48	8.1 (–12.5, 33.5)	21.2 (–15.3, 73.4)	–25.9 (–57.0, 27.8)	13.9 (–15.6, 53.7)	4.8 (–19.5, 36.5)	14.1 (–19.8, 62.3)

[a]Adjusted for temperature and relative humidity. *$p < 0.05$. **$p < 0.01$.

admission records. From July 2006 through June 2007 the proportion of OHCA attended by ambulance in Melbourne (≥ 35 years of age and presumed cardiac) who were declared deceased at the scene and not transported to hospital was 78.4%. The severe outcome of arrest that could be associated with air pollution would result in a selection bias for studies that relied on hospital admissions.

Although no studies have previously investigated the association between OHCA and forest fire smoke, several studies have investigated urban $PM_{2.5}$ and OHCA, with inconsistent findings. No significant associations were found in some (Levy et al. 2001; Raza et al. 2014; Sullivan et al. 2003); another study found null results overall except for a subgroup that had arrests that were witnessed by bystanders (Rosenthal et al. 2008); and a few large studies did find associations between urban $PM_{2.5}$ and OHCAs (Dennekamp et al. 2010; Ensor et al. 2013; Silverman et al. 2010). Even though the present study was rather small, it is likely we found a significant association due to the large $PM_{2.5}$ concentrations that occur during a forest fire season, and hence we were able to detect a significant increase in excess OHCAs.

Our data could not provide further insights into the underlying mechanisms involved. However, these have been extensively reviewed elsewhere (Brook et al. 2010). We have hypothesized that PM exposure may cause systemic inflammation that can lead to an increase in blood coagulability with resultant coronary thrombosis (Seaton and Dennekamp 2003). In addition, the risk of potentially lethal cardiac arrhythmias and cardiac arrest may be increased, possibly partly because of impaired cardiac autonomic control mechanisms (Brook et al. 2010; Luttmann-Gibson et al. 2010). However, this hypothesis is not supported by two large studies in patients with implantable defibrillators; the authors did not find associations between PM levels and tachyarrhytmic events (Anderson et al. 2010; Metzger et al. 2007). Studies have shown O_3 exposure to be associated with disturbed heart rate variability (Kop et al. 2001; Park et al. 2005; Utell et al. 2002).

Strengths and limitations. A major strength of this study is the health outcome data. The Victorian Ambulance Cardiac Arrest Registry is comprehensive, covering close to 100% of the OHCAs that occur in Melbourne and are attended by ambulance.

A limitation of this study (and with most other air pollution studies) is the use of one central monitoring location in Melbourne for the air pollutant concentrations that was used to represent exposure for the whole of metropolitan Melbourne. At the time of the study there were only two EPA Victoria monitors measuring $PM_{2.5}$, both in inner Melbourne. One of the sites has a significant number of data missing during the study period, whereas the other site had near complete data for all pollutants. Where data were available, we found that the correlation between the two monitors was very high (R = 0.95), and therefore the data from one monitoring station were used in this study. The exposure misclassification resulting from this is likely to underestimate the association and the number of attributable arrests. The fire-hour data were derived from a location 30 km from the EPA monitoring station. This would explain the fact that Figure 1 shows that on two occasions the concentration increases sharply and the fire-hour starts a couple of hours later. However, considering our analysis used the 48 hr before an OHCA, this is unlikely to have a measurable effect on our results. Modeled data that would more accurately reflect probable exposure in areas that do not have monitoring are recommended for future studies.

Another limitation is that it is not possible to draw conclusions regarding susceptible subgroups in the community, because we do not have detailed information on individual risk factors and co-morbidities. This is not a problem statistically, because in a case-crossover design these factors are adjusted for by design. However, the utility of the findings are reduced as advice can be provided only in general terms, rather than being specific to different risk groups.

In conclusion, the results suggest that exposure to forest fire smoke is associated with the occurrence of out-of-hospital cardiac arrests in men. It is estimated that in the 2006–2007 forest fire season the smoke was responsible for 24–29 excess arrests in Melbourne. The impact of this is likely to increase in the future, because forest fires are likely to increase in frequency and severity in many countries where forest fires occur in close proximity to large population centers. These findings have implications for public health messages to raise community awareness and for planning of emergency services during forest fire seasons.

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JAMA | Original Investigation

Association of Short-term Exposure to Air Pollution With Mortality in Older Adults

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IMPORTANCE The US Environmental Protection Agency is required to reexamine its National Ambient Air Quality Standards (NAAQS) every 5 years, but evidence of mortality risk is lacking at air pollution levels below the current daily NAAQS in unmonitored areas and for sensitive subgroups.

OBJECTIVE To estimate the association between short-term exposures to ambient fine particulate matter ($PM_{2.5}$) and ozone, and at levels below the current daily NAAQS, and mortality in the continental United States.

DESIGN, SETTING, AND PARTICIPANTS Case-crossover design and conditional logistic regression to estimate the association between short-term exposures to $PM_{2.5}$ and ozone (mean of daily exposure on the same day of death and 1 day prior) and mortality in 2-pollutant models. The study included the entire Medicare population from January 1, 2000, to December 31, 2012, residing in 39 182 zip codes.

EXPOSURES Daily $PM_{2.5}$ and ozone levels in a 1-km × 1-km grid were estimated using published and validated air pollution prediction models based on land use, chemical transport modeling, and satellite remote sensing data. From these gridded exposures, daily exposures were calculated for every zip code in the United States. Warm-season ozone was defined as ozone levels for the months April to September of each year.

MAIN OUTCOMES AND MEASURES All-cause mortality in the entire Medicare population from 2000 to 2012.

RESULTS During the study period, there were 22 433 862 million case days and 76 143 209 control days. Of all case and control days, 93.6% had $PM_{2.5}$ levels below 25 $\mu g/m^3$, during which 95.2% of deaths occurred (21 353 817 of 22 433 862), and 91.1% of days had ozone levels below 60 parts per billion, during which 93.4% of deaths occurred (20 955 387 of 22 433 862). The baseline daily mortality rates were 137.33 and 129.44 (per 1 million persons at risk per day) for the entire year and for the warm season, respectively. Each short-term increase of 10 $\mu g/m^3$ in $PM_{2.5}$ (adjusted by ozone) and 10 parts per billion (10^{-9}) in warm-season ozone (adjusted by $PM_{2.5}$) were statistically significantly associated with a relative increase of 1.05% (95% CI, 0.95%-1.15%) and 0.51% (95% CI, 0.41%-0.61%) in daily mortality rate, respectively. Absolute risk differences in daily mortality rate were 1.42 (95% CI, 1.29-1.56) and 0.66 (95% CI, 0.53-0.78) per 1 million persons at risk per day. There was no evidence of a threshold in the exposure-response relationship.

CONCLUSIONS AND RELEVANCE In the US Medicare population from 2000 to 2012, short-term exposures to $PM_{2.5}$ and warm-season ozone were significantly associated with increased risk of mortality. This risk occurred at levels below current national air quality standards, suggesting that these standards may need to be reevaluated.

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Supplemental content

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JAMA. 2017;318(24):2446-2456. doi:10.1001/jama.2017.17923

In the United States, the Clean Air Act[1] requires a review of National Ambient Air Quality Standards (NAAQS) for fine particulate matter ($PM_{2.5}$) and ozone every 5 years.[2] In 2012, the annual and 24-hour NAAQS for $PM_{2.5}$ were set to 12 μg/m^3 and 35 μg/m^3, respectively. With no annual standard for ozone, the 8-hour NAAQS for ozone was set to 70 parts per billion (ppb). Currently, the review of these standards is ongoing, with public comments expected in the fall of 2017.[3]

Several studies have provided evidence that short-term exposures to $PM_{2.5}$ and ozone were associated with mortality,[4-8] but these studies primarily included large and well-monitored metropolitan areas. While the US Environmental Protection Agency (EPA) is considering more stringent NAAQS, evidence is needed to clarify the association between mortality risk and exposure levels below the daily NAAQS and in rural and unmonitored areas.

The Clean Air Act[1] also requires the US EPA to set standards to protect "sensitive subgroups." To estimate the health risk of short-term exposure to air pollution for specific subgroups (eg, underrepresented minorities and those with low socioeconomic status, such as persons eligible for Medicaid), a large population is necessary to achieve maximum accuracy and adequate statistical power.

A case-crossover study was conducted to examine all deaths of Medicare participants in the continental United States from 2000 throughout 2012 and estimate the mortality risk associated with short-term exposures to $PM_{2.5}$ and ozone in the general population as well as in subgroups. The study was designed to estimate the association between daily mortality and air pollution at levels below current daily NAAQS to evaluate the adequacy of the current air quality standards for $PM_{2.5}$ and ozone.

Key Points

Question What is the association between short-term exposure to air pollution below current air quality standards and all-cause mortality?

Finding In a case-crossover study of more than 22 million deaths, each 10-μg/m^3 daily increase in fine particulate matter and 10–parts-per-billion daily increase in warm-season ozone exposures were associated with a statistically significant increase of 1.42 and 0.66 deaths per 1 million persons at risk per day, respectively.

Meaning Day-to-day changes in fine particulate matter and ozone exposures were significantly associated with higher risk of all-cause mortality at levels below current air quality standards, suggesting that those standards may need to be reevaluated.

Methods

This study was approved by the institutional review board at the Harvard T.H. Chan School of Public Health. As a study of previously collected administrative data, it was exempt from informed consent requirements.

Study Population

Using claims data from the Centers for Medicare & Medicaid Services, all deaths among all Medicare beneficiaries were identified during the period 2000 to 2012, providing enough power to analyze the risk of mortality associated with $PM_{2.5}$ and ozone concentrations much lower than the current standards (**Table 1**). For each beneficiary, information was extracted on the date of death, age, sex, race, ethnicity, zip code of residence, and eligibility for Medicaid (a proxy for low income) to assess the associations of mortality with $PM_{2.5}$ and ozone concentrations in potentially vulnerable subgroups. Self-reported information on race and ethnicity was obtained from Medicare beneficiary files.

Outcome

The study outcome was all-cause mortality. Individuals with a verified date of death between January 1, 2000, and December 31, 2012, were included. Individuals with an unverified date of death, or still living after December 31, 2012, were excluded.

Study Design

We estimated the association between short-term exposure to $PM_{2.5}$ (adjusted by ozone) and short-term exposure to ozone (adjusted by $PM_{2.5}$) and all-cause mortality using a case-crossover design.[9] Specifically, "case day" was defined as the date of death. For the same person, we compared daily air pollution exposure on the case day vs daily air pollution exposure on "control days." Control days were chosen (1) on the same day of the week as the case day to control for potential confounding effect by day of week; (2) before and after the case day (bidirectional sampling) to control for time trend[10,11]; and (3) only in the same month as the case day to control for seasonal and subseasonal patterns.[10,12] Individual-level covariates and zip code–level covariates that did not vary day to day (eg, age, sex, race/ethnicity, socioeconomic status, smoking, and other behavioral risk factors) were not considered to be confounders as they remain constant when comparing case days vs control days.

Environmental Data

Daily ambient levels of $PM_{2.5}$ and ozone were estimated from published and validated air pollution prediction models.[13,14] Combining monitoring data from the EPA, satellite-based measurements, and other data sets, neural networks were used to predict 24-hour $PM_{2.5}$ and 8-hour maximum ozone concentrations at each 1-km ×1-km grid in the continental United States, including locations with no monitoring sites. Cross-validation indicated good agreement between predicted values and monitoring values (R^2 = 0.84 for $PM_{2.5}$ and R^2 = 0.76 for ozone) and at low concentrations (R^2 = 0.85 when constraining to 24-hour $PM_{2.5}$ <25 μg/m^3 and R^2 = 0.75 when constraining to daily 8-hour maximum ozone <60 ppb). Details have been published elsewhere.[13,14] Warm season was defined to be from April 1 to September 30, which is the specific time window to examine the association between ozone and mortality.

Meteorological variables, including air and dew point temperatures, were retrieved from North American Regional Reanalysis data and estimated daily mean values were determined for each 32-km × 32-km grid in the continental United States.[15]

For each case day (date of death) and its control days, the daily 24-hour $PM_{2.5}$, 8-hour maximum ozone, and daily air and dew point temperatures were assigned based on zip code of residence of the individual (eAppendix 1 in the Supplement). Because we estimated air pollution levels everywhere in the continental United States, the number of zip codes included in this study was 39 182, resulting in a 33% increase compared with the number of zip codes with a centroid less than 50 km from a monitor (n = 26 115).

Table 1. Baseline Characteristics of Study Population (2000-2012)

Baseline Characteristic	Value
Case days, No.	22 433 862
Control days, No.	76 143 209
Among All Cases (n = 22 433 862), %	
Age at death, y	
≤69	10.38
70-74	13.37
75-84	38.48
≥85	37.78
Sex	
Male	44.73
Female	55.27
Race/ethnicity	
White	87.34
Black	8.87
Asian	1.03
Hispanic	1.51
Native American	0.31
Medicaid Eligibility (n = 22 433 862), %	
Ineligible	77.36
Eligible	22.64

Statistical Analysis

The relative risk (RR) of all-cause mortality associated with short-term exposures to $PM_{2.5}$ (adjusted by ozone) and warm-season ozone (adjusted by $PM_{2.5}$) was estimated by fitting a conditional logistic regression to all pairs of case days and matched control days (eAppendix 2 in the Supplement).[9] The regression model included both pollutants as main effects and natural splines of air and dew point temperatures with 3 *df* to control for potential residual confounding by weather. For each case day, daily exposure to air pollution was defined as the mean of the same day of death (lag 0-day) and 1 day prior (lag 1-day), denoted as lag 01-day.[5,16,17] Relative risk increase (RRI) was defined as RR − 1. The absolute risk difference (ARD) of all-cause mortality associated with air pollution was defined as ARD = α × (RR − 1)/RR, where α denotes the baseline daily mortality rate (eAppendix 3 in the Supplement).

The robustness of the analysis results was assessed with respect to (1) choosing the *df* used for the confounding adjustment for temperature, (2) using lag 01-day exposure as the exposure metric, (3) the definition of warm season, and (4) using only air pollution measurements from the nearest EPA monitoring sites. Splines on meteorological variables with 6 and 9 *df* yielded results with a difference of less than 5% of the standard error (eFigure 1 in the Supplement). The main analysis, which used the lag 01-day exposure, yielded the lowest values of the Akaike Information Criteria values, indicating better fit to the data (eTable in the Supplement). Different definitions of warm season yielded similar risk estimates (eAppendix 4 in the Supplement), and using exposure mea-

Table 2. Relative Risk Increase and Absolute Risk Difference of Daily Mortality Associated With Each 10-μg/m³ Increase in $PM_{2.5}$ and Each 10-ppb Increase in Ozone

	Relative Risk Increase, % (95% CI)		Absolute Risk Difference in Daily Mortality Rates, No. per 1 Million Persons at Risk per Day (95% CI)[a]	
Air Pollutant Analysis	$PM_{2.5}$	Ozone[b]	$PM_{2.5}$	Ozone[b]
Main analysis[c]	1.05 (0.95-1.15)	0.51 (0.41-0.61)	1.42 (1.29-1.56)	0.66 (0.53-0.78)
Low-exposure analysis[d]	1.61 (1.48-1.74)	0.58 (0.46-0.70)	2.17 (2.00-2.34)	0.74 (0.59-0.90)
Single-pollutant analysis[e]	1.18 (1.09-1.28)	0.55 (0.48-0.62)	1.61 (1.48-1.73)	0.71 (0.62-0.79)
Nearest monitors analysis[f]	0.83 (0.73-0.93)	0.35 (0.28-0.41)	1.13 (0.99-1.26)	0.45 (0.37-0.53)

Abbreviations: $PM_{2.5}$, fine particulate matter; ppb, parts per billion.

[a] The daily baseline mortality rate was 137.33 per 1 million persons at risk per day; the warm-season daily baseline mortality rate was 129.44 per 1 million persons at risk per day.

[b] Ozone analyses included days from the warm season only (April 1 to September 30).

[c] The main analysis used the mean of daily exposure on the same day of death and 1 day prior (lag 01-day) as the exposure metric for both $PM_{2.5}$ and ozone, and controlled for natural splines of air and dew point temperatures with 3 *df*. The main analysis considered the 2 pollutants jointly included into the regression model and estimated the percentage increase in the daily mortality rate associated with a 10-μg/m³ increase in $PM_{2.5}$ exposure adjusted for ozone and the percentage increase in daily mortality rate associated with a 10-ppb increase in warm-season ozone exposure adjusted for $PM_{2.5}$.

[d] The low-exposure analysis had the same model specifications as the 2-pollutant analysis and was constrained for days when $PM_{2.5}$ was below 25 μg/m³ or ozone below 60 ppb.

[e] The single-pollutant analysis estimated the percentage increase in the daily mortality rate associated with a 10-μg/m³ increase in $PM_{2.5}$ exposure without adjusting for ozone and the percentage increase in the daily mortality rate associated with a 10-ppb increase in ozone exposure without adjusting for $PM_{2.5}$.

[f] $PM_{2.5}$ and ozone monitoring data were retrieved from the US Environmental Protection Agency Air Quality System, which provides the daily mean of $PM_{2.5}$ and daily 8-hour maximum ozone levels at each monitoring site. Daily ozone concentrations were averaged from April 1 to September 30. Individuals were assigned to the $PM_{2.5}$ and ozone levels from the nearest monitor site within 50 km. Those living 50 km from any monitoring site were excluded.

surements from the nearest monitors resulted in attenuated, but still significant, risk estimates (**Table 2**).

The subgroup analyses were conducted by sex (male and female), race/ethnicity (white, nonwhite, and others), age (≤69, 70-74, 75-84, and ≥85 years), eligibility for Medicaid, and population density (quartiles). We fitted separate conditional logistic regressions to the data for each subgroup and obtained subgroup-specific estimates of RR and ARD. We implemented a 2-sample test for assessing statistically significant differences in the estimated RR and ARD between categories within each subgroup (eg, female vs male), based on the point estimate and standard error (se) (eAppendix 5 in the Supplement):

$$Z = \frac{RR_{male} - RR_{female}}{\sqrt{se(RR_{male})^2 + se(RR_{female})^2}}$$

The goal was to estimate mortality rate increases (both RRI and ARD) at air pollution levels well below the current daily NAAQS. The analysis was restricted to days with daily air pollution concentrations below 25 μg/m^3 for $PM_{2.5}$ and 60 ppb for ozone. We chose 25 μg/m^3 and 60 ppb instead of the current daily NAAQS (35 μg/m^3 for daily $PM_{2.5}$ and 70 ppb for 8-hour maximum ozone) because levels of $PM_{2.5}$ and ozone on most of the days included in the analysis were already below the current safety standards.

Exposure-response curves were estimated between $PM_{2.5}$ or ozone and mortality by replacing linear terms for the 2 pollutants with penalized splines for both $PM_{2.5}$ and ozone.

All analyses were performed in R software version 3.3.2 (R Foundation). Computations were run on (1) the Odyssey cluster supported by the Faculty of Arts and Sciences Division of Science, Research Computing Group at Harvard University and (2) the Research Computing Environment supported by the Institute for Quantitative Social Science in the Faculty of Arts and Sciences at Harvard University.

Results

During the study period, there were more than 22 million case days (deaths) and more than 76 million control days (Table 1). Of all case and control days, 93.6% had $PM_{2.5}$ levels below 25 μg/m^3, during which 95.2% of deaths occurred (21 353 817 of 22 433 862), and 91.1% of days had ozone levels below 60 ppb, during which 93.4% of deaths occurred (20 955 387 of 22 433 862). The baseline daily mortality rates were 137.33 and 129.44 (per 1 million persons at risk per day [per 1M per day]) for the entire year and for the warm season, respectively. The mean time between case and control days was 12.55 days (range 7-28 days), with minimal differences in air and dew point temperatures between case and control days (0.003°C and 0.01°C, respectively). During the study period, the mean concentrations of $PM_{2.5}$ and ozone were 11.6 μg/m^3 and 37.8 ppb, respectively. **Figure 1** and **Figure 2** show the daily $PM_{2.5}$ and ozone time series by state, respectively.

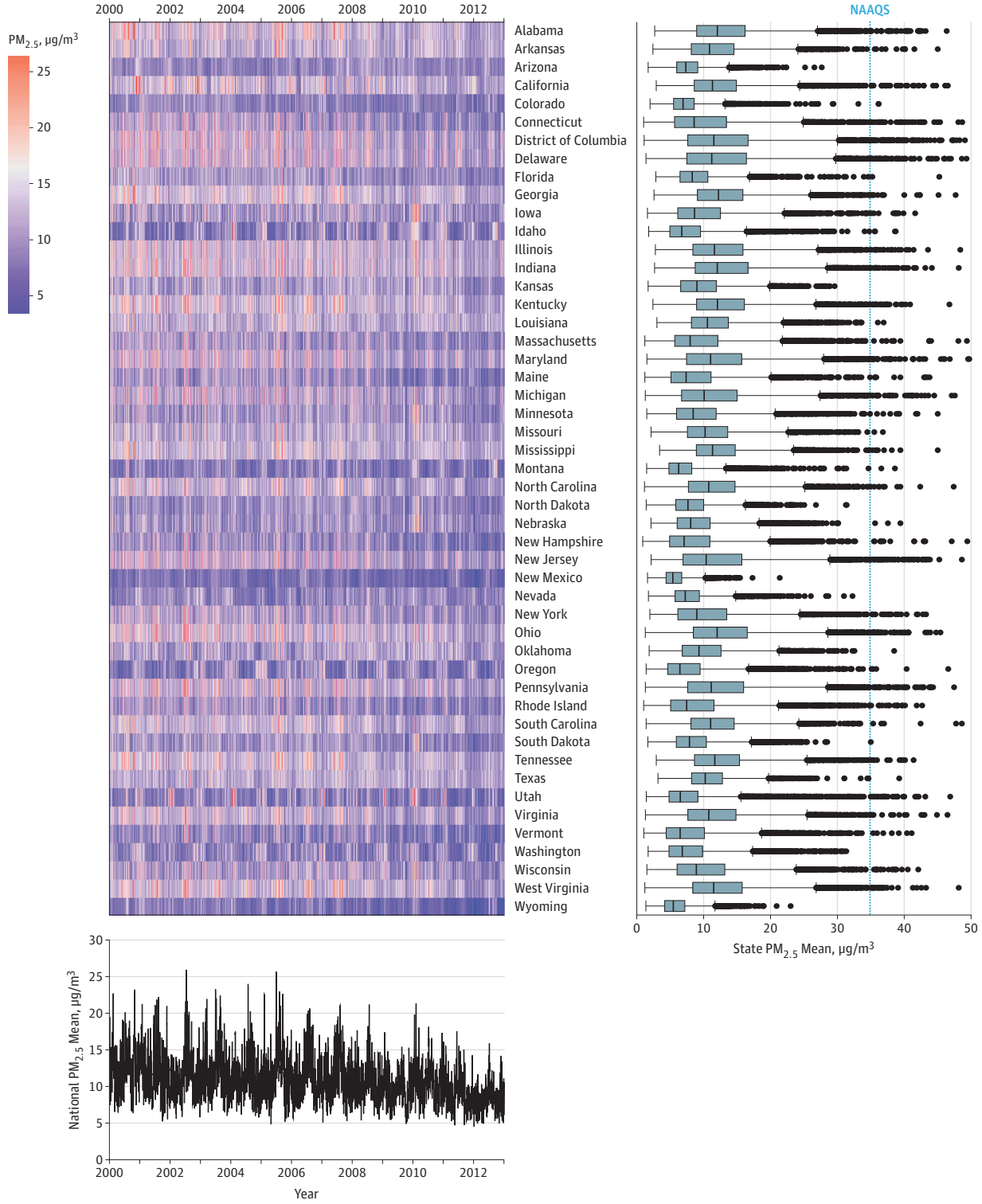
Each 10-μg/m^3 and 10-ppb increase in the lag 01-day exposure for $PM_{2.5}$ and warm-season ozone was associated with an RRI of 1.05% (95% CI, 0.95%-1.15%) and 0.51% (95% CI, 0.41%-0.61%) in the daily mortality rate. The ARDs were 1.42 (95% CI, 1.29-1.56) and 0.66 (95% CI, 0.53-0.78) per 1M per day. These associations remained significant when examining days below 25 μg/m^3 for $PM_{2.5}$ and below 60 ppb for ozone, with larger effect size estimates for both $PM_{2.5}$ and ozone (RRI: 1.61% [95% CI, 1.48%-1.74%] and 0.58% [95% CI, 0.46%-0.70%]; ARD: 2.17 [95% CI, 2.00-2.34] and 0.74 [95% CI, 0.59-0.90] per 1M per day, respectively) (Table 2). $PM_{2.5}$ was associated with higher mortality rate in some subgroups, including Medicaid-eligible individuals (RRI: 1.49% [95% CI, 1.29%-1.70%]; ARD: 3.59 [95% CI, 3.11-4.08] per 1M per day; interaction: $P < .001$), individuals older than 70 years (eg, for ≥85 years, RRI: 1.38% [95% CI, 1.23%-1.54%]; ARD: 5.35 [95% CI, 4.75-5.95] per 1M per day; interaction: $P < .001$), and females (RRI: 1.20% [95% CI, 1.07%-1.33%]; ARD: 1.56 [95% CI, 1.39-1.72] per 1M per day; interaction: $P = .02$) (**Figure 3** and **Figure 4**). The effect estimates for $PM_{2.5}$ increased with age. The effect estimate for black individuals was higher than that for white individuals ($P = .001$; eFigure 2 in the Supplement). For ozone, similar patterns were observed, but with less contrast between groups. No significant differences were found in the short-term associations between air pollution exposure ($PM_{2.5}$ and ozone) and mortality across areas with different population density levels (Figure 3 and Figure 4). Effect estimates using different lags of exposure are shown in eFigure 3 in the Supplement.

Figure 5 shows the estimated exposure-response curves for $PM_{2.5}$ and ozone. The slope was steeper at $PM_{2.5}$ levels below 25 μg/m^3 ($P < .001$), consistent with the low-exposure analysis (Table 2). Both $PM_{2.5}$ and ozone exposure-responses were almost linear, with no indication of a mortality risk threshold at very low concentrations. eFigure 4 in the Supplement shows the exposure-response curves for $PM_{2.5}$ when restricted to just the warm season and for ozone when not restricted to the warm season; results were similar.

Discussion

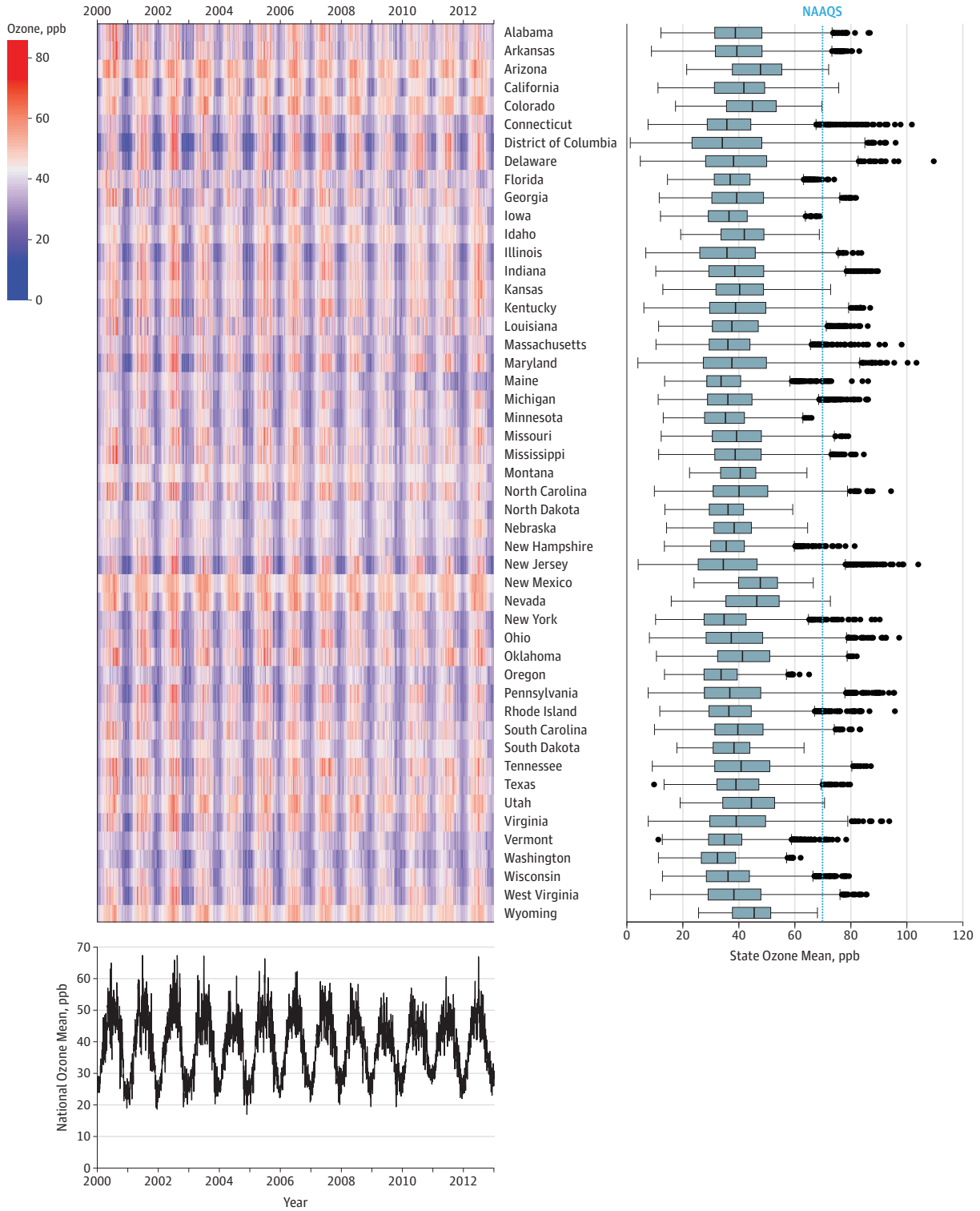
In this large case-crossover study of all Medicare deaths in the continental United States from 2000 to 2012, a 10-μg/m^3 daily increase in $PM_{2.5}$ and a 10-ppb daily increase in warm-season ozone exposures were associated with a statistically significant increase of 1.42 and 0.66 deaths per 1M per day, respectively. The risk of mortality remained statistically significant when restricting the analysis to days with $PM_{2.5}$ and ozone levels much lower than the current daily NAAQS.[18] This study included individuals living in smaller cities, towns, and rural areas that were unmonitored and thus excluded from previous time series studies. There were no significant differences in the mortality risk associated with air pollution among individuals living in urban vs rural areas. Taken together, these results provide evidence that short-term exposures to $PM_{2.5}$ and ozone, even at levels much lower than the current daily standards, are associated with increased mortality, particularly for susceptible populations.

Figure 1. Daily Mean $PM_{2.5}$ Concentrations in the Continental United States, 2000-2012



Daily mean fine particulate matter ($PM_{2.5}$) concentrations were calculated and plotted by state. The time-series plot at the bottom indicates the national daily mean values across all locations. Boxplots show the distribution of daily $PM_{2.5}$ levels for each state. The blue dashed line indicates the daily National Ambient Air Quality Standards (NAAQS) for $PM_{2.5}$ (35 µg/m^3). The line across the box, upper hinge, and lower hinge represent the median value, 75th percentile (Q3), and 25th percentile (Q1), respectively. The upper whisker is located at the smaller of the maximal value and Q3 + 1.5 × interquartile range; the lower whisker is located at the larger of the minimal value and Q1 – 1.5 × interquartile range. Any values that lie beyond the upper and lower whiskers are outliers.

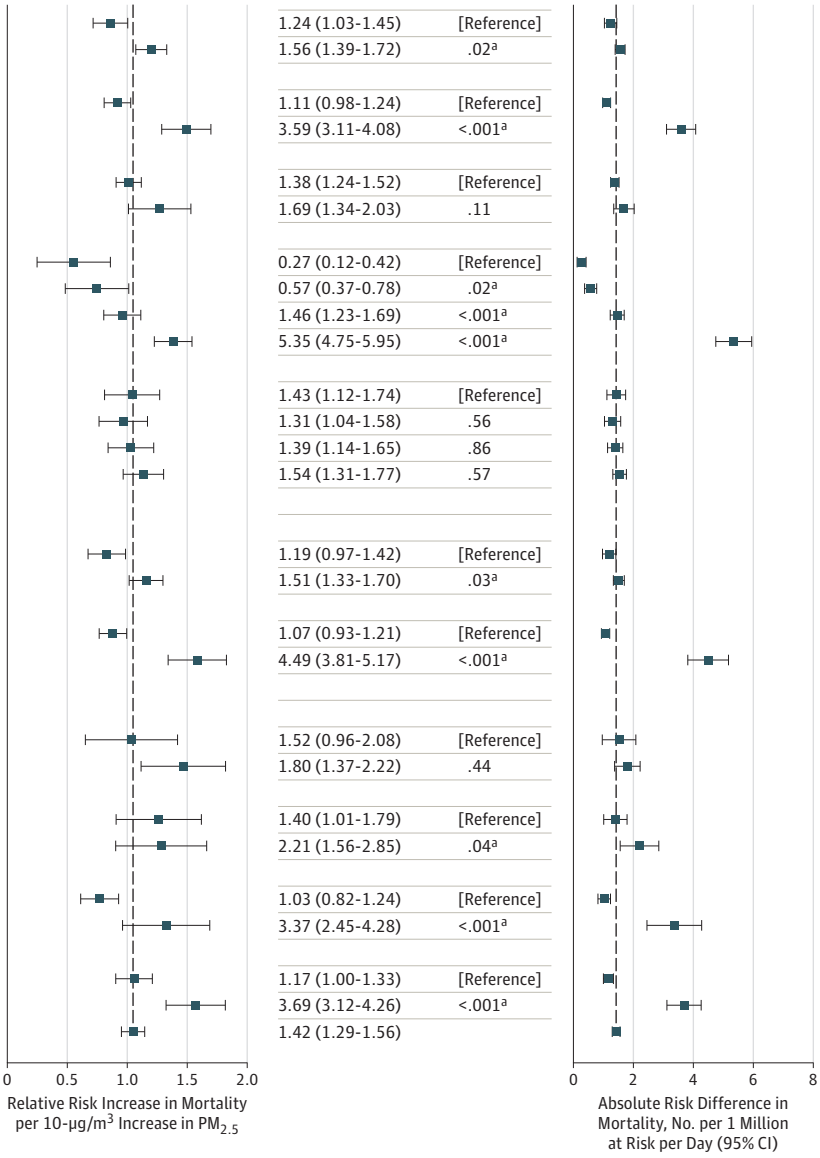
Figure 2. Daily 8-Hour Maximum Ozone Concentrations in the Continental United States, 2000-2012



Daily mean 8-hour maximum ozone concentrations were calculated and plotted by state. The time-series plot at the bottom indicates the national daily mean values across all locations. Boxplots show the distribution of daily ozone levels for each state. The blue dashed line indicates the daily National Ambient Air Quality Standards (NAAQS) for ozone (70 parts per billion [ppb]). The line across the box, upper hinge, and lower hinge represent the median value, 75th percentile (Q3), and 25th percentile (Q1), respectively. The upper whisker is located at the smaller of the maximal value and Q3 + 1.5 × interquartile range; the lower whisker is located at the larger of the minimal value and Q1 − 1.5 × interquartile range. Any values that lie beyond the upper and lower whiskers are outliers.

Figure 3. Relative Risk Increase and Absolute Risk Difference of Daily Mortality Associated With 10-μg/m^3 Increase in Fine Particulate Matter ($PM_{2.5}$)

Model	Relative Risk Increase in Mortality per 10-μg/m^3 Increase in $PM_{2.5}$, % (95% CI)	*P* Value for Effect Modification	Absolute Risk Difference in Mortality, No. per 1 Million at Risk per Day (95% CI)	*P* Value for Effect Modification
Sex				
Male	0.86 (0.72-1.00)	[Reference]	1.24 (1.03-1.45)	[Reference]
Female	1.20 (1.07-1.33)	<.001[a]	1.56 (1.39-1.72)	.02[a]
Medicaid eligibility				
Noneligible	0.92 (0.81-1.03)	[Reference]	1.11 (0.98-1.24)	[Reference]
Eligible	1.49 (1.29-1.70)	<.001[a]	3.59 (3.11-4.08)	<.001[a]
Race/ethnicity				
White	1.01 (0.91-1.12)	[Reference]	1.38 (1.24-1.52)	[Reference]
Nonwhite	1.27 (1.01-1.53)	.07	1.69 (1.34-2.03)	.11
Age, y				
≤69	0.55 (0.25-0.86)	[Reference]	0.27 (0.12-0.42)	[Reference]
70-74	0.75 (0.48-1.01)	.35	0.57 (0.37-0.78)	.02[a]
75-84	0.96 (0.80-1.11)	.02[a]	1.46 (1.23-1.69)	<.001[a]
≥85	1.38 (1.23-1.54)	<.001[a]	5.35 (4.75-5.95)	<.001[a]
Population density				
Low	1.04 (0.81-1.27)	[Reference]	1.43 (1.12-1.74)	[Reference]
Medium low	0.97 (0.76-1.17)	.64	1.31 (1.04-1.58)	.56
Medium high	1.03 (0.84-1.22)	.95	1.39 (1.14-1.65)	.86
High	1.13 (0.97-1.30)	.52	1.54 (1.31-1.77)	.57
Whites				
Sex				
Male	0.83 (0.67-0.99)	[Reference]	1.19 (0.97-1.42)	[Reference]
Female	1.16 (1.02-1.30)	.002[a]	1.51 (1.33-1.70)	.03[a]
Medicaid eligibility				
Noneligible	0.88 (0.77-1.00)	[Reference]	1.07 (0.93-1.21)	[Reference]
Eligible	1.58 (1.34-1.83)	<.001[a]	4.49 (3.81-5.17)	<.001[a]
Nonwhites				
Sex				
Male	1.03 (0.65-1.42)	[Reference]	1.52 (0.96-2.08)	[Reference]
Female	1.47 (1.12-1.82)	.01	1.80 (1.37-2.22)	.44
Medicaid eligibility				
Noneligible	1.26 (0.91-1.62)	[Reference]	1.40 (1.01-1.79)	[Reference]
Eligible	1.28 (0.90-1.66)	.94	2.21 (1.56-2.85)	.04[a]
Medicaid eligibility, males				
Noneligible	0.77 (0.61-0.93)	[Reference]	1.03 (0.82-1.24)	[Reference]
Eligible	1.32 (0.96-1.69)	.006	3.37 (2.45-4.28)	<.001[a]
Medicaid eligibility, females				
Noneligible	1.06 (0.90-1.21)	[Reference]	1.17 (1.00-1.33)	[Reference]
Eligible	1.57 (1.32-1.82)	<.001[a]	3.69 (3.12-4.26)	<.001[a]
Overall	1.05 (0.95-1.15)		1.42 (1.29-1.56)	



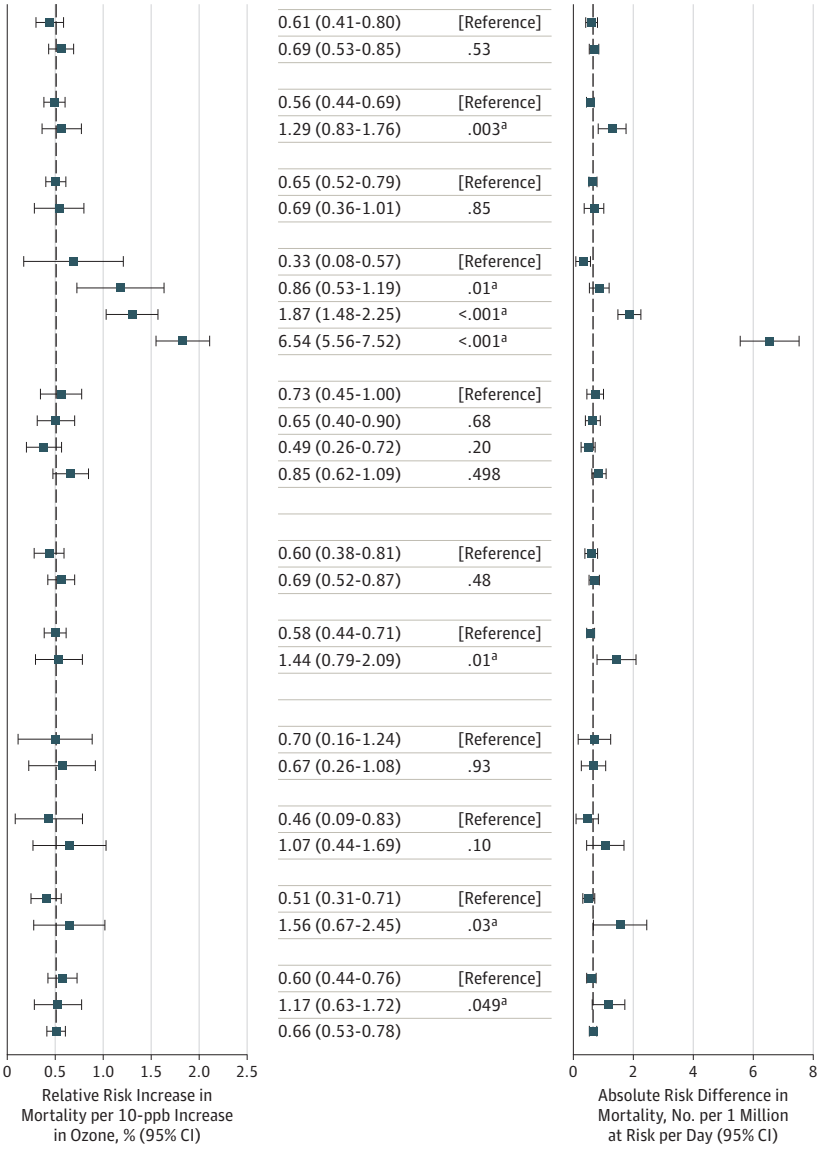
For the main analysis, subgroup analyses used a 2-pollutant analysis (with both $PM_{2.5}$ and ozone), based on the mean of daily exposure on the same day of death and 1 day prior (lag 01-day) as the exposure metric for $PM_{2.5}$, and controlled for natural splines of air and dew point temperatures (each with 3 *df*). Vertical lines indicate effects for the entire study population. Subgroup analyses were conducted for each subgroup (eg, male or female, white or nonwhite, Medicare eligible or Medicare ineligible, age groups, and quartiles of population density). For the main analysis and each subgroup, conditional logistic regressions were run to obtain relative risk increases and calculated absolute risk difference based on baseline mortality rates (eAppendix 2 in the Supplement). Numbers in the figure represent point estimates, 95% CIs, and *P* values for effect modifications. The reference groups were used when assessing effect modification.

[a] Statistically significant effect estimate (at 5% level) compared with the reference group.

The Clean Air Act[1] requires the administrator of the US EPA to set NAAQS at levels that provide "protection for at-risk populations, with an adequate margin of safety."[19] In this study, Medicaid-eligible individuals, females, and elderly individuals had higher mortality rate increases associated with $PM_{2.5}$ than other groups. Previous studies have found similar results in some subgroups.[20,21] Poverty, unhealthy lifestyle, poor access to health care, and other factors may make some subgroups more vulnerable to air pollution. The exact mechanism is worth exploring in future studies.

Figure 4. Relative Risk Increase and Absolute Risk Difference of Daily Mortality Associated With 10-Parts-per-Billion (ppb) Increase in Ozone

Model	Relative Risk Increase in Mortality per 10-ppb Increase in Ozone, % (95% CI)	*P* Value for Effect Modification	Absolute Risk Difference in Mortality, No. per 1 Million at Risk per Day (95% CI)	*P* Value for Effect Modification
Sex				
Male	0.44 (0.30-0.59)	[Reference]	0.61 (0.41-0.80)	[Reference]
Female	0.56 (0.43-0.69)	.23	0.69 (0.53-0.85)	.53
Medicaid eligibility				
Noneligible	0.49 (0.38-0.60)	[Reference]	0.56 (0.44-0.69)	[Reference]
Eligible	0.57 (0.36-0.77)	.53	1.29 (0.83-1.76)	.003[a]
Race/ethnicity				
White	0.51 (0.40-0.61)	[Reference]	0.65 (0.52-0.79)	[Reference]
Nonwhite	0.54 (0.28-0.80)	.81	0.69 (0.36-1.01)	.85
Age, y				
≤69	0.69 (0.17-1.21)	[Reference]	0.33 (0.08-0.57)	[Reference]
70-74	1.18 (0.73-1.63)	.16	0.86 (0.53-1.19)	.01[a]
75-84	1.30 (1.03-1.57)	.04[a]	1.87 (1.48-2.25)	<.001[a]
≥85	1.83 (1.55-2.11)	<.001[a]	6.54 (5.56-7.52)	<.001[a]
Population density				
Low	0.56 (0.35-0.78)	[Reference]	0.73 (0.45-1.00)	[Reference]
Medium low	0.51 (0.31-0.70)	.72	0.65 (0.40-0.90)	.68
Medium high	0.38 (0.20-0.57)	.22	0.49 (0.26-0.72)	.20
High	0.66 (0.48-0.85)	.49	0.85 (0.62-1.09)	.498
Whites				
Sex				
Male	0.44 (0.28-0.59)	[Reference]	0.60 (0.38-0.81)	[Reference]
Female	0.56 (0.42-0.70)	.24	0.69 (0.52-0.87)	.48
Medicaid eligibility				
Noneligible	0.50 (0.39-0.61)	[Reference]	0.58 (0.44-0.71)	[Reference]
Eligible	0.54 (0.29-0.78)	.78	1.44 (0.79-2.09)	.01[a]
Nonwhites				
Sex				
Male	0.50 (0.11-0.89)	[Reference]	0.70 (0.16-1.24)	[Reference]
Female	0.57 (0.22-0.92)	.79	0.67 (0.26-1.08)	.93
Medicaid eligibility				
Noneligible	0.43 (0.08-0.78)	[Reference]	0.46 (0.09-0.83)	[Reference]
Eligible	0.65 (0.27-1.03)	.42	1.07 (0.44-1.69)	.10
Medicaid eligibility, males				
Noneligible	0.40 (0.25-0.56)	[Reference]	0.51 (0.31-0.71)	[Reference]
Eligible	0.65 (0.28-1.02)	.24	1.56 (0.67-2.45)	.03[a]
Medicaid eligibility, females				
Noneligible	0.58 (0.42-0.73)	[Reference]	0.60 (0.44-0.76)	[Reference]
Eligible	0.53 (0.28-0.77)	.75	1.17 (0.63-1.72)	.049[a]
Overall	0.51 (0.41-0.61)		0.66 (0.53-0.78)	

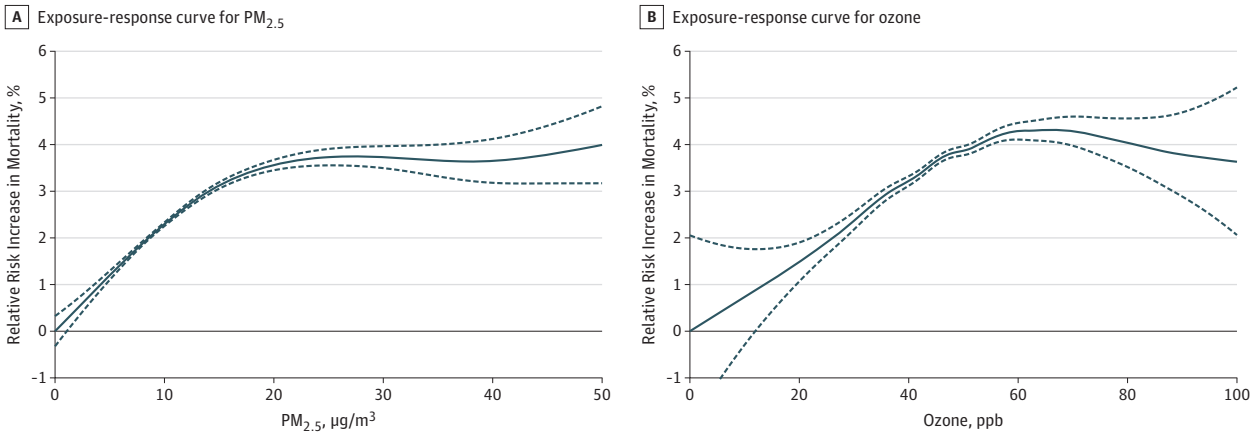


For the main analysis, subgroup analyses used a 2-pollutant analysis (with both $PM_{2.5}$ and ozone), based on the mean of daily exposure on the same day of death and 1 day prior (lag 01-day) as the exposure metric for ozone, and controlled for natural splines of air and dew point temperatures (each with 3 *df*). Vertical lines indicate effects for the entire study population. Subgroup analyses were conducted for each subgroup (eg, male or female, white or nonwhite, Medicare eligible or Medicare ineligible, age groups, and quartiles of population density). For the main analysis and each subgroup, conditional logistic regressions were run to obtain relative risk increases, and calculated absolute risk difference based on baseline mortality rates (eAppendix 2 in the Supplement). For ozone, analyses were restricted to the warm season (April to September). Numbers in the figure represent point estimates, 95% CIs, and *P* values for effect modifications. The reference groups were used when assessing effect modification.

[a] Statistically significant effect estimate (at 5% level) compared with the reference group.

The current NAAQS for daily $PM_{2.5}$ is 35 μg/m^3. When restricting the analysis to daily $PM_{2.5}$ levels below 25 μg/m^3, the association between short-term $PM_{2.5}$ exposure and mortality remained but was elevated. The current daily NAAQS for ozone is 70 ppb; when restricting the analysis to daily warm-season ozone concentrations below 60 ppb, the effect size also increased slightly. The exposure-response curves revealed a similar pattern. These results indicate

Figure 5. Estimated Exposure-Response Curves for Short-term Exposures to Fine Particulate Matter ($PM_{2.5}$) and Ozone



A 2-pollutant analysis with separate penalized splines on $PM_{2.5}$ (A) and ozone (B) was conducted to assess the percentage increase in daily mortality at various pollution levels. Dashed lines indicate 95% CIs. The mean of daily exposure on the same day of death and 1 day prior (lag 01-day) was used as metrics of exposure to $PM_{2.5}$ and ozone. Analysis for ozone was restricted to the warm season (April to September). Ppb indicates parts per billion.

that air pollution is associated with an increase in daily mortality rates, even at levels well below the current standards.

The exposure-response relationship between $PM_{2.5}$ exposure and mortality was consistent with findings of previous studies. One study combined exposure-response curves from 22 European cities and reported an almost linear relationship between $PM_{2.5}$ and mortality.[22] Another multicity study reported a linear relationship down to 2-µg/m^3 $PM_{2.5}$.[23] The present study found a similarly linear exposure-response relationship below 15-µg/m^3 $PM_{2.5}$ and a less steep slope above this level.

For ozone, the linear exposure-response curve with no threshold described in this study is consistent with earlier research. An almost linear exposure-response curve for ozone was previously reported with no threshold or a threshold at very low concentrations.[24] A study from the Netherlands also concluded that if an ozone threshold exists, it does so at very low levels.[25]

Findings from this study are also consistent with the literature regarding the observed effect sizes of both $PM_{2.5}$[5,8,16,26-28] and ozone.[7,20,29,30] This study further demonstrates that in more recent years, during which air pollution concentrations have fallen, statistically significant associations between mortality and exposures to $PM_{2.5}$ and ozone persisted.

The association of mortality and $PM_{2.5}$ exposure is supported by a large number of published experimental studies in animals[31-33] and in humans exposed to traffic air pollution,[34,35] diesel particles,[36] and unfiltered urban air.[37] Similarly, a review of toxicological studies and a recent panel study found that ozone exposure was associated with multiple adverse health outcomes.[38,39]

Strengths

This study has several strengths. First, to our knowledge, this is the largest analysis of daily air pollution exposure and mortality to date, with approximately 4 times the number of deaths included in a previous large study.[5] Second, this study assessed daily exposures using air pollution prediction models that provide accurate estimates of daily levels of $PM_{2.5}$ and ozone for most of the United States, including previously unmonitored areas. An analysis that relied only on exposure data from monitoring stations was found to result in a downward bias in estimates (Table 2). Third, the inclusion of more than 22 million deaths from 2000 to 2012 from the entire Medicare population provided large statistical power to detect differences in mortality rates in potentially vulnerable populations and to estimate mortality rates at very low $PM_{2.5}$ and ozone concentrations. Fourth, this study estimated the air pollution-mortality association well below the current daily NAAQS and in unmonitored areas, and it did not identify significant differences in the mortality rate increase between urban and rural areas. Fifth, this study used a case-crossover design that individually matched potential confounding factors by month, year, and other time-invariant variables and controlled for time-varying patterns, as demonstrated by the minimal differences in meteorological variables between case and control days.

Limitations

This study also has several limitations. First, the case-crossover design does not allow estimation of mortality rate increase associated with long-term exposure to air pollution. Long-term risks in the same study population have been estimated elsewhere.[40] Second, because this study used residential zip code to ascertain exposure level rather than exact home address or place of death, some measurement error is expected. Third, the Medicare population primarily consists of individuals older than 65 years, which limits the generalizability of findings to younger populations. However, because more than two-thirds of deaths in

the United States occur in people older than 65 years of age, and air pollution–related health risk rises with age, the Medicare population in this study includes most cases of air pollution–induced mortality. Fourth, Medicare files do not report cause-specific mortality. Fifth, the most recent data used in this study are nearly 5 years old, and it is uncertain whether exposures and outcomes would be the same with more current data.

Conclusions

In the US Medicare population from 2000 to 2012, short-term exposures to $PM_{2.5}$ and warm-season ozone were significantly associated with increased risk of mortality. This risk occurred at levels below current national air quality standards, suggesting that these standards may need to be reevaluated.

ARTICLE INFORMATION

Accepted for Publication: November 20, 2017.

Author Contributions: Mr Di had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Mr Di and Dr Dai contributed equally to this study.
Concept and design: Di, Dai, Zanobetti, Schwartz, Dominici.
Acquisition, analysis, or interpretation of data: All authors.
Drafting of the manuscript: Di, Dai, Choirat, Dominici.
Critical revision of the manuscript for important intellectual content: All authors.
Statistical analysis: Di, Dai, Choirat, Schwartz, Dominici.
Obtained funding: Zanobetti, Schwartz, Dominici.
Administrative, technical, or material support: Wang, Choirat.
Supervision: Zanobetti, Schwartz, Dominici.

Conflict of Interest Disclosures: All authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Mr Di reported receiving grants from the National Institutes of Health (NIH), Environmental Protection Agency (EPA), Health Effects Institute (HEI), and the National Cancer Institute. Dr Zanobetti reported receiving grants from the NIH, HEI, and EPA. Dr Choirat reported receiving grants from the NIH and EPA. Dr Schwartz reported receiving funding from the US Department of Justice, NIH, EPA, and HEI. Dr Schwartz is an expert consultant of the US Department of Justice regarding health impacts of Clean Air Act violations. No other disclosures were reported.

Funding/Support: This study was supported by grants R01 ES024332-01A1, ES-000002, ES024012, R01ES026217, and 4953-RFA14-3/16-4 from the NIH; grant 4953-RFA14-3/16-4 from the HEI; and grants 83587201-0 and RD-83479801 from the EPA.

Role of the Funder/Sponsor: The funders had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Disclaimer: The contents are solely the responsibility of the grantee and do not necessarily represent the official views of the funding agencies. Further, the funding agencies do not endorse the purchase of any commercial products or services related to this publication.

Additional Contributions: We thank Stacey C. Tobin, PhD, and Kathy L. Brenner, MAT, from Harvard T.H. Chan School of Public Health, for editorial assistance on the manuscript; Sarah L. Duncan, MDiv, and William J. Horka, BS, at the Institute for Quantitative Social Science, Harvard University, for their support with the Research Computing Environment; and Ista Zahn, MS, at the Institute for Quantitative Social Science, Harvard University, for programming support. Dr Tobin received compensation for editorial assistance.

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ENVIRONMENTAL HEALTH

RESEARCH Open Access

Time series analysis of fine particulate matter and asthma reliever dispensations in populations affected by forest fires

Catherine T Elliott[1,2*], Sarah B Henderson[1,2] and Victoria Wan[1]

Abstract

Background: Several studies have evaluated the association between forest fire smoke and acute exacerbations of respiratory diseases, but few have examined effects on pharmaceutical dispensations. We examine the associations between daily fine particulate matter ($PM_{2.5}$) and pharmaceutical dispensations for salbutamol in forest fire-affected and non-fire-affected populations in British Columbia (BC), Canada.

Methods: We estimated $PM_{2.5}$ exposure for populations in administrative health areas using measurements from central monitors. Remote sensing data on fires were used to classify the populations as fire-affected or non-fire-affected, and to identify extreme fire days. Daily counts of salbutamol dispensations between 2003 and 2010 were extracted from the BC PharmaNet database. We estimated rate ratios (RR) and 95% confidence intervals (CIs) for each population during all fire seasons and on extreme fire days, adjusted for temperature, humidity, and temporal trends. Overall effects for fire-affected and non-fire-affected populations were estimated via meta-regression.

Results: Fire season $PM_{2.5}$ was positively associated with salbutamol dispensations in all fire-affected populations, with a meta-regression RR (95% CI) of 1.06 (1.04-1.07) for a 10 ug/m^3 increase. Fire season $PM_{2.5}$ was not significantly associated with salbutamol dispensations in non-fire-affected populations, with a meta-regression RR of 1.00 (0.98-1.01). On extreme fire days $PM_{2.5}$ was positively associated with salbutamol dispensations in both population types, with a global meta-regression RR of 1.07 (1.04 - 1.09).

Conclusions: Salbutamol dispensations were clearly associated with fire-related $PM_{2.5}$. Significant associations were observed in smaller populations (range: 8,000 to 170,000 persons, median: 26,000) than those reported previously, suggesting that salbutamol dispensations may be a valuable outcome for public health surveillance during fire events.

Keywords: Fires, Smoke, Air pollution, Asthma, Pulmonary disease chronic obstructive, Epidemiology

Background

The public health effects of acute environmental exposures are often described as a pyramid, with the rarest outcomes at the peak and the more common outcomes at the base. The rarest outcomes are most severe, while the most common outcomes are the mildest. Many population-based studies focus on the upper part of the pyramid because severe outcomes are typically recorded in administrative databases. However, their rarity makes it challenging to evaluate short-lived exposures with adequate statistical power, even in large populations. In the case of forest fire smoke, only two [1,2] of five [3-5] studies have reported significant associations between smoke-related particulate matter (PM) and all-cause mortality, and effects specific to respiratory mortality were not clear. Similarly, time-series studies have reported significant associations between smoke-related PM and respiratory hospital admissions in multiple settings [6-10], but most have been conducted in large towns or cities,

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and not in the remote and rural areas most affected by fire smoke.

The effects of forest fire smoke on milder health outcomes have generally been examined in smaller panel studies [11-14] due to the absence of population-based information. However, administrative databases that capture common health outcomes could serve to advance forest fire smoke epidemiology by allowing us to study smaller populations and to detect smaller effect estimates with the increased statistical power. Electronic registries of pharmaceutical dispensations provide such data, and have previously been used to evaluate the public health impacts of other short-lived events, such as human [15] and natural [16] disasters. Short-acting beta-agonist (SABA) dispensations are specifically associated with acute exacerbations of obstructive lung diseases (such as asthma and chronic obstructive pulmonary disease), and they outnumber severe outcomes [17]. As such, SABA dispensations may be a more sensitive indicator of obstructive lung disease exacerbations within the population [18].

The Canadian province of British Columbia (BC) is regularly impacted by forest fires. It has both a comprehensive pharmaceutical database and a long-standing air quality monitoring network that covers many smaller communities. This setting provides a valuable opportunity to study the public health effects of forest fire smoke in smaller populations using a mild health outcome. Here we examine the associations between daily $PM_{2.5}$ (PM less than 2.5 microns in diameter) and dispensations of medications used to relieve exacerbations of chronic respiratory diseases in fire-affected versus non-fire-affected populations between 2003 and 2010.

Methods

Study area

The study was conducted in the province of British Columbia (BC), on the west coast of Canada. Forest fires burn an average of 980 km^2 per year in BC [19], and widespread infestation by the mountain pine beetle has left forests more susceptible to extreme events in recent years [20]. The province is geographically divided into 89 local health areas (LHAs), ranging in size from 40 – 130,000 km^2 (Figure 1), and in 2006 population from 542 – 352,783 people [21]. Geographically smaller LHAs typically have larger populations living in urban and suburban areas, while larger LHAs have smaller populations living in rural and remote communities.

Exposure assessment

The air quality monitoring network in BC is maintained by the BC Ministry of Environment. Ambient concentrations of particulate matter (PM) are continuously measured at several stations by $PM_{2.5}$ and/or PM_{10} (PM less than 10 microns in diameter) tapered element oscillating microbalances [22]. These instruments are heated to 30 or 40°C depending on their locations, and loss of volatile materials is expected to be less during the fires season than during the winter months [23]. We started by identifying all stations that had $PM_{2.5}$ and/or PM_{10} measurements for every fire season (April 1 through September 30) in the study period. Any LHA with one or more of these stations was included in the study (Figure 1). For LHAs with multiple stations, the one closest to the LHA population center was used. As such, the daily PM exposure of the population within each study LHA was estimated using data from a single monitoring station within that LHA.

All PM data were converted to $PM_{2.5}$ concentrations. For LHAs where $PM_{2.5}$ measurements were available for the whole period, those data were used. For LHAs with some $PM_{2.5}$ and some PM_{10} measurements, we adjusted PM_{10} to $PM_{2.5}$ using the regression coefficients from linear models applied to all fire seasons when both instruments were running simultaneously. For LHAs with PM_{10} measurements only, or with insufficient overlap between instruments, we used the time-weighted average of linear regression coefficients from the other stations with simultaneous $PM_{2.5}$ and PM_{10} measurements.

Fire-affected LHAs and extreme fire days

The air quality monitoring network measures PM contributions from all sources, including smoke from forest fires. We focused on the effects of fire smoke by using data from the Moderate Resolution Imaging Spectroradiometers (MODIS) to classify LHAs as fire-affected and non-fire-affected, and to identify extreme fire days,. These remote sensing instruments overpass most areas of the Earth four times daily, detecting fires at a resolution of 1 km^2 at nadir [24]. The information recorded for each detected fire includes its central latitude and longitude, and a measure of its intensity, known as the fire radiative power (FRP, in MW). The FRP is proportional to aerosol emissions, and serves as a good indicator of the smoke generated by the detected fire [25-27]. We downloaded these data for BC and its surrounding areas from the Fire Information Resource Management System (FIRMS) [28], and used a geographic information system (GIS) to map all of the fires detected during the study period.

To assess the impact of fire on each LHA we used the GIS to draw a 100 km radius circle around its PM monitoring station, and then calculated the daily sum of FRP from all fires detected within that circle. Next, we aggregated daily FRP sums for all LHAs to examine the percentiles of the overall distribution. Finally, an LHA was defined as fire-affected if the plotted time-series of daily FRP values showed that the overall 95th percentile was exceeded in three or more of the nine fire seasons

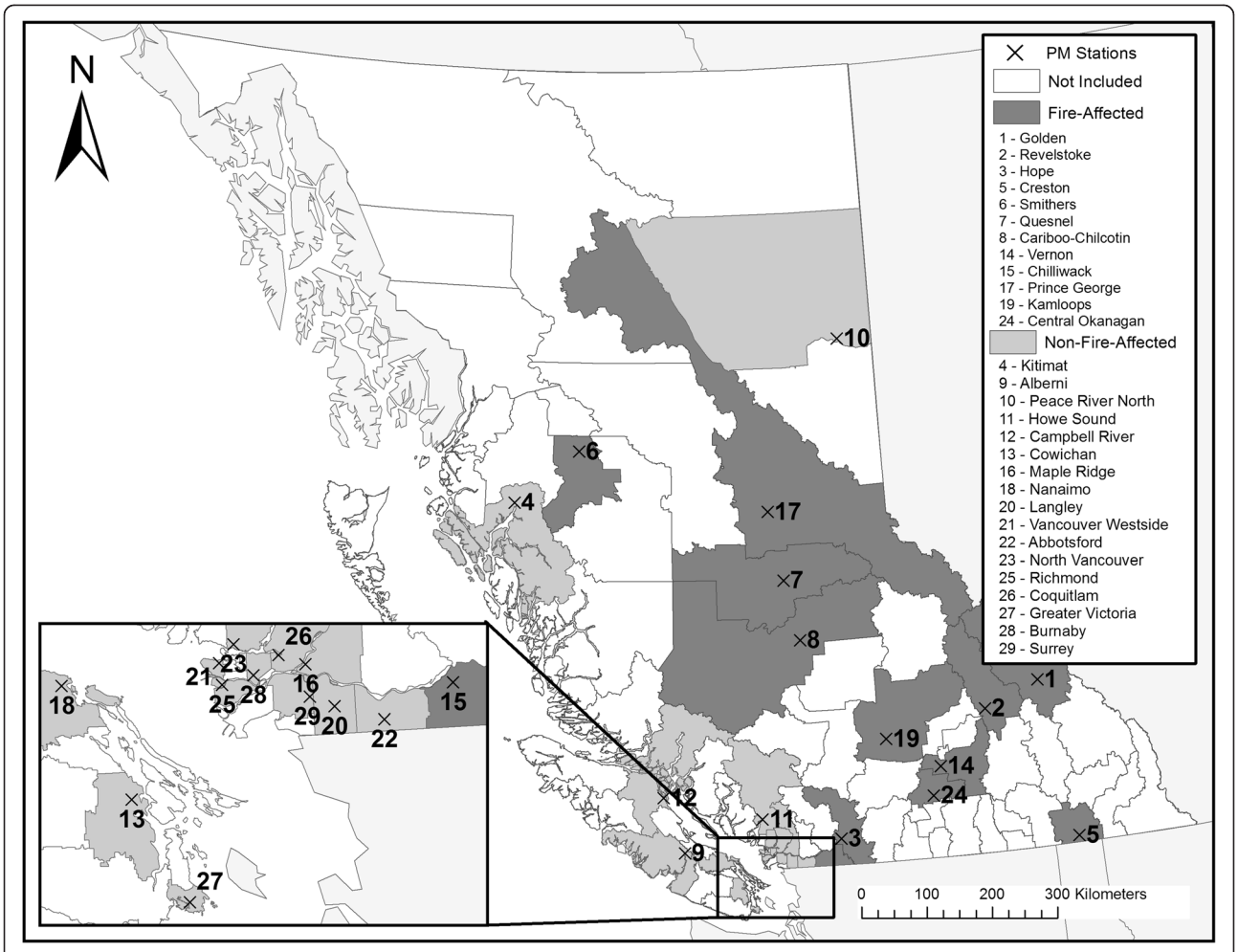

Figure 1 Map of the study region showing the local health areas (LHAs) included in the study, and the locations of the PM air quality monitoring stations. Study LHAs are numbered in order of increasing population, as in Table 1.

(Figure 2). To identify extreme fire days we summed all FRP values detected within and around BC for each day of the study period, and used the 80th, 90th, and 95th percentiles of the distribution to limit analyses to periods most likely affected by heavy smoke.

Pharmaceutical dispensations

Daily counts of pharmaceutical dispensations were received for each LHA from the BC PharmaNet database. Law requires that every prescription dispensed in the province be recorded in PharmaNet, regardless of the recipient or the payer [29]. We decided *a priori* to examine relationships between $PM_{2.5}$ and counts for inhaled salbutamol sulfate, a selective beta-2-adrenoreceptor agonist that is commonly and specifically used to rapidly relieve exacerbations of asthma, COPD, and other obstructive lung diseases. Dispensations included all inhaled preparations of salbutamol available in BC (i.e. aerosol inhalers, powder inhalers and nebulizer solutions). Salbutamol preparations for ingestion were excluded. Other selective beta-2-adrenergic inhalants were also excluded, because preliminary analyses showed that they were rarely prescribed in BC (less than 5% of dispensations).

Time-series models

For every LHA included in the study we estimated the effect of daily $PM_{2.5}$ concentrations on the rate of pharmaceutical dispensations for respiratory reliever medications, during all fire seasons and on extreme fire days. We used generalized linear models with natural cubic splines, adjusted for temperature, relative humidity, and temporal trends as shown in Equation 1.

$$O_t \sim Poisson\left(\mu_t, \sigma^2\right)$$

$$g(\mu_t) = PM_{lag01} + ns(T_t, df = 3) + ns(RH_t, df = 3) + YMDOW_t$$

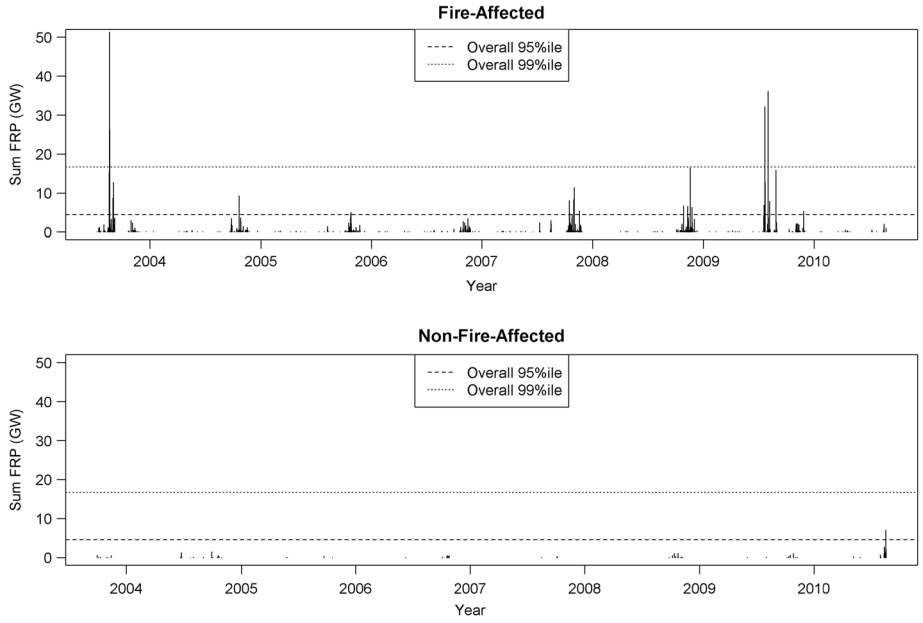

Figure 2 Examples of the time series of summed fire radiative power (FRP, in gigawatts) values for a fire-affected LHA (above, Central Okanagan) and a non-fire-affected LHA (below, Kitimat).

Where: O_t = observed dispensation count in the LHA on day t; PM_{lag01} = $PM_{2.5}$ concentration in the LHA averaged over days t and t-1.; T_t = mean temperature in the region of the LHA on day t, fitted as a natural cubic spline with three degrees of freedom; RH_t = mean relative humidity in the region of the LHA on day t, fitted as a natural cubic spline with three degrees of freedom; $YMDOW_t$ = year, month, and day of week (statutory holidays treated as Sundays) on day t, fitted as a factor variable with 378 levels (9 years * 6 months * 7 days). The resulting estimate of effect was the rate ratio (RR) associated with a 10 ug/m^3 increase in $PM_{2.5}$. A lag of 0-1 days was chosen for principal analyses based on model fit statistics from preliminary analyses, but lags of 0 through 7 days were tested. All analyses were completed in the R statistical computing environment [30]. After fitting individual models for each LHA, we conducted a random effects meta-regression using the inverse variance method [31] to estimate an overall effect for the fire-affected LHAs and the non-fire-affected LHAs during the fire season and on extreme fire days.

Results

Included LHAs

A total of 29 (out of 89) LHAs were included in the study (Table 1 and Figure 1). Their land areas ranged from 48 to 76,215 km^2 and their 2006 populations ranged from 7,024 to 352,783 people. The average daily salbutamol dispensations ranged from 4.3 to 103.4 (Table 1). Most of the LHAs had $PM_{2.5}$ measurements covering the majority of the study period, and PM_{10} concentrations were converted to $PM_{2.5}$ with coefficients ranging from 0.27 to 0.69, with an average of 0.49. Mean fire season $PM_{2.5}$ concentrations ranged from 2.8 to 11.8 ug/m^3 across all stations. Maximum concentrations ranged from 33.4 to 248.1 ug/m^3 for the 12 LHAs classified as fire-affected, and from 15.2 to 49.3 ug/m^3 for the 17 LHAs that were not-fire-affected (Table 1). The mean $PM_{2.5}$ concentrations on fire days in the 80th, 90th, and 95th percentiles of provincial FRP were 8.2, 9.6, and 11.2 ug/m^3, respectively. In the 95th percentile there were 28 extreme fire days in 2003, 22 in 2009, 21 in 2010, 18 in 2004, 7 in 2006, 2 in 2007, and none in 2005 or 2008.

Associations between $PM_{2.5}$ and salbutamol dispensations

Fire season $PM_{2.5}$ was positively associated with salbutamol dispensations in all fire-affected LHAs, with statistically significant results in 8 of 12 cases (Figure 3). The meta-regression RR (95% confidence interval) for a 10 ug/m^3 increase in $PM_{2.5}$ was 1.06 (1.04 – 1.07). The effect was evident at lags up to four days, decreasing to null by the fifth day. Fire season $PM_{2.5}$ was not significantly associated with salbutamol dispensations in 15 of 17 non-fire-affected LHAs (Figure 4), with a meta-regression RR (95%CI) of 1.00 (0.98 – 1.01). The two exceptions were a protective effect observed in Greater Victoria, and a positive association of 1.23 (1.00 – 1.49) in Kitimat, where an aluminum smelter is an important source of PM and other air emissions. When analyses were restricted to fire days in the 80th, 90th, and 95th percentile of the provincial sum of FRP, the meta-regression RRs remained stable for the fire-affected LHAs (Figure 3),

Table 1 Summary information for local health areas (LHAs) included in the analyses, listed in order of 2006 population

LHA name	2006 [a] population	Daily average salbutamol dispensations (fire season)	Area (km^2)	Fire season $PM_{2.5}$	Fire season PM_{10}	$PM_{2.5}/PM_{10}$ coefficient	Fire season Mean $PM_{2.5}$ ($\mu g/m^3$)	Fire season Max $PM_{2.5}$ ($\mu g/m^3$)	Fire-affected?
Golden	7,024	4.5	13,350	2003-2010	2003-2010	0.51	5.7	74.0	Y
Revelstoke	7,897	4.3	9,307	2007-2010	2003-2007	0.49[b]	7.1	81.7	Y
Hope	8,062	4.9	5,280	2004-2010	2003-2010	0.48	5.0	33.4	Y
Kitimat	10,443	5.1	19,639	2003-2010	2003-2010	0.44	2.8	24.8	
Creston	11,917	6.8	3,789	2010	2003-2009	0.49 [b]	7.3	48.9	Y
Smithers	16,073	7.1	9,827	2005-2010	2003-2010	0.27	4.2	66.8	Y
Quesnel	22,930	11.7	23,732	2003-2010	2003-2010	0.69	7.4	139.4	Y
Cariboo-Chilcotin	26,150	12.9	44,695	2003-2010	2003-2010	0.59	6.1	248.1	Y
Alberni	31,077	14.0	6,809	-	2003-2010	0.49 [b]	4.8	15.2	
Peace River North	32,642	14.4	68,765	-	2003-2010	0.49 [b]	11.8	49.3	
Howe Sound	32,327	12.6	9,236	-	2003-2010	0.49 [b]	8.3	33.7	
Campbell River	40,173	18.0	13,624	2006-2010	2003-2009	0.50	3.9	41.7	
Cowichan	54,855	22.9	735	2010	2003-2009	0.49 [b]	4.8	37.9	
Vernon	62,227	29.9	5,555	2003-2010	2003-2008	0.38	5.4	130.8	Y
Chilliwack	79,302	36.2	1,314	2003-2010	2003-2010	0.40	5.3	35.7	Y
Maple Ridge	88,020	32.3	1,450	2003-2010	2003-2010	0.45	5.5	36.5	
Prince George	94,852	44.3	76,215	2003-2010	2003-2010	0.53	7.4	176.4	Y
Nanaimo	98,561	46.5	1,289	2003-2010	-	-	3.7	48.7	
Kamloops	105,491	44.8	16,319	2003-2010	2003-2008	0.49	5.5	140.1	Y
Langley	122,219	46.7	323	2003-2010	2003-2010	0.51	5.5	34.0	
Vancouver Westside	129,011	24.6	48	2004-2010	2003-2008	0.52	5.5	32.1	
Abbotsford	130,008	51.1	413	2010	2003-2010	0.49 [b]	6.9	23.9	
North Vancouver	134,453	33.3	398	-	2003-2010	0.49 [b]	6.4	27.2	
Central Okanagan	167,323	59.1	2,942	2003-2010	2003-2008	0.56	5.5	185.6	Y
Richmond	182,652	37.5	124	2003-2010	2003-2010	0.51	5.0	40.3	
Coquitlam	205,495	52.6	733	2004-2010	2003-2008	0.50	6.2	42.0	
Burnaby	210,507	56.4	90	2003-2010	2003-2010	0.45	5.2	42.7	
Greater Victoria	217,374	65.4	113	2003-2010	-	-	5.0	17.3	
Surrey	352,783	103.4	333	-	2003-2010	0.49 [b]	7.1	19.1	

The forest fire season is defined as April 1 – September 30 each year.
[a] 2006 was a national census year, and was also the midpoint of the study period.
[b] Average from all other stations used.

but increased with each restriction for the non-fire-affected LHAs (Figure 4). The point estimate was the same for both population types at the 95th percentile, with wider confidence intervals for the non-fire-affected group. The province-wide meta-regression estimate was 1.07 (1.04 – 1.09) for the most extreme fire days.

Discussion

We found consistent associations between fire-related $PM_{2.5}$ and salbutamol dispensations. During the fire season a 10 ug/m^3 increase in $PM_{2.5}$ was associated with a 6% increase in salbutamol dispensations (RR = 1.06, 95% CI 1.04-1.07) in fire-affected populations, but no effect was observed in non-fire-affected populations. On extreme fire days the same $PM_{2.5}$ increase was associated with a 7% increase in salbutamol dispensations in both types of populations (global RR = 1.07, 95% CI 1.04-1.09). To the best of our knowledge there is only one other study of pharmaceutical dispensations and forest fires, which evaluated the aftermath of an extreme fire season in Galacia, Spain. Caamano-Isorna et al. [16] reported that male and female pensioners (age not specified) increased consumption of medications to relieve obstructive lung disease by 10.3% and 12.1%, respectively, in the months

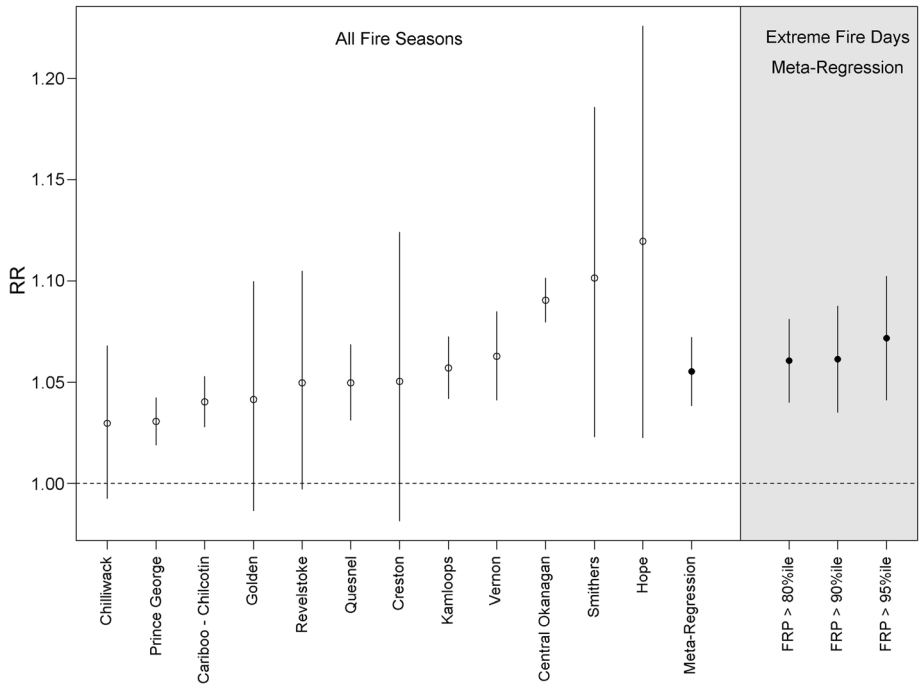

Figure 3 Regression results for the association between a 10 ug/m^3 increase in $PM_{2.5}$ (day-of and day-before average, lag_{01}) and dispensation counts for the respiratory relief medication salbutamol sulfate in fire-affected local health areas (LHAs). Results for individual LHAs are ordered by the rate ratio (RR) point estimates for all fire seasons, followed by the meta-regression estimates for all fire seasons, and extreme fire days in the 80th, 90th, and 95th percentiles.

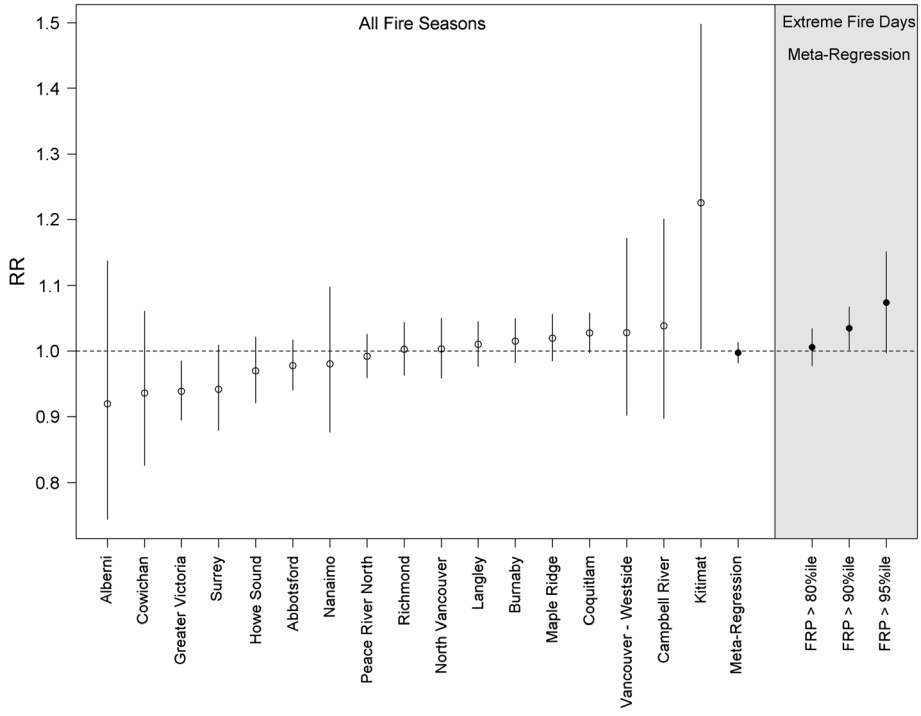

Figure 4 Regression results for the association between a 10 ug/m^3 increase in $PM_{2.5}$ (day-of and day-before average, lag_{01}) and dispensation counts for the respiratory relief medication salbutamol sulfate in non-fire-affected local health areas (LHAs). Results for individual LHAs are ordered by the rate ratio (RR) point estimates for all fire seasons, followed by the meta-regression estimates for all fire seasons, and extreme fire days in the 80th, 90th, and 95th percentiles.

following the severe fires, when compared with the previous months. No change was reported for non-pensioners.

Our results are consistent with other time-series studies on moderate to severe respiratory outcomes associated with exposure to forest fire smoke. Henderson et al. [9] reported that a 10 ug/m^3 increase in PM_{10} was associated with a 6% increase in the odds of an asthma-specific physician visit (OR = 1.06, 95% CI 1.03-1.08) during the 2003 fire season in BC. In the state of Victoria, Australia, Tham et al. (2009) [32] reported that a 10 ug/m^3 increase in PM_{10} was associated with a 2% increase in the relative rate (RR) of all respiratory ED visits (RR = 1.02, 95% CI 1.00-1.03). In Los Angeles, California, Delfino et al. [7] reported that a 10 ug/m^3 increase in $PM_{2.5}$ was associated with a 3% increase in the rate of all respiratory hospital admissions (RR = 1.03, 95% CI 1.01-1.04), a 5% increase in asthma admissions (RR = 1.05, 95% CI 1.02-1.08), and a 4% increase in chronic obstructive pulmonary disease admissions (RR = 1.04, 95% CI 1.00-1.08). Similarly, the Henderson et al. [9] study in BC also reported a 5% increase in the odds of all respiratory hospital admissions (OR = 1.05, 95% CI 1.00-1.10). Finally, in Sydney, Australia, Johnston et al. [33] reported that extreme smoke events were associated with a 9% increase in the odds of respiratory mortality, though the estimate was not statistically significant (OR = 1.09, 95% CI 0.88-1.36).

Because ambient $PM_{2.5}$ monitors cannot differentiate between PM sources, we used empirical remote sensing data to objectively classify populations as fire-affected or non-fire-affected, and to identify extreme fire days. While several other studies have used satellite-based methods to identify smoke-affected periods and areas [1,7,9,34], our work is the first to leverage the fire radiative power measurements (proportional to smoke emissions) to classify populations and periods in this way. One might expect $PM_{2.5}$ to have a clearer effect on salbutamol dispensations in LHAs where the smoke-related PM was extremely high, but this relationship was evident even in fire-affected LHAs with PM distributions that overlapped those of the non-fire-affected LHAs (Table 1). For example, the fire-affected LHA of Hope (population = 8,000; peak $PM_{2.5}$ = 33.4 ug/m^3; mean $PM_{2.5}$ = 5.0 ug/m^3) had a significant association (RR = 1.12, 95% CI 1.02-1.22), whereas the non-fire-affected LHA of Howe Sound (population = 32,000; peak $PM_{2.5}$ = 33.7 ug/m3, mean $PM_{2.5}$ = 8.2) did not (RR = 0.97, 95% CI 0.92-1.02). One outlier among the non-fire-affected LHAs was Kitimat (RR = 1.23, 95% CI 1.00-1.50), which is the site of an aluminum smelter.[a] The population may therefore be exposed to $PM_{2.5}$ that has a different toxicological profile than that in the other LHAs.

Fire smoke often affects small populations because forest fires most commonly burn in rural and remote areas; extreme events that affect large cities are relatively rare. However, it has been challenging to find associations between more severe respiratory outcomes and smoke exposure in smaller populations. During the 2003 fire season in BC, Moore et al. [35] detected an increase in weekly respiratory physician visits in one larger community (approximate population 185,000) with heavy smoke, but not in a smaller nearby community (approximate population 110,000) with more moderate smoke. In Darwin, Australia (approximate population 110,000) Johnston et al. [10,33] conducted two studies of respiratory

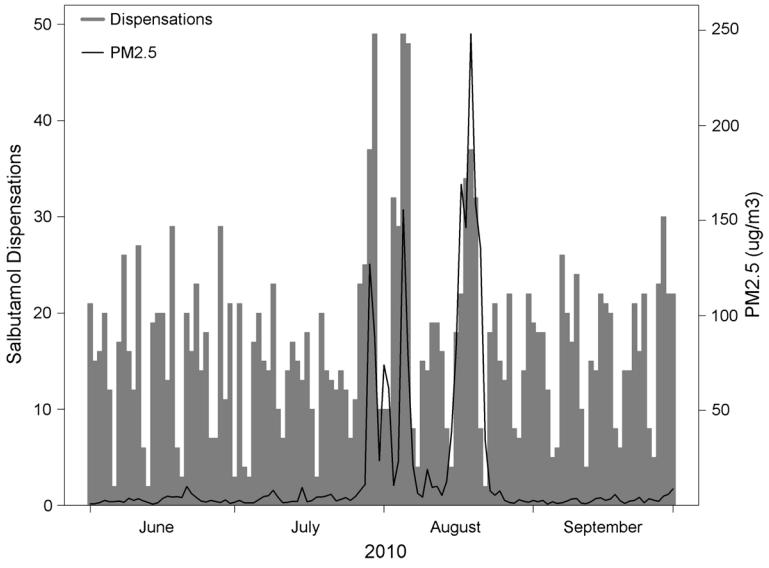

Figure 5 Daily time-series of salbutamol dispensations compared with $PM_{2.5}$ concentrations in the Cariboo-Chilcotin LHA during the summer of 2010. Days with low counts are weekends and holidays, when many pharmacies are closed.

outcomes associated with ambient PM_{10} during the forest fire season. The first was an ecological study that detected significant increases in daily asthma ED visits only when concentrations were over 40 ug/m^3 [33]. The second was a case-crossover study on three years of hospital admissions [10]. Although the maximum daily concentration was 70 ug/m^3, the positive associations were not statistically significant. In contrast, we have found strong and significant effects of $PM_{2.5}$ on salbutamol dispensations in fire-affected populations ranging in size from 8,000 to 170,000 persons. Given that dispensations occur more frequently than severe outcomes, we suggest that they are more useful for studying the health effects of forest fire smoke in small populations. Furthermore, we observed that salbutamol dispensations rose rapidly in response to heavy smoke and fell rapidly as the smoke cleared (Figure 5), suggesting that dispensations may also be a responsive outcome for public health surveillance during smoke events.

There are important limitations to our analyses. First, this was an ecological study design, so we were unable to explore effect modification by individual factors. Second, a pharmaceutical dispensation does not necessarily reflect a disease exacerbation. Individuals with chronic lung disease may have sufficient reliever medication on hand, and not require a new dispensation for each exacerbation. Those who fill a prescription may do so for reasons related to the fire smoke (e.g., anticipated smoke effects), or for reasons that are completely unrelated (e.g., routine prescription renewal). Finally, we used data from single air quality monitoring stations to represent the exposure of populations within entire LHAs, some of which cover large geographic areas. Although most people in each LHA live in the monitored community, this homogenous approach to exposure assessment cannot account for the spatial variability inherent to fire smoke exposure.

Effective public health response to forest fire smoke events requires an understanding of its short-term health effects in order to identify who is most at risk, and to implement strategies to protect them. During milder events, the public health response may be limited to public education, but it should be rapidly escalated to provision of air shelters and/or evacuation as health risks increase. We have shown that pharmaceutical dispensations can be used to assess the population health effects in small communities. Given that these data are available in near-real-time, routine surveillance of pharmaceutical dispensations could play an important role in public health situational awareness and response. Further analyses are required to characterize short-term trends, and to create the indicators necessary to support fire smoke response guidelines.

Conclusions

We report a clear association between fire-related $PM_{2.5}$ and salbutamol dispensations in BC. The changes in salbutamol dispensations were observed in smaller populations than previously reported for any respiratory outcome (range: 8,000 to 170,000 persons, median: 26,000). This suggests that pharmaceutical dispensations can be leveraged in further research on acute respiratory events among small populations. Furthermore, this outcome was responsive to smoke-related $PM_{2.5}$ concentrations, and may therefore be particularly useful for public health surveillance during forest fire smoke events.

Endnotes

[a] Note that the FRP time-series for Kitimat is the bottom panel of Figure 2, clearly showing little fire activity in the area.

Abbreviations

PM_{10}: PM less than 10 microns in diameter; $PM_{2.5}$: PM less than 2.5 microns in diameter; RR: Rate ratio; CI: Confidence intervals; SABA: Short acting beta agonist; ED: Emergency department; BC: British Columbia; km^2: Square kilometers; LHAs: Local health authority; FPR: Fire radiative power; MW: Megawatts; GIS: Geographic information system; FIRMS: Information Resource Management System; df: Degrees of freedom; O_t: Observed dispensation count in the LHA on day t; PM_{lag01}: $PM_{2.5}$ concentration in the LHA averaged over days t (same day) and t-1 (previous day); T_t: Mean temperature in the region of the LHA on day t; RH_t: Mean relative humidity in the region of the LHA on day t; $YMDOW_t$: Year, month, and day of week (statutory holidays treated as Sundays) on day t; OR: Odds ratio.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

CTE conceived of and helped design the study, critically reviewed the analysis and drafted the manuscript. SBH helped design the study, led the analysis, and helped draft the manuscript. VW assisted with data analysis and critically reviewed the manuscript. All authors read and approved the final manuscript.

Received: 21 August 2012 Accepted: 10 January 2013
Published: 28 January 2013

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doi:10.1186/1476-069X-12-11
Cite this article as: Elliott *et al.*: Time series analysis of fine particulate matter and asthma reliever dispensations in populations affected by forest fires. *Environmental Health* 2013 **12**:11.

ORIGINAL ARTICLE

Short-term effects of particulate matter on mortality during forest fires in Southern Europe: results of the MED-PARTICLES Project

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▶ Additional material is published online only. To view please visit the journal online (http://dx.doi.org/10.1136/oemed-2014-102459).

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Received 17 July 2014
Revised 16 January 2015
Accepted 27 January 2015
Published Online First 17 February 2015

ABSTRACT

Background An association between occurrence of wildfires and mortality in the exposed population has been observed in several studies with controversial results for cause-specific mortality. In the Mediterranean area, forest fires usually occur during spring–summer, they overlap with Saharan outbreaks, are associated with increased temperature and their health effects are probably due to an increase in particulate matter.

Aim and methods We analysed the effects of wildfires and particulate matter (PM_{10}) on mortality in 10 southern European cities in Spain, France, Italy and Greece (2003–2010), using satellite data for exposure assessment and Poisson regression models, simulating a case-crossover approach.

Results We found that smoky days were associated with increased cardiovascular mortality (lag 0–5, 6.29%, 95% CIs 1.00 to 11.85). When the effect of PM_{10} (per 10 $\mu g/m^3$) was evaluated, there was an increase in natural mortality (0.49%), cardiovascular mortality (0.65%) and respiratory mortality (2.13%) on smoke-free days, but PM_{10}-related mortality was higher on smoky days (natural mortality up to 1.10% and respiratory mortality up to 3.90%) with a suggestion of effect modification for cardiovascular mortality (3.42%, p value for effect modification 0.055), controlling for Saharan dust advections.

Conclusions Smoke is associated with increased cardiovascular mortality in urban residents, and PM_{10} on smoky days has a larger effect on cardiovascular and respiratory mortality than on other days.

What is already known

- Increase in natural mortality occur on forest fire days.
- In Europe, forest fires usually occur during the hot season, are associated with increased temperature and dust outbreaks and their health effects are probably due to an increase in particulate matter (PM).

What this paper adds

- Mortality for cardiovascular causes increases in cities during smoky days.
- PM_{10}-related cardiovascular mortality is modified during smoky days.
- PM_{10}-related respiratory mortality increases on smoky days.

INTRODUCTION

Forest fires contribute to the earth's planetary concentrations of organic carbon (OC) and elemental carbon (EC).[1] In Mediterranean countries, carbonaceous compound emissions from wildfires are made up of 71% carbon dioxide (CO_2), 26% carbon monoxide (CO) and 0.3% total particulate carbon.[2] Secondary aerosols may contribute greatly to increases in carbonaceous particulate matter (PM), since the large amounts of volatile organic compounds (VOCs) released during forest fires[3] may be converted into carbonaceous PM by anthropogenic agents, such as NO_x and O_3.[4] In addition, a number of polycyclic aromatic hydrocarbons arise from imperfect combustion of biomass.[5]

Exposure to emissions from forest fires is sporadic and short lasting; it entails high levels of combustion-related pollutants and is usually associated with high ambient temperature.[6 7] In the Mediterranean area, wildfires occur mainly during warm seasons, in high ambient temperatures, and are often concurrent with Saharan dust outbreaks.[8] Climatic conditions, including precipitation, winds and boundary layer height, may influence the occurrence of fires and exposure to the resulting air pollutants. All of these issues make it difficult to assess human exposure to forest fire emissions.

The assessment of human exposure to fires also presents operational difficulties since the surveillance of fire events is currently the responsibility of the fire department: they record dates, locations, durations and extent of burnt areas, but not information about proximity and size of the populated areas affected, which could be relevant when assessing exposure. Satellite data and dispersion models provide qualitative information about the spatial extent of wildfires; they also allow a rough estimate of the contribution of the fire to the ambient concentrations of particles, but they do not assess

To cite: Faustini A, Alessandrini ER, Pey J, *et al*. *Occup Environ Med* 2015;**72**:323–329.

concentrations at ground level. On the other hand, fixed monitors located in large cities monitor pollutants from anthropogenic sources, such as road traffic, domestic heating, shipping, industries and power generation. Therefore, routine air quality surveillance may fail to represent the atmospheric pollution resulting from forest fires,[9] while rural monitors are often sparse or unavailable in regions affected by fires.[10]

A few studies have reported increases in commonly monitored ambient pollutants, such as fine particles ($PM_{2.5}$), carbon monoxide (CO), sulfur dioxide (SO_2), ozone (O_3) and black carbon (BC), as possible indirect indicators of exposure to fires in urban areas.[10–12] Levoglucosan is the typical indicator of biomass-burning emissions[13] and is a well-known biomarker of fire exposure.[14] Soluble potassium has also been used as a biomass-burning tracer.[15] Currently, however, experience with these indicators to assess wildfires exposure is very limited.

The health effects of wildfires are probably due to PM (fine and ultrafine), but may also owe to other combustion-related factors such as inorganic gases and VOCs, and even the temperature increases generated by nearby fires.[6 7] Mortality is an important potential outcome of this exposure,[9 16–19] in addition to respiratory symptoms,[20] exacerbations of pre-existing diseases[21–24] and cardiovascular effects.[25 26]

As part of the MED-PARTICLES project funded by the European Union under the LIFE+ framework, we studied the short-term effects of forest fire smoke and PM on the mortality of the population living in large cities in southern Mediterranean Europe. Exposure to fires was defined using satellite observations, and it was confirmed against daily changes in temperature and concentrations of fire-related pollutants.

MATERIALS AND METHODS

The study included the cities that took part in the MED-PARTICLES LIFE+ project, namely Madrid and Barcelona in Spain; Marseille in France; Turin, Milan, Bologna, Parma, Modena, Reggio Emilia, Rome and Palermo in Italy; and Thessaloniki and Athens in Greece. Exposure assessment was performed for 9 years (2003–2011) whereas mortality data were collected in each city, for a variable period of 3–8 years, from 2001 to 2010. Data analyses were carried out for the period 2003–2010.

Exposure assessment

Forest fire events were identified on smoke surface concentration maps supplied by the NAAPS model (Navy Aerosol Analysis and Prediction System—US Naval Research Laboratory Marine Meteorology Division, http://www.nrlmry.navy.mil/aerosol/), which takes into account both the aerosol optical depth (AOD) from satellite measurements and the fire-related smoke plumes. Such aerosol maps are initially generated as forecast products, and are thereafter corrected from satellite AOD measurements. The smoke concentration at surface ranges from 1 to over 64 $\mu g/m^3$; however, the influence of low-magnitude wildfires cannot be assessed though they may greatly affect an urban area when they occur nearby. The use of satellite images helped us to distinguish between smoky days and smoke-free days, especially when NAAPS outputs diverged in consecutive days. The fire-related smoke plumes allowed us to assess the involvement of surrounding cities.

In order to be as conservative as possible, we defined a day as being 'fire smoke-affected, or smoky' when smoke concentrations were higher than 8 $\mu g/m^3$; additionally, fire smoke intensity was classified for each day as low (smoke concentration between 8 and 16 $\mu g/m^3$), medium (smoke concentration between 16 and 32 $\mu g/m^3$) or severe (smoke concentration above 32 $\mu g/m^3$). An additional assessment of smoke episodes was made on the basis of their duration, classifying them as isolated episodes (1-day duration), short episodes (2–4 consecutive days) and long episodes (5 or more days, where 1 day without smoke in a sequence of at least five days did not interrupt the sequence).

Finally, to confirm the fire smoke assessment, smoky days were classified according to the absolute changes of daily mean temperature,[27] PM_{10}, CO and O_3 levels measured at fixed monitors in each city. The absolute changes in these factors during smoke events of different duration and intensity (defined as a multilevel variable with smoke-free days as reference) were estimated using linear regression analysis adjusting for time trend (year) and seasonality (month).

The daily mean levels of PM_{10} and the other pollutants were provided for each of the 13 cities included in the study by their local monitoring networks.

We also identified the presence of Saharan dust advection and computed the Saharan dust load on daily PM_{10} concentrations.[28] Briefly, the estimate of Saharan dust load was performed by using a method adopted by the European Commission, employing data from rural monitors near each city (http://ec.europa.eu/environment/air/quality/legislation/pdf/sec_2011_0208).

Saharan days were classified as advection days without any Saharan-related PM increase at ground level, days with a PM_{10} load of 1–10 $\mu g/m^3$ and days with a PM_{10} load of more than 10 $\mu g/m^3$.

Health data

Daily death counts due to natural (International Classification of Diseases Ninth Edition (ICD-9) codes 001–799 or ICD-10 codes A00-R99, excluding injuries, poisoning and external causes) and cause-specific mortality (cardiovascular ICD-9 390–459 or ICD-10 codes I00—I99 and respiratory ICD-9 460–519 or ICD-10 codes J00-J99) were collected from each city, for all-age residents, from mortality registers. Deceased participants were considered only if they died in the same city.

Data analysis

We studied the associations of smoky days as assessed by satellite, and PM_{10} as measured from fixed monitors at ground level, with natural, cardiovascular and respiratory mortality, in the period 2003 and 2010. The effect estimates were obtained for each city using Poisson regression models, simulating a stratified case-crossover approach.[29] More specifically, time trends and seasonality were controlled for by including in the regression models a triple interaction of year, month and day of the week. All effect estimates were further adjusted for population decreases in the summer and during holidays, and influenza epidemics.[30]

Figure 1 illustrates the relationships we assumed between fires, PM, Saharan dust, temperature and mortality. In evaluating the association of fire smoke with mortality, we did not adjust for daily PM_{10}, as it is an intermediate factor between fires and mortality. While when evaluating the association of PM_{10} with mortality, we adjusted for the presence of fires. In a separate model we also assessed whether PM_{10} effects were modified by wildfires, adding an interaction term between smoky days and PM levels. The p value for relative effect modification (REM)[31] was used to test the interaction hypothesis. We further adjusted the estimates of fire smoke and PM_{10} effects for temperature and Saharan dust, since they are risk factors for mortality, and are associated with the occurrence of forest fires and with PM_{10} concentrations. Low temperatures were controlled for with a penalised cubic spline for 1–6 lagged values of air temperature

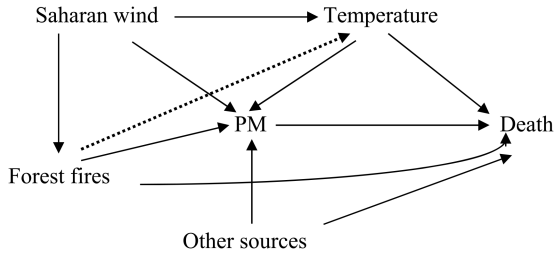

Figure 1 Direct acyclic graph exploring the effects of forest fires on Death. The contribution of forest fires on PM concentrations could not be assessed. The impact of forest fires on temperature could not be assessed.

below the median value in each city; similarly, high temperatures were controlled for with a penalised cubic spline for values of 0–1 lagged temperature above the median value at each city. Saharan dust was controlled for by adding the categorical, three-level variable specified above in the models.

We explored 6-day lags from 0 to 5 days preceding death for the association between PM_{10} and mortality. We also analysed cumulative exposure using unconstrained distributed lags.[32 33] For PM_{10} we adopted the best lags (0–1 for natural mortality and 0–5 for cause-specific mortality) previously reported from MED-PARTICLES.[34] The results were expressed as the percentage increase in risk (%IR) of natural or cause-specific mortality with 95%CIs. For PM_{10}, the effects are per 10 $\mu g/m^3$.

After city-specific analysis, pooled estimates were obtained from a random-effects meta-analysis for 10 cities (excluding Parma, Modena and Reggio-Emilia, located in the same region, where only three fire episodes occurred in 3 years). Heterogeneity across cities was assessed by χ^2 (Cochran's Q) and I^2 tests.[35] Pooled results have been reported for the best cumulative lag, as identified by the strength of the association and the lowest heterogeneity.

Finally, we carried out a sensitivity analysis by excluding the cities where temperature and PM_{10} did not increase consistently with fire smoke concentrations, suggesting a possible misclassification of exposure.

RESULTS

The number of smoky days in each city varied, with a total of 391 days affected (2.0% of the studied days). The cities with the highest number of smoky days were Thessaloniki (6% of days), Athens (4%), Madrid and Rome (3%) (table 1, figure 2). The cities most affected by severe smoke were, again, Thessaloniki, Athens and Rome (table 1). Wildfires were more likely to occur from April to September (83%) in all cities except Barcelona (38%; table 1). Thirty-two per cent of smoky days were concurrent with Saharan dust outbreaks contributing more than 1 $\mu g/m^3$ of PM_{10} at ground level. The largest overlap between smoke and Saharan dust was observed in Palermo (59% of smoky days), followed, far away by Rome (39%) and Madrid (37%), in hot as well as in cold seasons (see online supplementary figure SA).

The daily mean number of natural deaths was 36, across all cities studied. The daily mean number of cardiovascular deaths was 13 and the mean number of respiratory deaths was 4 (table 2).

Smoky days were associated with an increase of 1.78% (95% CI −0.91 to 4.53) in natural mortality (lag 0–1) and of 6.29% (95% CI 1.00 to 11.85) in cardiovascular mortality (lag 0–5). No association was observed for respiratory mortality (table 3).

Daily levels of PM_{10} (10 $\mu g/m^3$) were associated with natural mortality (lag 0–1) by 0.53% (95% CI 0.30 to 0.76), cardiovascular mortality by 0.74% (95% CIs to 0.30 to 1.18) and respiratory mortality by 1.99% (95% CI 0.80 to 3.20). The results did not change after adjusting for smoke-affected days (and Saharan dust). There was an indication that PM_{10}-related mortality was modified by smoke episodes (after controlling for Saharan dust); the effects of PM_{10} on smoky days were higher than on smoke-free days, amounting to 1.10% for natural mortality, 3.42% for cardiovascular mortality (with a borderline statistically significant effect modification; p-REM=0.055) and 3.90% for respiratory mortality (table 3).

Fire smoke intensity and duration were well correlated on the less affected days (smoke concentration between 8 and 16 $\mu g/m^3$) but not on the most affected days (smoke concentration above 32 $\mu g/m^3$); 84% of one-day events were mildly affected, whereas only 23% of 2–4-day events and 45% of 5-or-more-day events were medium/severely affected. Only 22 days were severely

Table 1 Smoke-free days and smoke-affected days by season, intensity and length of episodes in 13 cities of the MED-PARTICLES study area in 2003–2010

City	Study period	Study days (N)	No-smoky days (N)	Smoky days (N)	Smoky days (N) by season		Smoky days (N) by intensity*			Smoky days (N) by length of episodes		
					Warm†	Cold†	Mild	Med	Severe	1 day	2–4 days	5+ days
Madrid	2003–2009	2557	2490	67	59	8	45	17	5	20	42	5
Barcelona	2003–2010	2922	2875	47	18	29	45	2	0	18	22	7
Marseille	2003–2008	2190	2154	36	28	8	26	9	1	16	12	8
Turin	2006–2010	1826	1812	14	14	0	8	5	1	4	10	0
Milan	2006–2010	1826	1812	14	14	0	8	5	1	4	10	0
Bologna	2006–2010	1826	1812	14	14	0	8	5	1	4	10	0
Emilia-Romagna‡	2008–2010	1096	1093	3	3	0	3	0	0			
Rome	2005–2010	2191	2137	54	53	1	40	13	1	11	14	29
Thessaloniki	2007–2009	1096	1032	64	53	11	43	16	5	14	13	37
Palermo	2006–2009	1461	1427	34	28	6	28	5	1	8	7	19
Athens	2007–2009	1096	1052	44	42	2	30	8	6	2	16	26
TOTAL		20 087	19 696	391	326	65	284	85	22	101	156	131

*Model estimates according to Navy Aerosol Analysis and Prediction System (NAAPs).
†Warm season=April–September, cold season=October–March.
‡includes three cities (Modena, Parma and Reggio Emilia) in the Emilia Romagna region.

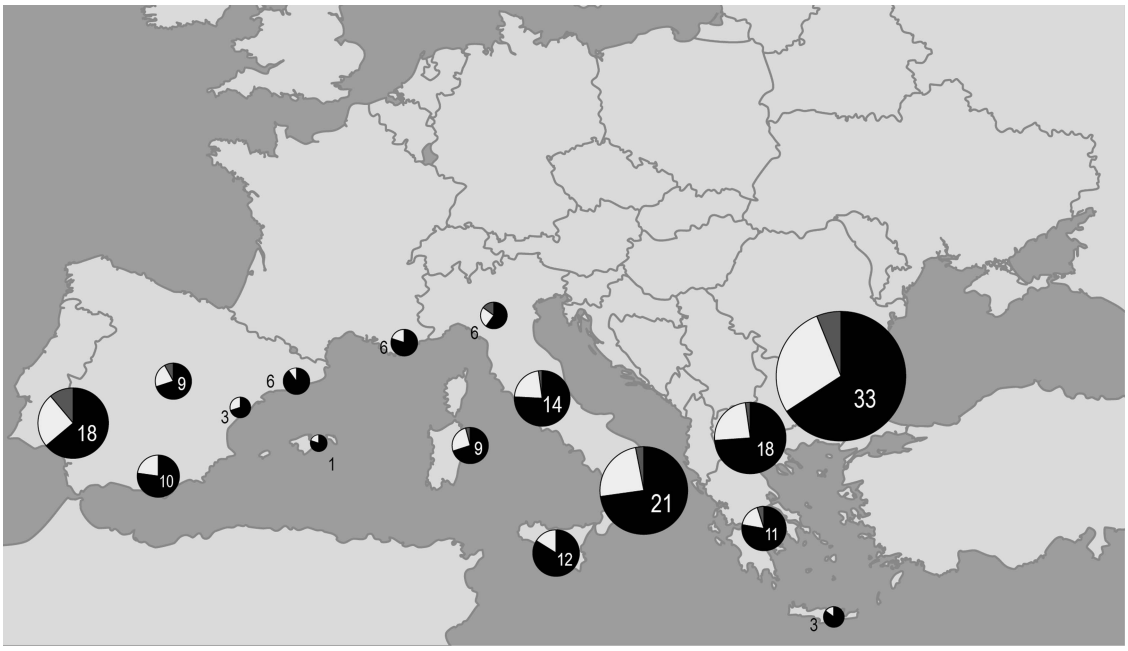

Figure 2 Location, intensity and number of forest fire episodes in the northern Mediterranean area, in the period 2003–2011.
The locations of forest fires are reported in the figure. The cities with fire areas are, from Western to East Europe: Huelva, Madrid, Malaga, Valencia, Barcelona, Palma de Mallorca, Marseille, northern Italy (Turin, Milan, Bologna), Rome, Cagliari, Napoli/Bari, Palermo, Thessaloniki, Athens, Crete, Sofia.
Intensity was classified as low (black, for smoke concentration between 8 and 16 μg/m3, medium (light grey) for smoke concentration between 16 and 32 μg/m3 or severe (dark grey) for smoke concentration above 32 μg/m^3.
The annual mean number of episodes in the location is reported in each circle.

smoke affected, but there were 131 days included in events that lasted 5 or more days (table 1).

When we estimated the changes of temperature and combustion-related pollutants according to episode length (see online supplementary figure SB), we found that mean daily temperature increased by 1.7 C° on smoky days compared to smoke-free days; it increased by 0.9 C° up to 2.3 C° in the long-lasting episodes. The average daily concentrations of PM_{10} increased by around 7 μg/m^{3}on smoky days compared to smoke-free days, and from 5 to 14 μg/m^3 in summer (data not shown). CO on smoky days increased by 0.2 mg/m^3 only during the long-lasting episodes. Similarly, a clear increase in O_3 concentrations (up to 9 μg/m^3) was observed during long-lasting smoke episodes (see online supplementary figure SB). When we estimated the changes in fire-related pollutants by fire smoke intensity, we found a stronger relationship with PM_{10}, and a weaker relationship with CO and ozone (see online supplementary figure SB).

After excluding Turin and Milan (where neither temperature nor PM_{10} increased during fire events) from the analysis, the pooled mortality estimates of PM_{10} showed a stronger increase of respiratory mortality on smoke-affected days than on smoke-free days, in comparison with the base estimates, which included the two cities (see online supplementary table SA).

Table 2 Mean number of deaths that occurred on smoke-free days and smoke-affected days by intensity in 13 cities of the MED-PARTICLES study area in 2003–2010

			Natural deaths (daily mean N)				Cardiovascular deaths (daily mean N)				Respiratory deaths (daily mean N)			
					By smoke intensity*				By smoke intensity*				By smoke intensity*	
City	Study period	Study days (N)	All days	All smoky days	Mild	Med-severe	All days	All smoky days	Mild	Med-severe	All days	All smoky days	Mild	Med-severe
Madrid	2003–2009	2557	60.1	55.8	55.0	57.4	18.0	16.2	16.0	16.8	9.6	7.8	8.0	7.4
Barcelona	2003–2010	2922	41.7	44.6	44.3	47.0	13.3	13.9	13.8	14.2	4.6	5.2	5.1	5.6
Marseille	2003–2008	2190	21.8	24.7	23.3	28.4	6.7	7.5	7.1	8.5	1.5	1.9	1.7	2.2
Turin	2006–2010	1826	20.5	20.9	20.5	21.3	7.9	8.6	8.6	8.7	1.6	1.6	1.5	1.8
Milan	2006–2010	1826	34.9	33.3	31.5	35.7	12.4	12.1	11.5	12.8	3.0	2.4	2.1	2.7
Bologna	2006–2010	1826	10.6	12.2	11.4	13.3	4.1	5.2	4.4	6.3	1.0	1.1	1.0	1.3
Emilia-Romagna†	2008–2010	1096	13.1	12.0	12.0	–	5.2	4.0	4.0	–	1.0	1.0	1.0	–
Rome	2005–2010	2191	57.9	54.5	53.2	58.1	23.6	21.6	21.9	20.8	3.6	2.9	2.8	3.1
Thessaloniki	2007–2009	1096	17.9	18.7	18.1	20.0	8.3	8.8	8.6	9.2	1.7	1.6	1.5	1.8
Palermo	2006–2009	1461	15.3	14.7	14.9	13.7	6.2	6.3	6.4	5.8	0.9	0.9	0.9	1.0
Athens	2007–2009	1096	80.6	84.1	81.6	89.4	36.3	38.1	36.8	41.0	9.2	8.4	8.7	7.9

*Model estimates according to Navy Aerosol Analysis and Prediction System (NAAPs).
†Includes three cities (Modena, Parma and Reggio Emilia) in the Emilia Romagna region.

Table 3 Pooled* estimates of the effects of smoke and PM_{10} (10 μg/m^3) on natural and cause-specific mortality (all ages) in 10 MED-PARTICLES cities in 2003–2010

	Natural mortality, lag 0–1						Cardiovascular mortality, lag 0–5						Respiratory mortality, lag 0–5					
	Per cent	95% CI		I^2 (%)	p-het	p REM	Per cent	95% CI		I^2 (%)	p-het	p REM	Per cent	95% CI		I^2 (%)	p-het	p REM
Smoke-affected days	1.78	−0.91	4.53	19	0.260		6.29	1.00	11.85	34	0.140		−3.49	−9.60	3.03	0	0.440	
PM_{10}	0.53	0.30	0.76	22	0.240		0.74	0.30	1.18	1	0.427		1.99	0.80	3.20	39	0.097	
PM_{10}†	0.51	0.16	0.86	50	0.035		0.70	0.14	1.27	25	0.213		2.17	0.89	3.46	43	0.068	
PM_{10}‡																		
On smoke-free days	0.49	0.14	0.85	49	0.040		0.65	0.10	1.19	21	0.252		2.13	0.85	3.42	43	0.072	
On smoke-affected days	1.10	−1.51	3.77	51	0.033	0.655	3.42	0.64	6.28	0	0.491	0.055	3.90	−1.63	9.74	0	0.888	0.549

*From random meta-analysis.
†Adjusted for smoky days and Saharan dust in three levels.
‡Adjusted for Saharan dust in three levels and stratified in smoke-free days and smoke-affected days.
p-het, p value of the heterogeneity test; PM, particulate matter; p REM means p value of the difference between the effects on the smoke free days and on smoke affected days; REM, relative effect modification.

DISCUSSION

We found that cardiovascular mortality was significantly higher in the Mediterranean cities on smoky days. There was a weaker association with natural mortality and no association was observed with respiratory mortality. We also found that PM_{10} effects on natural, cardiovascular and respiratory mortality were greater on smoky days than on other days, while an effect modification was clear only for cardiovascular mortality.

While high toxicity of particles from wood fires (higher than from particles originating from other sources) has been reported in experimental and toxicological research,[6 36] epidemiological studies have reported conflicting effects of particles on cause-specific mortality on smoky days,[9 17–19 22] or very similar effects of PM_{10} on smoke-affected and smoke-free days.[21 22] Our results indicate that PM_{10} from forest fires increases mortality more than PM_{10} from other sources does. It is possible that the stronger effects of particles during smoke-affected days are due to differences in their composition, but other factors also play a role in increasing mortality on those days, such as temperature increase. Cardiac patients are more susceptible than other participants to high temperatures that, in turn, are known to enhance the effects of ambient particles.[37]

The mortality increase associated with PM_{10} is consistent with the estimates reported in multicity European studies: APHEA2,[38] APHENA[39] and EpiAir.[40] All these studies also showed higher PM_{10} effects on respiratory mortality. Then, the effects we found on cardiovascular mortality during fires may be due to a different PM composition or increasing temperature.

In contrast, results from studies on the effects of wildfire emissions on cause-specific mortality have been inconsistent. Johnston[17] reported the highest effects on cardiovascular mortality, but Morgan[22] did not find any consistent effect with cardiovascular deaths in Australia, and Analitis[9] found the highest effects on respiratory mortality in Greece; this last study, however, used an exposure definition that differs from nearly every other fire smoke study. The toxicological studies on effects of fire smoke usually focus on lung damage and have consistently reported trachea-bronchial cell injuries, changes in the immune cell morphology in the lungs and diminishing ventilator responses.[6] On the other hand, it may be that different degrees of toxicity on cardiovascular and respiratory systems are due to different PM_{10} components or to varying gaseous emissions (CO, VOCs, NOx or SO_2) from wildfires. Natural mortality has been already reported as less affected by fires[9 17] when compared to cause-specific mortality. We did not attempt to explain the high heterogeneity of PM_{10} effects on natural mortality during fires, however, it is worth noting that natural mortality is likely to be penalised by a misclassification of accidental deaths (injuries, poisoning and external causes); these causes of death are usually not included as plausible effects of air pollution, but are likely to occur during fire episodes or result from them at longer distance, in the case of poisoning.

An underestimation of PM levels from wildfires at ground level is usually due to satellite observations, which incorrectly identify some aerosol plumes as clouds, and fires produce smoke as thick as some clouds.[10] On the other hand, an overestimate of PM from wildfires would occur because of their high prevalence of carbonaceous particles, increasing the absorption of the satellite signal. Therefore, a misclassification was the most likely bias affecting our assessment of exposure. The sensitivity analysis we performed excluding cities with no PM and temperature increases on smoky days, supports the hypothesis of a misclassification of smoky days in the two cities.

We did not have chemical transport models available to estimate PM aerosol vertical profiles, though they have been shown to improve the accuracy of satellite estimates of $PM_{2.5}$,[41] nor were we able to directly estimate the contribution to PM_{10} from forest fires. Therefore, to validate fire exposure, we used indirect indicators, such as fire-related pollutant levels from fixed monitors despite the important assumptions this required. We observed a clear PM increase on smoky days and this is consistent with previous studies, which used PM increases as a fire exposure indicator,[21 22] or validated the satellite data on fires using background $PM_{2.5}$.[41]

Assessment of fire smoke intensity is even more likely to be affected by misclassification; it relies on fire characteristics not directly related to human exposure, such as the extension of the burnt area,[9] AOD from satellites[42] or plume detection.[26 43] The weak consistency we observed between smoke intensity and duration with fire-related indicators, induces caution in relying on intensity estimates based on satellite data. Moreover, the high correlation we observed between the shortest episodes and the mild smoke intensity fell very much between the longest events and days of intense smoke. A recent study aids in understanding this issue; Yao and Henderson[44] validated an empirical model to estimate forest fire-related $PM_{2.5}$ using background PM, remotely sensed aerosols and remotely sensed fires, smoke plumes from satellite images, fire danger ratings and the venting

index (the probability of the atmosphere to disperse smoke from a fire). In contrast to our results, the correlation between estimated and observed values was 84%, and decreased on days with moderate to low levels of smoke up to 59%–58%. Thus the model more reliably assessed exposure to high-intensity smoke, than to smoke of low intensity.

CONCLUSIONS

We observed increases in natural and cause-specific mortality on smoky days; mortality from cardiovascular causes had the largest increase. PM_{10} had larger effects on cardiovascular and respiratory mortality on smoky days than on other days, suggesting a priority role of particulate as an effective component of fire smoke. Our study highlighted the need to make improvements in exposure assessments and estimations of fire-related health outcomes. Wildfire exposure assessment would benefit from remote sensors, source apportionment of particles during fires and from a detailed definition of their components, as well as assessing fire-related increases in temperature. A better understanding of the role that meteorology plays in influencing the direction and the spatiotemporal extension of wild fires is also important. Health assessments could benefit from the analysis of other health outcomes such as accidental causes of death during fires, and specific syndromes related to fire resulting at longer distance.

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Acknowledgements The authors thank Margaret Becker for revising the English and Simona Ricci for her help with the figures. The authors express their gratitude to the Naval Research Laboratory for the provision of NAAPS aerosol maps (http://www.nrlmry.navy.mil/aerosol/), without which this study would not have been possible.

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Funding This research was supported by the European Union under the grant agreement LIFE+ ENV/IT/327.

Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

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Short-term effects of particulate matter on mortality during forest fires in Southern Europe: results of the MED-PARTICLES Project

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Occup Environ Med 2015 72: 323-329 originally published online February 17, 2015
doi: 10.1136/oemed-2014-102459

Notes

Version 1. PLoS Curr. 2012 November 2; 4: e4f959951cce2c.
Published online 2012 November 2. doi: 10.1371/4f959951cce2c
PMCID: PMC3492003
PMID: 23145351
Research Article

Health Impacts of Wildfires

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Abstract

Introduction Wildfires are common globally. Although there has been considerable work done on the health effects of wildfires in countries such as the USA where they occur frequently there has been relatively little work to investigate health effects in the United Kingdom. Climate change may increase the risk of increasing wildfire frequency, therefore there is an urgent need to further understand the health effects and public awareness of wildfires. This study was designed to review current evidence about the health effects of wildfires from the UK standpoint. Methods A comprehensive literature review of international evidence regarding wildfire related health effects was conducted in January 2012. Further information was gathered from authors' focus groups. Results A review of the published evidence shows that human health can be severely affected by wildfires. Certain populations are particularly vulnerable. Wood smoke has high levels of particulate matter and toxins. Respiratory morbidity predominates, but cardiovascular, ophthalmic and psychiatric problems can also result. In addition severe burns resulting from direct contact with the fire require care in special units and carry a risk of multi – organ complications. The wider health implications from spreading air, water and land pollution are of concern. Access to affected areas and communication with populations living within them is crucial in mitigating risk. Conclusion This study has identified factors that may reduce public health risk from wildfires. However more research is needed to evaluate longer term health effects from wildfires. An understanding of such factors is vital to ensure preparedness within health care services for such events.

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Introduction

Wildfires, in the form of bush fires, vegetation fires, forest fires, heath and grass fires, are prevalent throughout the world. Recent high profile events in Chile [1], Australia [2] and California [3] have reminded the global community of the devastating effects uncontrolled fire may cause. The threat is closer to home too; there are on average 70 000 forest fires annually in Europe alone [4], and whilst these predominantly occur in countries with warmer climates such as Portugal and Greece, wildfires, in the form of uncontrolled burning of vegetation (albeit not full forest fires), do occur with increasing frequency in the UK [5]. According to the Department for Communities and Local Government, Fire Statistics Branch, there were over 58 000 grass and heathland fires in Great Britain in 2010/11 [6].

The UK Climate Change Risk Assessment (CCRA) 2012 has cited wildfires amongst the top seven risks to the natural environment in England [7]. With climate change, the risk of wildfires is likely to increase and a 30% to 50% increase in wildfires by 2080 is predicted [8]. The health effects of heat waves and heat exposure

are well documented, but the health effects of wildfires are less widely known. Whilst much work has been done by agencies such as the Forestry Commission, the Meteorological Office and the Fire and Rescue Service to investigate how best to predict and limit damage from fires, little equivalent work has been done from the perspective of healthcare. Understanding the health impacts of wildfires and ensuring that our front line health care services are equipped to deal with them can help to reduce suffering in the aftermath of a wildfire.

Open in a separate window
Location and size of Wildfires in England FY 2009/10 – 2010/11

Courtesy of Forestry Commission England.

Data source: Department for Communities and Local Government (National Incident Recording System)

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Aim

The object of this study was to collate and review the evidence regarding human health impacts from global wildfire experience. Understanding these impacts, and mitigation measures adopted in other countries, should help to increase awareness amongst health professionals and enable the UK to prepare effectively for the increase in wildfires that are likely to occur during this century as a result of climate change.

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Methods

Protocol

A search strategy was devised to study the available literature across various databases. Literature up to the end of January 2012 was included.

Eligibility Criteria

Inclusion criteria were:

- All papers relating to wildfires and human health

Exclusion criteria were:

- All subjective reports containing no scientific data
- Reports relating only to geographical and forestry issues around wildfires
- Literature relating to smoke and fires from sources other than vegetation

Information Sources

The following databases were searched

- Medline
- Embase
- Cochrane
- Google Scholar

Search

Search items included in various searches were:

Wildfires
Forest Fires
Bushfire
Grass Fire
Health
Human

These were used in varying combinations using OVID as the primary interface. The Cochrane database was searched but revealed no further relevant papers. The first 100 hits on google scholar were reviewed by title alone, which revealed some useful background literature but no further key papers.

There is no set definition of a "wildfire" but it is widely understood to mean the uncontrolled burning of vegetation. The search terms were chosen after a collective discussion between experts in the field to select the most appropriate terminology accounting for both technical and colloquial description of the above.

Search - Examples of search using Ovid interface

1 (forest and fire).mp. [mp=title, abstract, full text, caption text]
2 (Grass and fire).mp. [mp=title, abstract, full text, caption text]
3 (Wild and fire).mp. [mp=title, abstract, full text, caption text]
4 (Bush and fire).mp. [mp=title, abstract, full text, caption text]
5 health.mp. [mp=title, abstract, full text, caption text]
6 human.mp. [mp=title, abstract, full text, caption text]
7 1 OR 2 OR 3 OR 4
8 5 AND 6
9 7 AND 8
10 limit 9 to (clinical medicine and original articles)

Total hits: 424

(Hand searched by abstract to include relevant papers only)

Study Selection

Studies were selected by review of titles and abstracts. If full text was readily available this was also reviewed. The bibliographies of each relevant paper were hand searched for further relevant documents.

The majority of literature pertaining to this subject was in the form of epidemiological observational studies and case reports.

A total of 81 useful and relevant papers were identified from the literature searches and bibliography searches. These were mainly in English although one Spanish document was identified.

Risk of bias in individual studies

There is a risk of selection and observational bias within the papers chosen, but due to the paucity of original research in this area, studies were not excluded because of this risk alone.

Risk of bias across studies

Publication bias may also be an issue as it is likely that data from high profile wildfires is more likely to reach the public domain than that from smaller episodes. Funding bias may also be an issue as the chance of gaining sponsorship to investigate and report effects from smaller fires may be difficult.

Grey literature was reviewed and expert opinions sought. Meetings and telephone conferences were arranged with experts in the field, including representatives from the Forestry Commission and Fire Services.

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Results

Study Selection

A total of 76 relevant papers were identified from the literature searches and bibliography searches. These were mainly in English although one Spanish document was identified.

Study Characteristics

The majority of literature pertaining to this subject was in the form of epidemiological observational studies and case reports.

In light of the high incidence of wildfires, there is surprisingly little literature relating solely to their health effects. Much of the literature refers to just a few renowned events, some of which are presented below (table 1) whilst examples of recent wildfires in England illustrate the fact that although these wildfires may be smaller than those abroad, their potential impact may be significant (table 2).

Table 1 - Case studies: reports of international wildfire events of note

Location, date	Details
Sydney, 1994	Over 800 extensive bush fires spread along the coast of New South Wales in summer 1993-1994. Four people were killed - two civilians and two fire fighters, and 27 250 people were evacuated. 800 000 hectares were burnt. 225 homes and other buildings destroyed and a further 150 were damaged [9].
Indonesia, 1997	Widespread bushfires in Indonesia in 1997 (over 5 018 000 hectares [10] resulted in a haze of air pollution which resulted in severe adverse health effects in Indonesia, Malaysia and

Location, date	Details
	Singapore [11] .
Canada, 2003	2500 fires started in British Columbia in 2003 during a period of particularly hot, dry weather [12]. Many urban areas were affected; 334 homes were destroyed and 45 000 people evacuated. The total cost of the fire storm is thought to be around $700 million (Canadian dollars). Three deaths were reported – all pilots who died when trying to put out the blaze.
California, 2007	Wildfires in Southern California in October 2007 burnt over an area of 202 300 hectares, destroying around 1 500 homes. Nine people died [13].
Victoria, Australia, 2009	Black Saturday, one of Australia's worst natural disasters occurred on 7 February 2009, when temperatures in Melbourne reached 46.4°C (the hottest on record). Bushfires which had started earlier in the day swept across the region, blown by 100km/h winds. Over 141 600 hectares burned. 173 people died, 414 were injured [14].
Russia, 2010	In summer 2010, the western part of the Russian Federation experienced extreme heat and severe wildfires. More than 20 000 forest fires over an area of 2800 km2 were recorded, emitting high levels of carbon monoxide and particulate matter. Cumulative excess deaths in July and August of 2010 amounted to 54 000 compared to the same period in 2009 [15].

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Table 2 - Case studies 2: Examples of recent wildfires in England

Location, date	Details
Swinley Forest, May 2011	May 2011 saw one of the biggest English wildfires – in Swinley, Berkshire [16]. Over 105 hectares of forest burnt, fanned by strong winds on a background temperature of 30°C [17]. Several local schools were closed due to the risk of smoke induced illnesses. The fire spread close to Broadmoor high security hospital, and urgent plans for evacuation of patients were made. Several local homes adjacent to the site were evacuated.
Pitbright Ranges, 2003 and 2010	In 2003 and 2010 over 850 hectares of lowland heath burnt at Pirbright Ranges [18], owned by the Ministry of Defence. Again high temperature, low humidity and strong winds created a large fire front, at times 2 km wide. Along with road closures and significant local disruption, military housing adjacent to the ranges was evacuated and smoke threatened a large housing estate to the west with vegetation ash landing several km away in Farnborough and Aldershot.
Thursley Common, 2006	Natural England's National Nature Reserve in Surrey burnt in 2006, the wildfire covering over 160 hectares [19]. As well as threatening several private properties adjacent to the site and closing several roads, three fire fighters suffered burns requiring hospital treatment.

Results from the searches can be summarised as follows:

- *Health threats and the Source-Pathway-Receptor model*. This is a model of the means by which wildfires and health concerns can be explored further (see below).
- *Toxicology of wildfire smoke*. Some research has been done looking at the toxicology of wildfire smoke. Identifying its toxic components could help improve our understanding of adverse health effects caused. Particulate matter from wildfire has been shown to differ from other sources of particulate matter, of relevance to our understanding of its toxicology.
- *Health effects*. Many systems are affected by wildfire smoke, predominantly through the respiratory system. Cardiovascular effects and ocular problems can also occur as well as acute burns. Psychological and psychiatric effects can be significant in relation to larger fires.
- *Water and land pollution*. Both water and soil pollution can cause longer term threats to human and ecosystem health after a wildfire.

- *Resource and access*. The effect that a wildfire has on access to healthcare services and vital resources can adversely affect health. Serious wildfires could overwhelm local healthcare resources unless a clear plan is in place.
- *Communication*. The importance of effective communication in mitigating against adverse health effects is emphasised.

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Health Threats: source-pathway-receptor model

The Source-Pathway-Receptor Exposure Model, used for risk assessing the impact of acute and chronic chemical exposures on health e.g. in flooding and contaminated land studies [20] can also be used to examine the potential health effects of wildfires.

Source

Heat and smoke from burning vegetation can cause adverse health effects. The majority of UK wildfires are small and contained, but as shown in 2010 and 2011 larger, more landscape scale incident can occur. The most significant land use type of areas burnt were open habitats (mountain, moor, heath and grasslands). Types and amount of fuel, past and present weather conditions and topography can all affect fire intensity, and thus the degree and type of health effect [21]. Even within England, sources of fuel for wildfires vary significantly (Figure 2).

Open in a separate window
Land types burnt in wildfires FY2009/10-2010/11

Courtesy of Forestry Commission England.

Data source: Department for Communities and Local Government (National Incident Recording System) [22]

Pathway

Four main exposure pathways exist:

- Direct exposure to the flames and radiant heat;
- Exposure to smoke from burning or smouldering material dispersed through the air;
- Exposure to Land/soil contaminated by the chemical products of burning vegetation, from soil erosion caused by vegetation removal during the fire, or from suspended dust dispersed through the air;
- Water contamination, caused by particulate matter deposition on water or leachates from the land directly affected by fire.

Receptor

The receptors in this study are humans who live in the vicinity of the fires who may experience adverse health effects. The vulnerability of the population to potential harm depends on many factors [23], most notably:

- Prevalence of demographically vulnerable groups such as those at the extremes of age, pregnant women, those of poor socio-economic status
- Prevalence of pre-existing disease in the community (especially cardiac and respiratory)
- Access to publically - available information on risk mitigation
- Emergency Preparedness including the presence of an early warning system
- Location of dwellings, workplaces and critical infrastructure in relation to the fire, and access/egress from them.
    - An example of this is the 2011 Swinley Wildfire in Berkshire that threatened buildings including Broadmoor Psychiatric Hospital (Case studies 2).

Vulnerable populations are more likely to suffer adverse effects from wildfire smoke, thus actions should be taken to mitigate risk where possible, for example by ensuring clear access to reliable public information and a robust system of emergency preparedness.

Toxicology of wildfire smoke

Wildfire smoke consists of particulate matter and gaseous products of combustion. Adverse health effects, including an increase in daily mortality, have been linked to air pollution associated with bushfires and dust storms. A recent Australian study looking at the effects of bushfires between 1997 and 2004 illustrates this [24]: A 5% increase in non-accidental mortality (OR 1.05 (95%CI: 1.00–1.10)) was observed on days of high air pollution from bushfire smoke.

Particulate matter is the predominant air pollutant seen in bushfire smoke, caused especially by the burning of vegetation and wood [25]. PM_{10} particles (which are able to pass through the upper respiratory tract and are deposited in the airways), and $PM_{2.5}$ particles (may be respired deeper within the lungs and deposited in the gaseous exchange region of terminal bronchi and alveoli) are produced by burning vegetation. Health effects of particulate matter are well documented: a 0.5-2% increase in mortality with each increase of 10 $\mu g/m^3$ of urban $PM_{2.5}$ has been observed [26]. COMEAP have reported a 6% (2- 11 95%CI) increase in all cause mortality associated with this increase in $PM_{2.5}$.

Gaseous emissions including carbon monoxide, nitrous oxides and benzene are produced [27], as are carcinogens including polycyclic aromatic hydrocarbons (also be present on particulate matter), aldehydes, and volatile organic compounds [28]. These compounds would also be expected to be adsorbed on the surface of the particulate products. Levels of ozone and nitrous dioxide are also seen to rise [27]. Wildfires often have a high proportion of smouldering fuel (i.e. thermal breakdown of fuel in a normal oxygen level without flame). This is a form of incomplete combustion and is likely to produce high levels of toxins [29]. As the fires are open air, the direct health effect of these toxins is likely to be low as their concentration is quickly dispersed, and toxins such as carbon monoxide are unlikely to cause immediate clinical concern. However, the longer term health effects from low level exposure to aldehydes and other carcinogens from bushfire smoke may remain a cause for medical concern.

Tan et al. [30] monitored white blood count levels in humans after the Indonesian wildfires (case studies 1) and found increased levels of polymorphonucleocytes indicating possible increased bone marrow activity which may be linked (although inconclusively) to the toxins in wildfire smoke.

Studies of the effects of particulate matter in wildfire smoke

$PM_{10}$from wildfires appear to have different effects on health than urban PM_{10}. An 8 year study investigating air pollution levels, including those from bushfires, and hospital admissions showed that a 10 $\mu g/m^3$ increase in bushfire (but not urban) PM_{10} was associated with a 1.24% increase in all respiratory admissions, a 3.80% increase in COPD admission and a 5.02% increase in adult asthma admissions. Increased levels of urban PM_{10} were associated with an increase in all-cause and cardiovascular mortality but not respiratory mortality [31].

Studies from Darwin, Australia, are particularly useful as there is very little background urban PM_{10} pollution and therefore most rises in air pollution are secondary to bushfires [32]. Studies from this area [33, 34] report a significant increase in asthma and COPD presentations associated with raised PM_{10} levels from bushfire smoke.

The difference between urban and wildfire smoke is also illustrated in a study looking at the effect on macrophages exposed to wildfire and urban smoke in a murine model [35]. This showed that although cytokine production in response to wildfire smoke was lower than with urban derived particles, there was increased inflammatory (determined by measuring proinflammatory cytokines) and cytotoxic activity (as measured by biochemical markers of toxicity, apoptotic activity and nitrous oxide production) per cubic metre of air containing wildfire particles than with air containing only urban particulate matter. This was probably as a result of a higher concentration of PM_{10} particles in the wildfire smoke (10.3μg/m3 compared with 5.5μg/m3 in urban air). This increased particulate size means that particles can accumulate in the lung more easily, which may have public health implications.

Wood smoke particles have also been shown to cause an inflammatory response in otherwise healthy humans. Ghio et al.'s [36] study of 10 human volunteers exposed to woodfire smoke showed an increased level of blood neutrophils, and a neutrophilic influx into the lung from bronchial and bronchoalveolar lavage samples. Although it was a small study, the authors suggest that systemic and pulmonary inflammation in human subjects can result from exposure to wood smoke particles.

Firefighters are at particular risk of inhalation of wildfire smoke particles. In recognition of this, suitable filters need to be used in breathing apparatus., such as the POVF (particulate/organic vapour/formaldehyde) filter [37].

A crude extrapolated estimate from Finland suggested that high $PM_{2.5}$ levels following wildfires in 2002 caused additional total mortality of 9-34 cases in a population of 3.4 million compared with what would normally be expected [38]. High levels of PM_{10} (both urban and from bushfires) were associated with a 1.8% increase in ED attendances in a study in Victoria (carried out during a bushfire season in 2003-2003) [39]. A system of monitoring air pollution during and after wildfire events may provide useful public health information, facilitating preparedness for increased pressure on health care services, and should be considered. It would be important to monitor $PM_{2.5}$ levels, not just PM_{10} levels. PM_{10} monitoring alone may not adequately represent the adverse effects of air quality that may be caused by the $PM_{2.5}$ fraction [38].

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Other Health Effects

Health effects of wildfires are wide ranging, and may result directly from both the thermal effects and smoke. Further health implications include: psychological reactions to an extreme event, physical concerns such as trauma during evacuation, and pressures on local resources, from increased demand on health services and inability of patients with chronic health care conditions to access healthcare facilities.

Respiratory symptoms

Certain population groups are at particular risk of respiratory effects from bushfire smoke, including young children, those with pre-existing cardiopulmonary conditions, and smokers [40].

Patients with COPD have been noted to be at increased risk as a result of air pollution. A study looking at symptoms of 21 local patients with COPD in the two months following the Denver wildfires of 2002 revealed that dyspnoea, cough, chest tightness, wheeze and sputum production all increased on days when $PM_{2.5}$, PM_{10} and carbon monoxide levels in the atmosphere increased, thus illustrating the link between air pollution from wildfires and COPD exacerbation [41].

In a paediatric cohort study 32 children with a history of wheeze suffered adverse effects from increased PM_{10} particles after the 2009 Australian bushfires, although whilst symptoms of evening wet cough worsened, dry cough and wheeze did not [42].

A study of 465 non-asthmatic teenagers affected by 2003 wildfires in Spain revealed that healthy patients with estimated smaller airways who performed on the lowest quartile of lung function tests were more susceptible to the respiratory effects of wildfire smoke [43]. Those with smaller airways and poorer pre-existing lung function were more vulnerable to smoke effects.

Data from the 1994 Sydney Bushfires show that there was no increase in acute asthma related admissions in central Sydney [44] or Western Sydney [45] in the aftermath of the fires. This may not however reflect the true prevalence of asthma exacerbations, as only the more severe cases would present to the ED. In the days following the 1987 Californian bushfire, there was a 40% increase in ED attendances [46].

A cohort study from Darwin Australia shows that studies looking at hospital attendances alone may underestimate the respiratory symptoms[47]. In this study, 251 adults and children were asked to keep a record of their asthma symptoms during a 7 month bushfire period in 2004. During this time, PM_{10} ranged from 2.6 – 43.3 $\mu g\ m^{-3}$. High PM_{10} levels were significantly associated with an onset of asthma symptoms, use of oral steroid medication, the mean daily symptom count and the mean daily dose of beta 2 agonists. However, there was no increase in the numbers of health care attendances or severe asthma attacks

An increase in respiratory symptoms and deteriorating lung function was also seen in a study of findings reported by respiratory physicians and governmental reports in Indonesia at the time of the Indonesian bushfires of 1997 [48]. Worsening of respiratory symptoms were seen during the same period in surrounding countries such as Malaysia [49] and Singapore [50], illustrating the ability of particulate matter and air pollution to spread widely. 94% of the air particles noted in Singapore in the haze following the Indonesian fires were $PM_{2.5}$, and emergency department attendances related to the haze increased although overall hospital admissions due to respiratory effects did not[50]. These effects were observed more than 500 km from the bushfires.

Delayed health effects may also occur: one study looking at health effects after the 2003 Canadian wildfires showed that there was no increase in presentations to medical services for mental health or cardiovascular problems, but there was a peak in respiratory consultations 5 weeks after the fires. This may be because of delayed respiratory health effects of wildfires smoke [51]. In addition, long term exposure to particulate matter may increase susceptibility to infection possibly through an impairment of respiratory clearance systems [52], thus helping to explain an increase in pneumonia and acute bronchiolitis seen after the 2003 Californian wildfires.

Despite frequent fires in Europe, very little literature exists on potential associated health effects. The vegetation fires which surrounded Vilnius in Lithuania in 2002 caused increases in hospital attendances for respiratory conditions and asthma [53]. These peaked in September (the fires started in early August), possibly due to delayed respiratory effects or increased air pollutant levels. High levels of ozone, PM_{10}, nitrogen dioxide and carbon monoxide were noted in the atmosphere. Possible recommendations may be to administer steroids more readily than usual for asthmatics, and recommend the avoidance of outdoor activities for those vulnerable to respiratory pathology [52].

Although these studies on the respiratory effects reported from wildfires have been undertaken, little data on the levels of pollutant exposure has been documented reducing the ability to extrapolate this information to UK wildfires.

Burns

An obvious risk to those in very close proximity to wildfires (most likely those failing to vacate the fire area or fire fighters) is that of direct flame burns and thermal burns. Clinical management will be similar to

that of a normal burns patient where there has also bee inhalation of combustion products. Patients admitted to the ICU with burns after the Victoria Wildfires differed from "usual" burns patients in that the degree of early multi-organ failures and the severity of inhalational burn were higher than expected for the degree and percentage of burns [14]. Another major factor is the number of casualties and potential demand on healthcare resources, depending on the scale of the fire and its location in relation to urbanisation.

Severe burns require specialist multidisciplinary resources, which could become overwhelmed in the case of severe wildfire. National burns disaster plans, such as the Australian Mass Casualty Burn Disaster Plan, instigated at the time of the "Black Saturday" disaster in Victoria, Australia [54], can help to mitigate against problems of overwhelmed resources.

In the 72 hours following the 2009 Victoria Wildfires, 17 patients presented to local EDs with burns of >10% of body surface area (BSA) and another 129 with burns of <10% of BSA [14]. 20 patients were managed at the specialist tertiary adult burns centre, 19 of whom needed surgical procedures (such as escharotomies or debridements). The total theatre time for these burns patients in the first 72 hours was 48.7 hours [14]. This illustrates the extra impact on resources of a surge of burns patients; even a small number of casualties can require significant resources.

If there are large numbers of burns presenting to hospital, careful triage is mandatory. Initial measures such as fluid resuscitation, analgesia and covering affected areas with cling film can be instigated in the ED relatively easily even to larger numbers of casualties. Such triage will avoid overloading specialist burns centres with the more minor burns that can be effectively treated locally. The triage category for the more severely injured may also have to be adapted; for example, under the Australian mass casualty burns disaster plan, the threshold of burns for admittance to the specialist burns centre (>20%BSA) was revised to BSA >30% in order to avoid overwhelming specialist services [14].

Heat Induced Illness

Heat induced illness can be caused by working in hot and humid conditions and will be affected by proximity to the fire. The extent of heat related illness will not be covered in full in this paper except to note that those directly involved with fire fighting are particularly vulnerable.

Modern fire fighters' personal protective equipment (PPE) is comprised of tunics and leggings. The retention of heat within the structural PPE can cause heat related illness, initially heat exhaustion leading to heat stroke. Careful design of wildfire PPE, rest periods, adequate hydration and health awareness of those involved is extremely important [55].

Cardiovascular effects

There was a slight increase in admissions to the ED with ischemic heart disease on days when there was air pollution from Sydney wildfires [31]. Cardiovascular mortality rates also increased on days with high levels of bushfire smoke in a study of Sydney air pollution between 1997 and 2004 (OR 1.10 (95%CI: 1.00–1.20))[56]. This may be secondary to high levels of $PM_{2.5}$ but further research needs to be carried out to establish this link.

A similar picture was seen after the 2003 Californian wildfires [52], when a 6.1% increased rate admission for cardiovascular complaints was seen following the fires, including an 11.3% increased rate of admission due to cardiac failure in comparison to the air pollution levels reported before the wildfires started.

After the Darwin wildfires, an increase in cardiovascular complications was seen in the Australian indigenous population only, with a 3 day lag after raised bushfire smoke levels [34].

Ophthalmic effects

Eye irritation from air pollution has been noted, as well as reduced visibility as a result of ambient smoke [57]. Reduced visibility from wildfire smoke has caused fatal road traffic collisions, so care must be taken when travelling around areas near wildfires [58]. The police and highway agencies have an important role in restricting access to at risk areas.

Corneal abrasions have also been reported; 13% of patients fire-related presentations to the emergency department the week following Almeda County wildfires in California 1991 had corneal abrasions [59].

Psychological effects

Overseas studies demonstrate that large wildfires can be devastating, destroying not only lives but livelihoods, homes and communities. This is strikingly illustrated by Mcfarlane et al.[60], who looked at the mental health impact of bushfires in an Australian community. Twelve months after the fires, 42% of the population exposed to wildfires were classified as potential psychiatric cases (scored according to the General Health Questionnaire [61]) – more than double that seen in the non-exposed population.

A study of 357 patients who sought healthcare assistance (therefore not a random sample of exposed persons) after the 2003 Californian wildfires also gives a dramatic picture, with 33% showing symptoms of major depression and 24% showing symptoms of Post Traumatic Stress Disorder [62]. Property damage and physical injury during the fires were significantly – associated with psychopathology. This suggests that screening people who present for emergency relief centres in the aftermath of a wildfire may be of help to identify people who are particularly likely to suffer from psychopathology.

A cross sectional case control study [63] looked at those affected by the Greek wildfires of 2007. Increased symptoms of somatisation, depression, anxiety, hostility, and paranoia were found in those who were victims of the fire compared with controls. Another study of 30 adults 6 weeks post exposure to wildfires showed increased levels of posttraumatic stress, depression, and anxiety symptoms [64].

There is strong psychological link between local populations and their geographical surroundings, which assume great cultural, social and personal significance. The devastating effects of a wildfire on physical surroundings can translate to psychological distress [65]. Immense efforts to preserve buildings of importance to the community were made by Greek firefighters in the August 2007 fires who saved the temples of Ancient Olympia (home of the Olympic Games). A study of the mental health of these firefighters noted that 19 of 102 had post-traumatic stress disorder as defined by ICD-10 [66]. The authors note that early detection of post traumatic stress may help to mitigate against post disaster psychiatric morbidity.

Increased smoking and anxiolytic use has been observed after wildfire exposure. 2063 adolescents and young adults were assessed to see whether exposure to a traumatic event, in this case the 2003 Australian wildfires, increased tobacco smoking [67]. Exposure to traumatic events during the disaster, independent of PTSD symptoms, was a predictor of increased tobacco use (OR 1.12, 95%CI 1.03-1.21). Increased consumption of anxiolytics was noted in men after exposure to the 2006 wildfires in Northern Spain [68].

The nature of the media coverage following a wildfire can make a difference to the population's psychological health. Vicarious traumatisation – i.e. symptoms suggestive of post-traumatic stress disorder in patients who themselves have not been exposed to the tragedy directly but only through exposure to the media has also been noted following the 2001 New South Wales bushfires [69].

Studies of rescue workers show that psychological effects can be both delayed and in some cases beneficial. 469 firefighters were followed up for 25 months after exposure to a bushfire [61, 70]. Delayed and chronic psychiatric morbidity was more prevalent than acute morbidity, and severity of morbidity was linked to the firefighters' losses and extent of exposure. Those involved in the recovery may show some beneficial as well as detrimental psychological effects, since team building and working together have been described [71].

Paediatric Psychological Morbidity

Many studies have focused primarily on paediatric psychiatric morbidity in relation to wildfires. Younger children are at especially high risk of PTSD symptoms, as are children who perceive their own lives to be at risk or experience ongoing loss or disruption [72]. This was identified in a follow up study of 155 8-18 year olds exposed to the Canberra wildfires in 2005, leading the authors to advise that identifying and supporting younger patients and those who are experiencing ongoing disruption may help to mitigate against their development of PTSD symptoms.

Risk factors for depressive illness were assessed in a study of 2379 school children exposed to wildfires in Australia who were followed up for 6 months after the event [73]. Factors contributing to psychiatric morbidity (as measured by increased emotional stress and anxiety) included evacuation and experience. These factors were also predictive for ongoing emotional distress, as was a perceived threat to self or parents.

Continuation of psychological morbidity in to adulthood may be of concern, as illustrated by a cohort of 806 children who had been exposed to bushfires in Australia in 1983 who were followed up for 20 years [74]. Results suggested that although the impact of the bushfires on overall adult psychiatric morbidity was small, after 20 years, seventy-five per cent of the bushfire-exposed group reported some degree of distress in relation to the bushfire exposure.

A longitudinal study of children after Australian bushfires indicated that the mother's psychological reaction to the disaster had a greater impact on the child's psychiatric morbidity than the child's own exposure to the disaster. Thus targeting mothers when offering psychological support may be worthwhile [75].

Go to:

Water and Land Pollution

Examination of potential water contamination in areas surrounding the Lithuanian fires of summer 2002 show that in the autumn of that year there was a substantial (60-81%) increase in heavy metal (copper, lead and zinc) levels in surrounding rivers [76].

Ash debris following the Californian wildfires of 2007 was found to contain high levels of heavy metals, including arsenic, cadmium, copper, and lead. A national clean up campaign was organised because of concerns that exposure to high levels of such metals could cause long term health effects [77].

After the Russian wildfires in 2010, concern was raised that up to 4% re - suspension of radioactively contaminated (from Chernobyl) soil could occur in areas affected by wildfire. Increased levels of caesium, strontium and plutonium occurred [78]. However the associated health risk to the firefighters and the general public was thought to be negligible.

Go to:

Resources and Access

Access and egress routes to local hospitals may be blocked by traffic congestion as people leave the wildfire area or by the fire itself. Two hospitals were in the direct line of the 2003 San Diego Wildfire, and had to prepare for complete evacuation of the hospital at very short notice [79]. Healthcare workers in these hospitals suffered adverse health consequences from ash and smoke in the hospital ventilation system.

Some were forced to decide between responding to the hospitals' calls for more assistance and the need to evacuate their own homes.

Difficulty in accessing commodities including food and regular medication can have a significant impact, particularly on patients with chronic health conditions who may be unable to collect their normal medication or attend medical appointments. The elderly, the isolated, and those with chronic health problems will be particularly vulnerable. The US document "Wildfire Smoke, a guide for public health officials" advises that patients have at least a 5 day stock of medication available, as well as several day's worth of non-perishable food [80].

As homes can be caught up in wildfires, it is imperative that access routes for inhabitants are as safe as possible, and well signposted, especially in rural areas, so that emergency services can find them easily if needed. A leaflet published by the Scottish Wildfire forum highlights this point, advising clear signage to rural properties [81]. Power supplies may be disrupted, compounding effects on the local population. There is also a risk of electrocution from fallen power lines arcing because of water and smoke.

Go to:

Communication

A Global Early Warning System for Wildland Fire has been proposed [82]. Spanish systems of satellite surveillance have been trialled with success, and may be worth developing elsewhere [83]. Safety measures to the public faced with a wildfire threat can be issued.

An example of such a public health message is the "Ready, Set, Go" campaign in Texas [84]:

- Be ready for a fire threat
- Have situational awareness if a fire threat occurs and be "set" to leave if you need to
- Go early - leave at risk areas early

The UK government's advice [85] to "Go in, stay in, tune in", although aimed at general emergencies, may be useful to prevent exposure to air pollution from fire smoke, as sheltering can reduce exposure [86]. Obviously this is less advisable for those who are in the direct path of the fire, who may need to evacuate.

Go to:

Development Planning

In the UK the new National Planning Policy Framework [87] provides scope to improve wildfire resilience in new and existing developments under both natural hazard and climate change (mitigation and adaptation) policies. This will include:

- Residential, commercial and industrial properties,
- Nursing / care homes,
- Health care facilities (hospitals, care centres),
- Schools and other educational facilities,
- Emergency service centres,
- Transport infrastructure (road, rail, air and inland waterways etc.)
- Utility infrastructure (generation and movement of; water and sewage, gas, electricity, fuel, communications etc.)

- Other National and critical infrastructure facilities, structures and properties identified on National and Community Risk Registers

Developments, facilities, structures and properties that adjoin high risk habitats, land uses and/or landscapes are within the 'Urban / Rural Interface'. Where wildfire could be a risk to human life it must be mitigated within the Local Authority's Local Development Framework and agreed by the appropriate agencies and authorities.

Go to:

Summary of pointers to good practice

This study identifies the main health protection issues to be considered in the event of wildfire. It points to evidence based actions in response to acute events and ways to prepare for a potential increase in wildfires uedue to climate change. The issues highlighted below can be used as guidance in formulating plans to mitigate against risk to health from wildfires.

Respiratory health impacts

Emergency services and GPs should be prepared for increasing numbers of patients attending with **respiratory symptoms.**

- Those with chronic respiratory illness may experience a worsening in their respiratory symptoms.
- There may be an increased incidence of mild respiratory symptoms amongst previously healthy individuals, which may require some medical treatment.
- Increased doses of anti-inflammatory and bronchodilator medication may be required. Stocks of drugs should be sufficient to accommodate for this.

Minimising exposure to smoke

Considering the potential toxicity of wood fire smoke, it is advisable to **minimise exposure:**

- Air quality reports should be checked. These may have the potential to be used in conjunction with syndromic surveillance to understand health effects and their link with air pollution.
- Indoor air should be kept as unpolluted as possible by keeping windows and doors closed and shutting off external ventilation.

Other Systemic Health Effects

- **Burns** may pose a significant problem.
    - In severe fires systems should be in place to cope with increased pressure on resources needed to manage burns patients
    - Careful triage and judicious use of specialist burns services is needed
- **Cardiovascular morbidity** may increase – Emergency Departments and GPs should be aware of this.
- **Psychological effects** may be significant.
    - Support should be available to vulnerable groups
    - Responsible media reporting of events is important

Access and Egress

Access to homes, health care facilities and resources may be impeded.

- Systems should be in place to ensure delivery of medication and provisions to those who need them, especially vulnerable groups.
    - People living in areas prone to wildfires may be advised to keep a stock of 5 day's worth of non perishable provisions and medications.
- Measures to maximise safety of routes to and from vulnerable areas should be in place.
- Housing and evacuation routes in rural areas should be clearly signposted.

Water and Land Pollution

Water and land near the fire site may become polluted by substances present in wildfire ash.

- An assessment of land and water pollution with remediation may be necessary.
- Local authorities will be responsible for cleaning – up operations in conjunction with the Environment Agency

Visibility

Visibility can be problematic

- Road users should be made aware of the potential for low visibility when driving
- Anyone presenting with eye irritation should be screened for corneal abrasion

Communication

Good communication is vital

- Public health information should be clear and as accurate as possible
- An **early warning system** should be in place to allow communities to prepare for wildfires and, if necessary, evacuate threatened areas
    - This may be enhanced using satellite data as has been used in Spain
    - Early surveillance and models for fire prediction would also be useful
- People with pre-existing health conditions should be made aware of the potential adverse health impact of wildfire smoke. For example asthma sufferers could be advised to increase their medication if they are likely to be exposed to smoke.

Go to:

Areas for Further Work

Relatively little work has been published regarding health effects of wildfires, in the UK despite their frequency although more has been published for events abroad.

Studies focusing particularly on air pollution from wildfires within the UK could be of use, as pollution from UK fires may vary from that found elsewhere in the world.

Health care workers treating casualties from wildfires should be encouraged to publish case studies of health effects to increase the evidence base available to the international medical community dealing with

wildfires. Long term longitudinal studies looking at health effects in populations exposed to wildfires are needed as this will help to determine the level of pollution causing adverse health effects.

Go to:

Conclusion

Wildfires can cause significant health effects both in the population in the immediate vicinity and in those further from the fire (predominantly from the effects of air pollution). Simple public health advice can help to mitigate risk to health. With an increasing risk of wildfire in the UK, health care workers such as general practitioners, respiratory and emergency physicians need to understand more about the health risks of wildfires.

More research is needed to evaluate long term health effects from exposure to wildfires, and careful identification and follow up of those exposed could help in this process. The better our preparedness for wildfires in the UK, the more we can do to mitigate against their adverse health effects.

Go to:

Competing Interests

The authors have declared that no competing interests exist.

Go to:

Acknowledgments

Many thanks to Adrienne Dunne for her assistance in reviewing the toxicology of wood smoke.

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Go to:

Appendix A

Glossary

BSA	Body Surface Area
CCRA	UK Climate Change Risk Assessment 2012
CO	Carbon Monoxide
ED	Emergency Department
ICU	Intensive Care Unit
PM 10	Particulate Matter with a diameter of 10 micrometer or less
PM 2.5	Particulate Matter with a diameter of 2.5 micrometer or less
WBC	White Blood Cell

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Appendix B

Prisma Checklist

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AMERICAN JOURNAL OF INDUSTRIAL MEDICINE
INCORPORATING ENVIRONMENTAL AND OCCUPATIONAL HEALTH

Volume 50, Issue 3
March 2007
Pages 208-216

Article

Socioeconomic status, particulate air pollution, and daily mortality: Differential exposure or differential susceptibility

Francesco Forastiere MD, Massimo Stafoggia MS, Carola Tasco MS ... See all authors

First published: 17 July 2006 | https://doi.org/10.1002/ajim.20368 | Cited by: 125

Abstract

Background

Short-term increases in particulate air pollution are linked with increased daily mortality and morbidity. Socioeconomic status (SES) is a determinant of overall health. We investigated whether social class is an effect modifier of the PM_{10} (particulate matter with diameter <10 micron)-daily mortality association, and possible mechanisms for this effect modification.

Methods

Area-based traffic emissions, income, and SES were available for each resident in Rome. All natural deaths (83,253 subjects) occurring in Rome among city residents (aged 35+ years) during the period 1998–2001 were

Related Info

Metrics

Citations: 125

Am score 17

Details

Keywords

air pollution income mo
particulate matter
socioeconomic status

Publication History

Issue Online:

identified. For each deceased individual, all the previous hospitalizations within 2 years before death were available via a record linkage procedure. PM_{10} daily data were available from two urban monitoring sites. A case-crossover analysis was utilized in which control days were selected according to the time stratified approach (same day of the week during the same month). Conditional logistic regression was used.

Results

Due to the social class distribution in the city, exposure to traffic emissions was higher among those with higher area-based income and SES. Meanwhile, people of lower social class had suffered to a larger extent from chronic diseases before death than more affluent residents, especially diabetes mellitus, hypertension, heart failure, and chronic obstructive pulmonary diseases. Overall, PM_{10} (lag 0–1) was strongly associated with mortality (1.1% increase, 95%CI = 0.7–1.6%, per 10 μg/m^3). The effect was more pronounced among persons with lower income and SES (1.9% and 1.4% per 10 μg/m^3, respectively) compared to those in the upper income and SES levels (0.0% and 0.1%, respectively).

Conclusions

The results confirm previous suggestions of a stronger effect of particulate air pollution among people in low social class. Given the uneven geographical distributions of social deprivation and traffic emissions in Rome, the most likely explanation is a differential burden of chronic health conditions conferring a greater susceptibility to less advantaged people. Am. J. Ind. Med. 50: 208–216, 2007. © 2006 Wiley-Liss, Inc.

21 February 2007

Version of Record online:
17 July 2006

Manuscript accepted:
22 May 2006

Citing Literature

Citing Literature

Journal of the Air & Waste Management Association

ISSN: 1096-2247 (Print) 2162-2906 (Online) Journal homepage: https://www.tandfonline.com/loi/uawm20

Impact of smoke from prescribed burning: Is it a public health concern?

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To cite this article: Anjali Haikerwal, Fabienne Reisen, Malcolm R. Sim, Michael J. Abramson, Carl P. Meyer, Fay H. Johnston & Martine Dennekamp (2015) Impact of smoke from prescribed burning: Is it a public health concern?, Journal of the Air & Waste Management Association, 65:5, 592-598, DOI: 10.1080/10962247.2015.1032445

To link to this article: https://doi.org/10.1080/10962247.2015.1032445

Published online: 14 Apr 2015.

TECHNICAL PAPER

Impact of smoke from prescribed burning: Is it a public health concern?

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Given the increase in wildfire intensity and frequency worldwide, prescribed burning is becoming a more common and widespread practice. Prescribed burning is a fire management tool used to reduce fuel loads for wildfire suppression purposes and occurs on an annual basis in many parts of the world. Smoke from prescribed burning can have a substantial impact on air quality and the environment. Prescribed burning is a significant source of fine particulate matter ($PM_{2.5}$ aerodynamic diameter < 2.5μm) and these particulates are found to be consistently elevated during smoke events. Due to their fine nature $PM_{2.5}$ are particularly harmful to human health. Here we discuss the impact of prescribed burning on air quality particularly focussing on $PM_{2.5}$. We have summarised available case studies from Australia including a recent study we conducted in regional Victoria, Australia during the prescribed burning season in 2013. The studies reported very high short-term (hourly) concentrations of $PM_{2.5}$ during prescribed burning. Given the increase in $PM_{2.5}$ concentrations during smoke events, there is a need to understand the influence of prescribed burning smoke exposure on human health. This is important especially since adverse health impacts have been observed during wildfire events when $PM_{2.5}$ concentrations were similar to those observed during prescribed burning events. Robust research is required to quantify and determine health impacts from prescribed burning smoke exposure and derive evidence based interventions for managing the risk.

Implications: Given the increase in $PM_{2.5}$ concentrations during PB smoke events and its impact on the local air quality, the need to understand the influence of PB smoke exposure on human health is important. This knowledge will be important to inform policy and practice of the integrated, consistent, and adaptive approach to the appropriate planning and implementation of public health strategies during PB events. This will also have important implications for land management and public health organizations in developing evidence based objectives to minimize the risk of PB smoke exposure.

Introduction

With the advent of global warming, wildfires are set to increase in frequency and severity in the future (Keywood et al., 2013). Wildfires produce a large amount of smoke that disperses widely and affects population far from the fire source. Prescribed burning, also known as planned burning, is a purposeful application of fire under specified environmental conditions to a predetermined area to reduce fuel loads for wildfire suppression purposes (Penman et al., 2011). The available evidence is that the spatial area and intensity of wildfires will be reduced in proportion to the area of land burned by prescribed fires (Boer et al., 2009; Bradstock et al., 2012). Prescribed burning is also used for regenerating forests after timber harvesting (regeneration burning), and for protection and promotion of ecological assets (ecological burning) (Burrows, 2008; Wain et al., 2009). Prescribed burns are geographically widespread, and smoke production can have significant impacts on air quality (Naeher et al., 2007; Tian et al., 2008; Keywood et al., 2013).

Unlike wildfires that are of high intensity, prescribed fires are cool low-intensity burns and produce relatively short plumes (Williamson et al., 2013). While low-intensity prescribed burns (low heat, light emissions) cause minimal risk to life and property, they can however emit large amounts of smoke particulates (Wain et al., 2009; Bell et al., 2006). Furthermore, prescribed burns are conducted on a regular basis (annually) and impact communities each year. Wildfires, on the other hand, are unpredictable and episodic events. There may also be differences in the pattern of smoke exposure (such as duration and frequency) from prescribed fires compared to wildfires. Exposures to smoke plumes from prescribed fires are generally shorter in duration but occur more frequently than wildfire events, although studies are required to quantify the impacts from this. Prescribed burns are conducted under favorable meteorological conditions, for example, light winds and wind gusts, low temperature, and moderate humidity. These conditions limit the ventilation rate and smoke dispersion and

Journal of the Air & Waste Management Association, 65(5):592–598, 2015. ISSN: 1096-2247 print
DOI: 10.1080/10962247.2015.1032445 Submitted October 28, 2014; final version submitted March 13, 2015; accepted March 16, 2015.

thus promote the buildup of air pollution. As a result, smoke from prescribed burning can have a substantial impact on rural/regional areas, along with potential to impact urban airsheds due to long-range transport of smoke particles.

One of the important pollutants present in high concentrations in smoke from prescribed burns and wildfires is fine particulate matter ($PM_{2.5}$ with aerodynamic diameter <2.5 μm), and research studies have shown that $PM_{2.5}$ concentrations consistently exceed the air quality guidelines (Reisen and Brown, 2006; Naeher et al., 2007). Smaller particles are of greater public health concern than larger size fractions for two reasons: First, they remain in the atmosphere for longer periods of time, and second, they can penetrate further in the respiratory system, where they promote local and systemic inflammation.

The impacts of smoke production and other unwanted effects from prescribed burns need to be investigated in the context of the substantial public health impacts of wildfires. The latter include increases in mortality from extreme air pollution, injury, loss of assets, and degradation of water supplies (Johnston, 2009). As with any health intervention, the risks and benefits of preventive action must be balanced. If a system of elective burning operations with less extensive and more manageable fires is a practical and safer option than a regime of emergency responses to more severe and highly polluting wildfires, we need to know the safest ways of achieving this. This requires us to better characterize the impacts of prescribed fires on air quality and health, and to investigate interventions for reducing the community impacts of smoke and other risks associated with prescribed fires. In this short discussion paper we highlight (a) the impact of smoke from prescribed burning on air quality especially fine particulate matter and (b) the potential adverse impacts on health.

Prescribed Burning Practices

We restrict this discussion to the use of fire to manage fuel loads and mitigate wildfire risk in temperate climates. Tropical deforestation or savannah fires set for economic or agricultural activities are excluded, but we acknowledge that these contribute to the majority of vegetation fire emissions on a global scale.

Fuel reduction burns are carried out around the world in temperate climates. For example, in Australia around 100,000–200,000 hectares of land are burned annually for fuel reduction purposes (Wain et al., 2009). After the 2009 wildfires in Victoria, Australia, the Royal Commission inquiry into wildfires recommended expanding the prescribed burning program by burning at least 5% of the land each year, equating to 385,000 hectares, to reduce the risk of large and devastating wildfires (Teague et al., 2010). Before the 2009 fires the target area for prescribed burning in Victoria was only 130,000 hectares (2%). Another example is the fuel reduction burn activity in the southern United States (Georgia, Florida, Alabama, South Carolina), where as much as 3–4 million hectares of land are burned every year (Zeng et al., 2008).

Smoke Management

In many parts of the world smoke management programs and guidelines are being introduced to minimize smoke impacts on populations (Fernandez and Botelho, 2003; Wain et al., 2009; Sun and Tolver, 2012; Williamson et al., 2013; EPA-Tasmania, 2013). This has resulted in various air quality assessment tools being implemented and used to monitor smoke from prescribed burning, as well as smoke emissions from other sources. A good example of a smoke management program in Australia is Base Line Air Network (BLANkET) of Environment Protection Authority Tasmania (EPA-Tasmania, 2010), which is a statewide monitoring network. It consists of 19 monitors that are used to measure near real-time PM concentrations during smoke events from prescribed burning. It is also used to monitor smoke from domestic wood heaters and wildfires and generally used to provide a measure of air quality in rural areas of Tasmania. Another example from the United States is the Interagency Real Time Smoke Monitoring program (AIRSIS), which provides real-time PM concentrations from portable smoke monitors and is used during fuel reduction activities (U.S. Forest Service [USFS], 2013). Remote sensing and smoke forecasting using air quality models are also currently operational worldwide (Hu et al., 2008; Zeng et al., 2008; Johnston et al., 2010; Price et al., 2012; Johnston and Bowman, 2013; Williamson et al., 2013; Yao and Henderson, 2014; EPA, 2014). Nevertheless, there is considerable variation within and between countries, and while information describing impacts of prescribed burning (Pearce et al., 2012; Schweizer and Cisneros, 2014) on air quality is increasing, relatively little is known about the implications for human health. This is especially important in rural/regional areas where most of the prescribed burning is conducted, and where air quality can also be affected by smoke particulates from residential wood heaters, agricultural burning, and wildfires (Bell and Oliveras, 2006; Reisen et al., 2011), but where air quality monitoring is limited.

Fine Particulate Concentrations During Prescribed Burning Smoke Events

Table 1 summarizes available Australian data from internal reports and published papers that looked at impacts of prescribed burning on $PM_{2.5}$ concentrations. We only include studies from prescribed burns conducted for fuel reduction purposes and omit studies from regeneration burns. Table 1 highlights daily and hourly $PM_{2.5}$ concentrations measured over a prescribed burning season and/or during specific burn events. It includes our recent study where we investigated in 2013 the impact of prescribed burning smoke on $PM_{2.5}$ concentrations in the Yarra Valley, Victoria, a region that is regularly impacted by prescribed burns. The Yarra Valley is surrounded by mountains with steep slopes and dense forests with significant fuel loads. Monitoring was carried out at two sites approximately 9 km from each other during the autumn prescribed burning season in April. The E-sampler Aerosol

Table 1. $PM_{2.5}$ concentrations and duration of exposure during prescribed burning events

Location	Measurement period	Measurement method##	$PM_{2.5}$ max daily ($\mu g/m^3$)*	$PM_{2.5}$ maximum hourly ($\mu g/m^3$)	Duration of exposure (hr)###	Reference
Yarra Valley, VIC	April 2013	E-sampler aerosol monitor	56	222	9–15	
Manjimup, WA	October–December 2007	DustTrak	76	319	2–11	Reisen et al., 2011
Ovens, VIC	April–May 2007		151	377	16	
Sheffield, TAS	October 20–21, 2010	DustTrak	35	250	Several hours	BLANkET technical report 11#
George Town, TAS			26	80		
Carrick, TAS			23	150		
Lilydale, TAS	November 19, 2010	DustTrak	4	40	3	BLANkET technical report 12#.
Judbury, TAS	March 14, 2012	DustTrak	19	62	8	BLANkET technical report 20#
Huonville, TAS			12	48	5	
Geeveston, TAS			9	26	1	
Bryn Estyn, TAS	April 1–2, 2012	DustTrak	16	110	9	BLANkET technical report 21#
Hobart, TAS			11	40	4	
Gretna, TAS			9	70	1	
Hobart's Eastern shore regions, TAS	May 29, 2013 May 30, 2013	DustTrak	30–90 100–200	100 300	Several hours	BLANkET technical report 24#

Notes: #, BLANkET technical reports: http://epa.tas.gov.au/epa/blanket-report. ##, Continuous $PM_{2.5}$ data have been calibrated against gravimetric measurements. ###, Duration of exposure to smoke events when hourly concentrations of $PM_{2.5} > 25$ $\mu g/m^3$. *$PM_{2.5}$ maximum daily assumes a 24-hr average concentration. TAS: Tasmania; VIC: Victoria; WA: Western Australia.

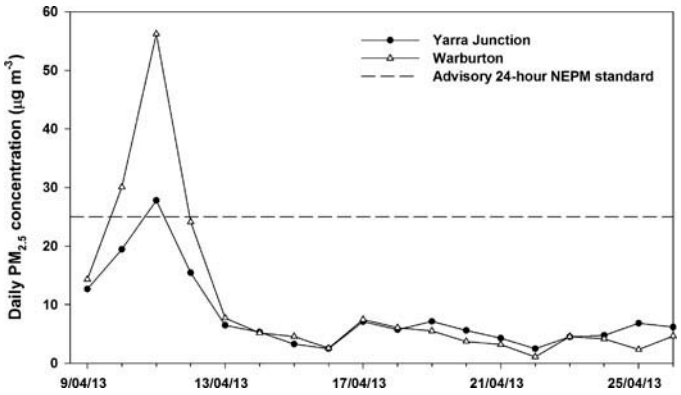

Figure 1. Yarra Valley (Warburton and Yarra Junction): daily concentrations of $PM_{2.5}$ during prescribed burning event (2013).

Monitor (E-sampler-9800, Met One Instruments, Inc., Oregon, USA) was used to measure concentrations of $PM_{2.5}$. Figures 1 and 2 give an example of $PM_{2.5}$ concentrations measured during a prescribed burning event in the Yarra Valley in April 2013.

During prescribed burning events, air quality may also be impacted by smoke from domestic wood heaters; however, the elevated $PM_{2.5}$ concentrations depicted in Table 1 were mostly a consequence of smoke from prescribed burning conducted in the region. This is because most of the prescribed burning was conducted during relatively warmer months (March/April) when there is limited use of wood heaters. Second, the remote/regional areas selected for prescribed burns were generally located away from other potential sources of $PM_{2.5}$ air pollutant (e.g., traffic emissions, industrial emissions). Moreover, concentrations of levoglucosan, a biomass burning marker, also correlated well with increase in $PM_{2.5}$ concentrations during prescribed burning events, indicating smoke to be the primary contributor to $PM_{2.5}$ levels. However, it should be noted that smoke from domestic wood heaters could also lead to increase in levoglucason levels.

The data in Table 1 show that exposure to smoke from prescribed burning was usually of short duration, less than a day. The duration of exposure was based on the number of hours that $PM_{2.5}$ levels were above 25 µg/m^3. On several occasions, exceedances of the Australian Advisory Air Quality 24-hr standard of 25 µg/m^3 $PM_{2.5}$ were observed. The data also show that prescribed burning smoke can result in very high short-term (hourly) peak exposures, up to 15 times higher than the daily advisory standards (Figure 2 and Table 1).

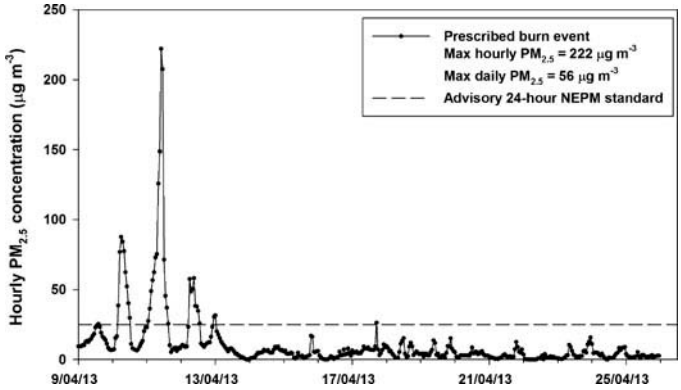

Figure 2. Yarra Valley (Warburton): hourly concentrations of $PM_{2.5}$ during prescribed burning event (2013).

Few overseas studies have also investigated the impact of prescribed burning smoke on air quality. For example, Tian et al. (2009) utilized air quality models to simulate the air quality impacts in Atlanta, GA (2002), and observed that prescribed burning was the largest source of $PM_{2.5}$ concentrations (50–80%). Another study by Zeng et al. (2008) used both model simulations and ground/satellite observation to investigate the impact of prescribed burning on air quality over the southeastern United States and showed daily and monthly mean enhancement of $PM_{2.5}$ levels of up to 8%. Hu et al. (2008) also used a forecasting system and modeling simulations to study smoke impacts from prescribed burning fires in Atlanta, GA (2007), and observed total daily (35 µg/m^3) and hourly (121 µg/m^3) simulated $PM_{2.5}$ concentrations.

Most studies have utilized different exposure assessment methods (e.g., air quality monitoring, model simulations) to measure $PM_{2.5}$ levels, and therefore the results are not comparable; however, the study findings indicate an increase in $PM_{2.5}$ concentrations during the prescribed burning period.

What Could be the Impact of Smoke From Prescribed Burning on Health?

Most research to date has focused on the health impacts of particulate matter exposure from wildfire smoke when it affects large population centers (Delfino et al., 2009; Morgan et al., 2010; Johnston et al., 2011; Henderson et al., 2012; Martin et al., 2013).

The most commonly investigated and established adverse health impact of $PM_{2.5}$ exposure from wildfire smoke exposure relates to pulmonary diseases (asthma, chronic obstructive pulmonary disease, infections) (Dennekamp and Abramson, 2011; Henderson and Johnston, 2012) and increase in clinical endpoints (hospital admissions, emergency department visits, increase in asthma symptoms and medication usage, decrease in pulmonary function) (Johnston et al., 2006; Delfino et al., 2009; Ignotti et al., 2010; Henderson et al., 2011; Do Carmo et al., 2013; Elliott et al., 2013; Martin et al., 2013). Evidence for adverse cardiovascular outcomes is also emerging, although the results have been null or inconclusive so far (Delfino et al., 2009; Johnston et al., 2007; Henderson et al., 2011; Rappold et al., 2011; Rappold et al., 2012; Martin et al., 2013; Youssouf et al., 2014; Liu et al., 2015). There is also strong evidence of the impact on nontraumatic mortality rates due to exposure to high concentrations of $PM_{2.5}$ during wildfires (Hanninen et al., 2009; Johnston et al., 2011; Kochi and Champ 2012; Youssouf et al., 2014; Liu et al., 2015). Most of the health impacts have been observed in vulnerable groups of people (especially the elderly and people with preexisting health conditions) (Delfino et al., 2009; Ignotti et al., 2010; Do Carmo et al., 2013; Rappold et al., 2011; Kochi and Champ, 2012; Rappold et al., 2012).

Indeed, the adverse health impacts due to PM related wildfire smoke exposure have been observed at comparatively low PM concentrations, well within current air quality standards (Chen et al., 2006; Johnston et al., 2006, Naeher et al., 2007; Morgan et al., 2010). Studies have also shown that slight increases of particulates from wildfire smoke were associated with increased incidence of hospital admissions for respiratory conditions especially asthma (Johnston et al., 2006; Chen et al., 2006). Given that prescribed fires cause more regular exposure to peak concentrations of particulate pollution, the impact on human health needs further investigation. Furthermore, due to the widespread nature of the smoke particles, numerous communities could be potentially impacted. This is especially important for at-risk groups of people exposed to smoke from prescribed burns on an annual basis.

Research Challenges

Given that wildfires are likely to increase in frequency and intensity in the context of warming climate (Bowman et al., 2009; Flannigan et al., 2009; Gill et al., 2013), prescribed burning is being used more frequently for wildfire suppression purposes (Bell and Adams, 2009; Teague et al., 2010; Penman et al., 2011). The increased $PM_{2.5}$ concentrations observed during prescribed burning events, their regular occurrence, and the likely adverse health impacts associated with these increases indicate the need for further research in this area. However, investigating the health impacts from exposure to smoke from prescribed burning presents a few challenges. Prescribed burning is conducted in rural/regional areas where the population size is small and sparsely located. This could significantly reduce the power of the study to detect any effect. Therefore, individual based studies are required to investigate the health impacts from prescribed burning smoke exposure. Conducting such studies is of logistic and financial concern, thereby limiting research in this area of need. The other challenges involved include:

- Very short window of opportunity present to investigate health parameters and conduct exposure assessment measurements. This is because the prescribed burning season is limited by the availability of suitable conditions, particularly dry fuel and stable weather patterns (light winds, low temperature, and moderate humidity) required to reduce fire intensity and rate of spread.
- Lack of easy accessibility to health care services (e.g., hospitals, health clinics, etc.) in regional areas could also impact on hospital service usage.
- Lack of exposure assessment due to limited air quality monitors in regional areas targeted for prescribed burning. The use of portable monitors can be expensive and data analysis can be time-consuming. However, the increased use of remote-sensing tools and satellite data and the increased development of low-cost particle sensors will assist in providing air quality data in areas with lack of monitoring facilities (Yao et al., 2013; Yao and Henderson, 2014).

Conclusion

Currently, smoke dispersion from prescribed burning is not required to be monitored and there is no known safe level of pollutant exposure below which adverse health impacts are not observed (Naeher et al., 2007; Craig et al., 2008). There is a need for the development of innovative methods for better prediction and effective exposure assessment in regional areas with a lack of air quality monitors. Air quality models are required to provide for accurate deterministic concentrations and predict spatial and temporal distribution of air pollution and smoke from prescribed burning. This information will be useful for communities living in and around the vicinity of the prescribed burns for advance warning, especially for at risk people with preexisting health conditions. Land managers would also benefit from such information to better manage the impacts of air pollution during prescribed burning. The challenge is for the land managers and scientists to work collaboratively to successfully integrate evidence-based knowledge, and experience contributing toward an adaptive management strategy. A recent study by Rappold et al. (2014) projected the mitigation of health impact of wildfire exposure based on forecasting the smoke plume and associated public health messaging that in theory would change behavior of the exposed population and limit exposure dose.

Prescribed burning is a valuable tool for managing fuels and in ultimately reducing the severity of high-intensity wildfires. As burns need to be conducted relatively close to communities to be effective (Gibbons et al., 2012), prescribed fires can be major contributors to local air pollution, despite being on much smaller scale than wildfires. Given the known adverse health impacts from wildfire smoke-sourced $PM_{2.5}$ exposure, prescribed burning smoke exposure is of public health concern. However, more research is required to quantify and determine health impacts, identify high-risk individuals, and derive evidence-based interventions for managing the risk.

Funding

This research was funded by Bushfire & Natural Hazard Cooperative Research Centre, and the Yarra Valley study conducted in Victoria was also funded by Department of Environment and Primary Industries, Melbourne, Victoria, Australia.

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ORIGINAL RESEARCH

Impact of Fine Particulate Matter ($PM_{2.5}$) Exposure During Wildfires on Cardiovascular Health Outcomes

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***Background*—**Epidemiological studies investigating the role of fine particulate matter ($PM_{2.5}$; aerodynamic diameter <2.5 μm) in triggering acute coronary events, including out-of-hospital cardiac arrests and ischemic heart disease (IHD), during wildfires have been inconclusive.

***Methods and Results*—**We examined the associations of out-of-hospital cardiac arrests, IHD, acute myocardial infarction, and angina (hospital admissions and emergency department attendance) with $PM_{2.5}$ concentrations during the 2006–2007 wildfires in Victoria, Australia, using a time-stratified case-crossover study design. Health data were obtained from comprehensive health-based administrative registries for the study period (December 2006 to January 2007). Modeled and validated air exposure data from wildfire smoke emissions (daily average $PM_{2.5}$, temperature, relative humidity) were also estimated for this period. There were 457 out-of-hospital cardiac arrests, 2106 emergency department visits, and 3274 hospital admissions for IHD. After adjusting for temperature and relative humidity, an increase in interquartile range of 9.04 $\mu g/m^3$ in $PM_{2.5}$ over 2 days moving average (lag 0-1) was associated with a 6.98% (95% CI 1.03% to 13.29%) increase in risk of out-of-hospital cardiac arrests, with strong association shown by men (9.05%,95%CI 1.63% to 17.02%) and by older adults (aged ≥65 years) (7.25%, 95% CI 0.24% to 14.75%). Increase in risk was (2.07%, 95% CI 0.09% to 4.09%) for IHD-related emergency department attendance and (1.86%, 95% CI: 0.35% to 3.4%) for IHD-related hospital admissions at lag 2 days, with strong associations shown by women (3.21%, 95% CI 0.81% to 5.67%) and by older adults (2.41%, 95% CI 0.82% to 5.67%).

***Conclusion*—**$PM_{2.5}$ exposure was associated with increased risk of out-of-hospital cardiac arrests and IHD during the 2006–2007 wildfires in Victoria. This evidence indicates that $PM_{2.5}$ may act as a triggering factor for acute coronary events during wildfire episodes. **(*J Am Heart Assoc*. 2015;4:e001653 doi: 10.1161/JAHA.114.001653)**

Key Words: coronary disease • heart arrest • ischemic heart disease • particulate matter • wildfires

Cardiovascular disease (CVD) continues to impose a heavy burden worldwide in terms of illness, disability, and premature death.[1] The most common form of cardiovascular disease is coronary heart disease (CHD), also known as ischemic heart disease (IHD). Acute myocardial infarction (AMI) and cardiac arrest are frequent manifestations of CHD. Epidemiological studies have investigated the role of fine particulate matter air pollutant ($PM_{2.5}$ median aerodynamic diameter <2.5 μm) in triggering acute IHD events, including cardiac arrest[2–19]; however the findings from these studies have been inconclusive. The updated American Heart Association scientific statement specifically characterized $PM_{2.5}$ exposure as a modifiable factor that contributes to CVD mortality and morbidity.[20] A meta-analysis[21] also concluded that fine particulate matter is a risk factor for acute cardiac events.

One of the most important sources of $PM_{2.5}$ air pollution is from wildfire (bushfire) smoke exposure.[22–24] Smoke from wildfires disperses widely and affects large portions of the population away from the fire source.[25] $PM_{2.5}$ levels are significantly elevated during wildfire episodes[26,27] and can exceed levels set by regulatory bodies (World Health Organization air quality guidelines[28] for $PM_{2.5}$:10 $\mu g/m^3$ annual

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Received December 17, 2014; accepted May 20, 2015.

mean, 25 μg/m^3 daily mean). It has been hypothesized for a long time that $PM_{2.5}$ is particularly toxic because of its capacity to penetrate deep into the lungs and to induce systemic inflammatory and oxidative stress responses.[4,20,29–31] This effect could potentially trigger a cascade of pathophysiological events in the body and lead to a variety of manifestations of CHD, including chronic atherosclerosis and angina, plaque instability, and rupture, typically following myocardial infarction and cardiac arrest.[4,20,32–37]

The objective of this study was to investigate the association between $PM_{2.5}$ exposure from wildfire smoke and cardiovascular health outcomes. In particular, we aimed to investigate the effects on out-of-hospital cardiac arrests, hospital admissions, and emergency department (ED) visits for cardiovascular end points (IHD, AMI, angina) during the 2006–2007 wildfire episode in Victoria, Australia. We used modeled air exposure data to estimate wildfire-related $PM_{2.5}$ levels. Modeling enabled wider coverage of affected areas, especially rural or regional areas that had no air-quality monitors. There is very limited understanding of the health impacts of wildfire smoke exposure on communities in such areas.

Methods

Health and Population Data

We received deidentified information on the health outcomes from 3 large administrative data sets in Victoria. Out-of-hospital cardiac arrests were identified from the Victorian Ambulance Cardiac Arrest Registry (VACAR), which captures data on all cardiac arrest patients attended by ambulance personnel (prehospital setting) in Victoria. VACAR is one of the largest prehospital cardiac arrest registries in the world.[38] The registry is based on internationally recognized data variables and definitions.[39] Hospital admissions (unscheduled visits) and ED visits for cardiovascular end points were obtained from the Victorian Admitted Episodes Dataset (VAED) and the Victorian Emergency Minimum Dataset (VEMD), respectively. Both VAED and VEMD are comprehensive administrative data sets maintained by the health department of Victoria documenting deidentified demographic and clinical information on ED visits and hospital admissions from hospitals in Victoria. Deidentified information included event date, home postcode, age, sex, event location (eg, house, work, street), principal diagnosis according to the International Classification of Diseases, version 10 (ICD-10) codes for CVD conditions (ICD-10AM, I00 to I99), and a unique event identifier.

Victoria is a second most populous state in southeast Australia. According to the Australian Bureau of Statistics, the estimated resident population of Victoria in 2006 was 5.1 million. Approximately 3.6 million people resided in metropolitan Melbourne, and 1.5 million resided in rural or regional areas. We included people from both metropolitan and rural or regional areas of Victoria (those whose residential postcodes fell within the boundary of the state of Victoria). We included only people aged ≥35 years because adverse cardiovascular events such as cardiac arrest in younger people are mainly due to nonischemic causes (eg, structural abnormalities such as cardiomyopathy, congenital heart defects, inherited rhythm disorders [eg, long QT syndrome]).[40]

Study Period

The study period included 2 months of intense wildfires in Victoria (December 1, 2006, to January 31, 2007). Landscape, climate (mild moist winters followed by hot dry summers), vegetation (dry eucalypt forests, vast grasslands), and protracted droughts make Victoria one of the most fire-prone regions in the world. The 2006–2007 wildfire was the longest running collection of fires that burned ≈1 million hectares of land in Victoria and lasted for >60 days. This wildfire event was characterized by a few highly active fire days interspersed with days of low fire activity. The air quality during this period was substantially diminished, with elevated surface concentrations of $PM_{2.5}$ found for most of the wildfire event. The maximum daily (24-hour) concentration of $PM_{2.5}$ measured during the wildfire event was ≈100 μg/m^3. This concentration greatly exceeded the allowable air-quality standards for $PM_{2.5}$ set by regulatory bodies worldwide (in Australia, the advisory standard is 25 μg/m^3 for a 24-hour period; in the United States, it is 35 μg/m^3 for a 24-hour period).

The long duration of these wildfires led to widespread smoke dispersion over a wide geographic area. A large population was exposed to smoke from these fires for a long period of time. This provided us with a unique opportunity to investigate acute health impacts in the community.

Air Pollution Data

Smoke dispersion modeling for the 2006–2007 Victorian wildfire was undertaken using the atmospheric dispersion model called The Air Pollution Model (TAPM) coupled with a chemical transport model.[41] The modeling technique was developed by scientists from the Commonwealth Scientific and Industrial Research Organization (CSIRO) Marine and Atmospheric Research organization located in Victoria. The chemical transport model is a 3-dimensional model with the capability of modeling the emission, transport, chemical transformation, and deposition of gaseous and aerosol species. It predicts regional air quality from a defined inventory of pollutant sources.

For meteorology, the chemical transport model used the synoptic weather model predictions for Australia provided by

the Bureau of Meteorology and downscaled by TAPM to the region of interest. Dynamic downscaling generates high-resolution meteorological fields within a regional area from lower resolution continental or global data from numerical weather models. Downscaling reliably predicted the meteorological parameters at the required spatial resolution (9×9 km) for this study. Of the many meteorological variables, temperature and humidity were among those most accurately predicted. The accuracy of the air pollution model for downscaling temperature and humidity has been presented in numerous papers and reports.[41–43]

For this study, the TAPM–chemical transport model was configured with a complete emissions inventory that included industrial, domestic, transportation, and natural sources of aerosol in addition to smoke from the wildfire event. Of these sources, however, only wildfires contributed significant amounts of $PM_{2.5}$ above the natural background concentration (5 to 10 $\mu g/m^3$). Mean daily surface $PM_{2.5}$ concentrations were computed for each 9×9-km cell in an 80×80 grid domain centered on Melbourne. This covered all Victoria except for small areas at the extreme eastern and western boundaries. Model validation, accuracy, and spatial variation have been tested extensively and discussed in detail elsewhere.[44–46] Model accuracy was confirmed by comparing the modeled $PM_{2.5}$ values with observed $PM_{2.5}$ at all stations of the Victorian Environmental Protection Authority pollution-monitoring network during the wildfire event.[44,46] The correlation coefficient between daily predicted $PM_{2.5}$ and daily observed $PM_{2.5}$ concentrations was >0.5.

The daily observed $PM_{2.5}$ data were obtained either directly from TEOMs (tapered element oscillating microbalance; Thermo Fisher Scientific) located at the monitoring stations or from surrogate aerosol observations. For monitoring stations that were not equipped with $PM_{2.5}$ TEOMS, the backscattering coefficient was obtained from nephelometers located at each station. For this study, a very high correlation was found between $PM_{2.5}$ and backscattering coefficient (r^2=0.966).This enabled $PM_{2.5}$ to be estimated with a high level of confidence when monitoring stations were not equipped with $PM_{2.5}$ TEOMS.

Comprehensive observations of smoke composition (including aerosol chemical composition, plume aging, and secondary aerosol formation) were also made at the CSIRO air-quality monitoring station in Melbourne during the wildfire event. Size, characterization, and chemical composition of smoke aerosol are also detailed in a report by Meyer et al.[47] Both the aerosol composition and the detailed trajectory analysis confirmed that the only significant source of $PM_{2.5}$ during the pollution events was the wildfires. The smoke aerosol was composed of organic content, inorganic carbon content, mineral ion content, and anhydrous sugar levoglucosan. Levoglucosan is a unique tracer for wood burning and is produced in high concentrations in biomass smoke.[22] $PM_{2.5}$ contribution from other nonwildfire sources (eg, industry, transportation, sea salt, windblown dust) during this wildfire period was negligible. The smoke composition during biomass combustion was relatively consistent and stable and did not change significantly over a 24-hour time period.[47]

Modeled daily average temperature, relative humidity, and ground-level $PM_{2.5}$ for the study period were matched by postcodes.

Statistical Analysis

A time-stratified case-crossover study design[48–50] was applied to investigate the association between wildfire-related $PM_{2.5}$ air pollutant and cardiovascular health outcomes using conditional logistic regression models. In this study design, the $PM_{2.5}$ exposure on the day of the health event (eg, out-of-hospital cardiac arrest; case day) was compared with $PM_{2.5}$ exposure on several nonevent days (referent; control days). The referent exposure days were selected by time-stratified sampling and were matched for day of the week, month, and year of the health event (eg, exposure on the day of an event on Monday in January was compared with exposures on all other Mondays in January). This resulted in 3 or 4 control periods for each case period. Because the matching referent periods were close in time and on the same day of the week as the event day, this study design automatically controlled for time-dependent risk factors such as day of the week and monthly, seasonal, and long-term trends.[51–53] Moreover, because the persons who experienced the health event also served as their own controls, time-independent factors such as age, sex, smoking, socioeconomic status, preexisting health conditions, and other individual risk factors were controlled for with this approach.[48,52,54]

The primary outcome measures in our study were CVD end points: out-of-hospital cardiac arrests, hospital admissions, and ED visits for IHD (ICD 10-AM, I20 to I25, I46, I49), AMI (I21), and angina (I20). The primary exposure measure was modeled wildfire-derived $PM_{2.5}$ (daily average) concentrations. We also included daily average temperature and relative humidity as confounding variables. We conducted a subgroup analysis by age group (35 to 64 years, ≥65 years) and by sex. Various lag periods were also investigated: lag 0 (exposure concentrations on the day of event), lag 1 (exposure concentrations 1 day before the event), lag 2 (exposure concentrations 2 days before the event), and 2-day lagged moving average (lag 0 to 1: averages of exposure concentrations on the day of the event and exposure concentrations 1 day before the event). The analysis of different lag periods was performed individually. The models were adjusted for lag 0 to lag 2 of temperature and relative humidity.

The overall results are presented as an increase in interquartile range for daily average $PM_{2.5}$ concentrations and the associated percentage change in the risk of CVD end

points after controlling for temperature and relative humidity at various lag periods.

The data were analyzed using the statistical package Stata (version 12.1; StataCorp). *P* values <0.05 were considered statistically significant.

Ethics

The study was approved by Monash University human research ethics committee.

Results

The descriptive characteristics for out-of-hospital cardiac arrests and IHD events (ED visits and hospital admissions) are shown in Table 1. Of the 457 cases of out-of-hospital cardiac arrests during the study period, the largest percentage was for patients aged ≥65 years (67%), and most were male (67%) rather than female (33%). Of the 2106 cases of ED attendance for IHD, the largest percentage was for patients aged ≥65 years (62%), and most were male (63%) rather than female (37%). Of the 3274 cases of hospital admissions for IHD, the largest percentage was for those aged ≥65 years (64%), and most were male (64%) rather than female (36%). The ED attendance data also indicated 788 cases of AMI and 1131 cases of angina, and hospital admissions data indicated 1554 cases of AMI and 1534 cases of angina. Table 2 provides an overview of modeled daily average $PM_{2.5}$ concentration, temperature, and relative humidity during the study period.

Table 1. Descriptive Characteristics of Out-of-Hospital Cardiac Arrests and IHD Events in Victoria, Australia (December 1, 2006, to January 31, 2007)

Number of out-of-hospital cardiac arrests	457
Sex	
Male	304 (67)
Female	153 (33)
Age, y	
35 to 64	152 (33)
≥65	305 (67)
IHD	
ED visits	2106
Sex	
Male	1320 (63)
Female	786 (37)
Age, y	
35 to 64	808 (38)
≥65	1298 (62)
Hospital admissions	3274
Sex	
Male	2068 (64)
Female	1206 (36)
Age, y	
35 to 64	1180 (36)
≥65	2094 (64)

Values are number (percentage). ED indicates emergency department; IHD, ischemic heart disease.

Table 2. Descriptive Summary of Daily Average Air Exposure Data (Modeled, December 2006 to January 2007)

			Percentile			
Variable	Mean	Maximum	25%	50%	75%	IQR
$PM_{2.5}$, $\mu g/m^3$	15.43	163.44	6.08	7.35	15.12	9.04
Temperature, °C	18.66	32.03	15.62	17.86	22.06	6.44
RH, %	59.88	87.20	51.46	61.38	68.90	17.44

IQR indicates interquartile range (values are calculated for a change in $PM_{2.5}$ levels from the 25th to the 75th percentile); $PM_{2.5}$, particulate matter; RH, relative humidity.

Out-of-Hospital Cardiac Arrests

An interquartile range increase in $PM_{2.5}$ of 9 $\mu g/m^3$ over the 2-day moving average (lag 0 to 1) was associated with an increase in risk for out-of-hospital cardiac arrests of 6.98% (95% CI 1.03% to 13.29%) after adjusting for temperature and relative humidity. An association of 4.55% (95% CI 0.54% to 8.72%) was also observed on the day of the exposure (lag 0), although this was not as strong as that observed over the 2-day exposure period. None of the other lag periods showed any association (Table 3).

Subgroup analysis by age and sex

When we conducted the subgroup analysis by age and sex, we found those aged ≥65 years to be at higher risk by 7.25% (95% CI 0.24% to 14.75%; *P*=0.04) compared with younger participants (aged 35 to 64 years: 5.8%; 95% CI −5.04% to 17.89%; *P*=0.30) and men to be at higher risk by 9.05% (95% CI 1.63% to 17.02%; *P*=0.01) compared with women (3.19%; 95% CI −6.4% to 13.84%; *P*=0.53) (Figure 1).

IHD

Hospital admissions

After adjusting for temperature and relative humidity, an interquartile range increase in $PM_{2.5}$ of 9 $\mu g/m^3$ was associated with an increase in risk of hospital admissions for IHD by 1.86% (95% CI 0.35% to 3.44%) and for AMI by

Table 3. Percentage Change % (95% CI) in Risk of Out-of-Hospital Cardiac Arrests, Hospital Admissions, and ED Visits for IHD, AMI, and Angina for an IQR Increase in $PM_{2.5}$ by 9 $\mu g/m^3$ at Various Lag Days

Health Outcome	Lag 0	Lag 1	Lag 2	Lag 0 to 1
Out-of-hospital cardiac arrests	4.55 (0.54 to 8.72)	2.85 (−0.46 to 6.28)	2.88 (−0.22 to 6.09)	6.98 (1.03 to 13.29)
Hospital admission				
IHD	−1.12 (−2.67 to 0.45)	0.69 (−0.78 to 2.20)	1.86 (0.35 to 3.4)	−0.96 (−3.30 to 1.43)
AMI	−1.50 (−3.80 to 0.85)	0.41 (−1.7 to 2.6)	2.34 (0.06 to 4.67)	−1.71 (−5.15 to 1.84)
Angina	−0.93 (−3.15 to 1.30)	0.56 (−1.55 to 2.66)	0.90 (−1.22 to 2.48)	−0.72 (−4.09 to 2.10)
ED visits				
IHD	−2.10 (−4.03 to −0.12)	1.63 (−0.39 to 3.71)	2.07 (0.09 to 4.09)	−0.98 (−3.96 to 2.08)
AMI	−3.86 (−6.90 to −3.61)	2.34 (−0.75 to 5.53)	0.75 (−2.44 to 4.06)	−1.86 (−6.65 to 3.11)
Angina	−2.44 (−5.08 to 0.27)	1.54 (−1.11 to 4.26)	1.71 (−0.74 to 4.23)	−1.64 (−5.74 to 2.64)

Statistics reflect the adjustment for temperature and relative humidity. AMI indicates acute myocardial infarction; ED, emergency department; IHD, ischemic heart disease; $PM_{2.5}$, fine particulate matter.

2.34% (95% CI 0.06% to 4.67%) at lag 2. No significant association was found for presentation with angina at lag 2. No other lag periods showed any association (Table 3). Although we presented our results for lag periods (lag 0, 1, 2, 0 to 1), we extended our analysis for hospital admissions to include lag periods of up to 5 days but did not find any significant effect (data not shown).

ED visits

After adjusting for temperature and relative humidity, an interquartile range increase in $PM_{2.5}$ of 9 $\mu g/m^3$ was associated with an increase in risk of ED attendance for IHD by 2.07% (95% CI 0.09% to 4.09%) at lag 2. No association was found for either AMI or presentation with angina at lag 2. No other lag periods showed any association (Table 3).

Subgroup analysis by age and sex. When we conducted the subgroup analysis by age and sex, we found a higher risk of IHD-related hospital admissions for those aged ≥65 years by 2.41% (95% CI 0.54% to 4.31%; *P*=0.01) compared with younger participants (aged 35 to 64 years: 0.26%; 95% CI −2.37% to 2.98%; *P*=0.84) and for women by 3.21% (95% CI 0.82% to 5.67%; *P*=0.02) compared with men (0.99%; 95% CI −0.94% to 2.9%; *P*=0.33) (Figure 2). No association by age and sex was found for ED attendance.

Discussion

This study found a positive association between $PM_{2.5}$ air-pollutant exposure and acute CHD events during the

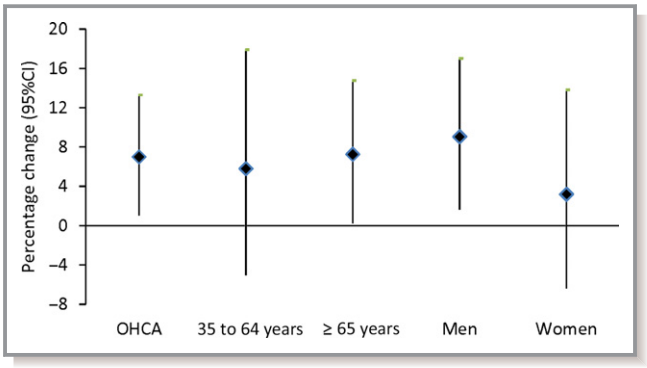

Figure 1. Percentage increase in risk (and 95% CI) of out-of-hospital cardiac arrests by age and by sex for interquartile range increase in $PM_{2.5}$ by 9 $\mu g/m^3$ after adjusting for temperature and relative humidity for lag 0 to 1 (lag 0 to 1: averages of exposure concentrations on the day of the event and exposure concentrations 1 day before the event). OHCA indicates out-of-hospital cardiac arrests; $PM_{2.5}$, fine particulate matter.

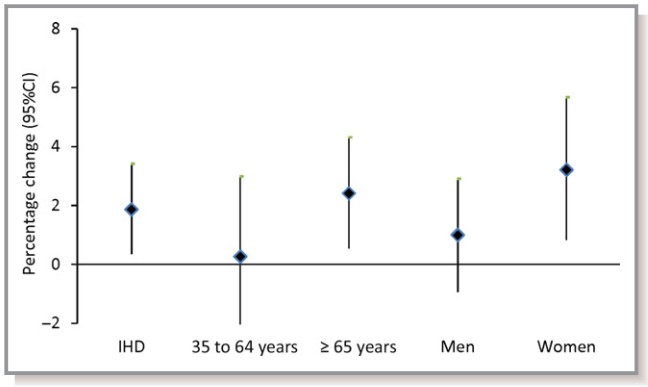

Figure 2. Percentage increase in risk (and 95% CI) of hospital admission for IHD (by age and by sex) for an interquartile range increase in $PM_{2.5}$ by 9 $\mu g/m^3$ after adjusting for temperature and relative humidity at lag 2 (exposure concentrations 2 days before the event). IHD indicates ischemic heart disease; $PM_{2.5}$, fine particulate matter.

2006–2007 wildfire period in Victoria. Specifically, associations were observed for out-of-hospital cardiac arrests, hospital admissions, and ED visits for IHD after 2 days of exposure to wildfire smoke at $PM_{2.5}$ concentrations. This association was observed mainly in older adults (aged ≥65 years), with men showing a higher risk of out-of-hospital cardiac arrests events and women showing a higher risk of IHD-related hospital admissions.

Sustained effects of wildfire smoke exposure and cumulative biological effects could be responsible for a delayed effect of $PM_{2.5}$ exposure on acute CHD events.[55] From a mechanistic point of view, exposure to $PM_{2.5}$ from wildfire smoke 2 days before the events may amplify the pathobiological processes in the body (induce an inflammatory cascade) and lead to ischemic events, plaque rupture, and development of arrhythmias. Multiple pathways (possibly interlinked) have been postulated[4,17,20,34–36,56,57] by which $PM_{2.5}$ could instigate adverse cardiac events including (1) induction of systemic pulmonary inflammation and oxidative stress, leading to increased levels of inflammatory markers (eg, C-reactive protein, prothrombotic and inflammatory cytokines); (2) direct translocation into blood, leading to increase in blood viscosity, thrombus formation, plaque erosion, and rupture and acceleration of the atherosclerotic process; and (3) dysregulation of the cardiac autonomic system (increase in heart rate and decrease in heart rate variability), leading to arrhythmias and cardiac arrests. Some of these potential mechanisms, however, must be seen as somewhat speculative.

A convincing explanation of how wildfire-related $PM_{2.5}$ air pollutants might induce different biological responses to non–wildfire-related $PM_{2.5}$ remains an area of research. It is conceivable that the difference in the magnitude of the inflammatory response could occur because of variation in the duration and intensity of exposure to particulate matter.[20] Unlike $PM_{2.5}$ derived from urban pollution, wildfire-related $PM_{2.5}$ concentrations can become extreme with variations in the duration of exposure to smoke events.[25,58] The difference in biological response could also be due to the variation in the chemical composition of particulate matter. Some studies have suggested that the chemical components present in $PM_{2.5}$ pollutant (eg, transition metals) from urban pollution can catalyze an oxidative stress reaction in the lungs, leading to inflammatory lung injury and arrhythmias.[5,59–61] In contrast, a study by Weggser et al[30] (using mouse bioassay) showed that $PM_{2.5}$ from wildfires was particularly toxic to the lungs, especially to the alveolar macrophages, compared with $PM_{2.5}$ exposure from urban pollution. This finding requires further understanding.

Other less known and studied possibilities for the delayed impact of wildfire $PM_{2.5}$ exposure could be due to behavioral, cultural, social, and environmental conditions that determine a person's use of health services during such extreme events.[62] Similar to respiratory health points, the CVD health points at various lags may be influenced by individual perceptions and decisions to seek medical care during wildfire episodes.[63] People may delay deciding to go to the hospital, for example, until symptoms become too severe during the wildfire event.[55] This important possibility needs further exploration.

Only a limited number of studies to date have investigated the effect of fine particulate matter exposure on out-of-hospital cardiac arrests. These studies[6,16–19] reported an increase in the risk of out-of-hospital cardiac arrests by 4% to 10% for an increase in $PM_{2.5}$ concentrations by 5 to 10 $\mu g/m^3$, and this is consistent with our findings. Nonetheless, it is noted that some studies have not shown an increase in implantable cardioverter-defibrillator discharges, which typically reflect occurrences of life-threatening ventricular arrhythmias at times of significant air pollution.[64–66]

Only a few epidemiological studies have investigated cardiovascular health impact and exposure to wildfire smoke-related particulate matter.[55,58,63,67–72] Most studies investigated the impact on IHD-related hospital admissions and found null or inconsistent results.[55,58,63,67–70] In contrast, numerous studies have investigated the association between cardiovascular health impact and exposure to particulate matter pollutants from urban sources (eg, vehicular emissions).[20] Studies have reported a 2% to 20% increase in risk of acute IHD-related morbidity for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ levels.[3,4,7,11,31,73]

Some challenges are involved in investigating the health impacts of wildfire smoke exposure. Wildfires are episodic and short-lived events; therefore, the brief periods of $PM_{2.5}$ exposure may not be enough to detect all but the most sensitive health outcomes.[74] Moreover, many wildfire smoke–affected areas (especially rural or regional areas) do not have routine air-quality monitoring programs. Consequently, exposure assessment is challenging in these areas.[75] Our study is unique in 3 respects: (1) use of spatially resolved $PM_{2.5}$ air pollutant data obtained from a novel air-quality modeling technique, (2) wildfire $PM_{2.5}$ estimates derived from areas with lack of monitoring facilities (eg, rural or regional areas outside a major city), and (3) extensive and long duration of a wildfire event with widespread $PM_{2.5}$ particulate dispersion affecting a large population.

Given that the burden of CHD remains high globally (CHD accounts for 64% of all CVD deaths) and is predicted to remain so for the next 20 years,[76] further research is required to understand the role of wildfire-related $PM_{2.5}$ as a triggering factor for acute CHD events. Our findings contribute to this important area of research.

We found people aged ≥65 years were at higher risk for cardiac events due to $PM_{2.5}$ exposure, similar to the findings

observed in other studies.[6,16,55,63,67,70] Given that older adults may already suffer from multiple comorbidities including atherosclerosis (often asymptomatic), exposure to $PM_{2.5}$ may attenuate any underlying IHD, thereby triggering potentially fatal coronary events.[2]

Similar to our findings, 2 studies showed that men had a higher risk of out-of-hospital cardiac arrests due to PM exposure.[16,19] The reason for different outcomes between men and women is still unclear. It has been suggested that women suffer cardiac arrests half as often as men of the same age.[77] Pathobiological processes other than atherosclerosis (eg, coronary vasospasm, valvular heart disease) may also be more common in women.[78] Limited studies have shown women to be at increased risk of CHD morbidity due to PM exposure.[79–82] This is consistent with our findings, in which we observed women to be at increased risk of IHD. Although the evidence is sparse, it has been hypothesized that hormonal alteration, increase in inflammatory biomarkers (eg, C-reactive protein), poor coronary circulation due to microvascular coronary dysfunction (more plaque erosion and distal embolization, small arterial size), and endothelial dysfunction in women may be factors contributing to female-specific ischemic disease.[83] Concentrations of the inflammatory biomarker C-reactive protein are also known to increase due to PM exposure.[20,84,85] This could explain the increase in risk of IHD in women during wildfire episodes, when $PM_{2.5}$ levels are significantly high. Difference in acute cardiac events between men and women during wildfire smoke exposure is an area needing further research.

The main strength of our study was the use of novel modeling techniques to estimate air exposure data during the wildfire period. Modeled data had an advantage in that they provided fine temporal and spatial resolution and wider coverage of areas with no monitoring facilities, especially rural or regional Victoria. This is in contrast to studies using monitored data, in which exposure information is obtained only from limited areas (in and around the monitors).This means that the monitored data might not adequately represent the smoke impact in areas that lack monitoring facilities (rural or regional areas), thereby limiting the ability to detect associations that might be present.[86]

Another important strength of this study was that the majority of the $PM_{2.5}$ pollutant included in the air pollution model was derived from wildfires. This allowed us to directly analyze the contribution of smoke-sourced $PM_{2.5}$ levels to increased risk of CHD events. The wide spread and long duration of the 2006–2007 wildfire event provided us with an opportunity to assess the health impacts of protracted exposure to uncontaminated biomass combustion. In Australia, air-quality exceedance in rural areas is associated mostly with biomass combustion from either domestic wood burning or wildfires.[27,47,87] In these areas, most pollution events are biomass combustion aerosol largely uncontaminated from other pollutant types. Moreover, towns are widely separated, and for the most part, the air quality is determined by local pollutant sources (particularly domestic wood heaters); advection of pollutant plumes from other towns or cities is rare. The exception is smoke plumes from wildfires. Consequently, Australian cities and especially regional areas provide a rare test bed for investigating the impacts of biomass combustion aerosol uncontaminated by other anthropogenic sources of $PM_{2.5}$.

We used comprehensive statewide health data sets to obtain information relevant for the purposes of the study. Access to health information from such large registries further reduced the risk of selection bias and strengthened case ascertainment.

A limitation of our study was the lack of information on personal risk factors such as socioeconomic status, smoking, obesity, and underlying health conditions; however, the case-crossover study design controls for confounding factors because the participants serve as their own controls.[48] We also lacked data on indoor $PM_{2.5}$ concentrations during wildfire episodes. Research has shown that during major wildfires, the impact of outdoor air quality on indoor air quality can be severe.[27] Although we did not adjust for the coarser fraction of particulate matter air pollutant (PM_{10}) in the study, the bulk of the PM_{10} emitted during wildfires is $PM_{2.5}$.[22,88] On average, 87% of PM_{10} due to wildfires consists of $PM_{2.5}$.[89] A strong positive correlation has been observed between wildfire PM_{10} and $PM_{2.5}$ (r=0.9).[47] We also performed many tests that could have resulted in increased probability of obtaining spurious associations. We acknowledge that we were unable to account for repeated health events because we obtained only deidentified health data.

Robust evidence-based research is required to fill the knowledge gaps that currently exist in this important area of public health. Novel air exposure–modeling techniques to improve forecasting, effective spatial coverage, and health impact assessment of at-risk groups (eg, women, children, and older adults) are areas of significant need. Moreover, health impacts of wildfire smoke exposure in rural or regional communities remain largely unknown and urgently require understanding. Pathophysiology and pathways that trigger acute cardiac events due to $PM_{2.5}$ exposure (short and long term) remains speculative and need further evidence. Importantly, the variation in $PM_{2.5}$ exposure (duration and intensity) from wildfire episodes and from urban air pollution on cardiovascular health end points requires understanding. Detailed analysis of chemical composition of wildfire particulates is also needed to understand toxicity of source-specific fractions of $PM_{2.5}$ on clinical outcomes. Information is currently insufficient to determine a safe $PM_{2.5}$ exposure threshold during wildfire episodes below which there are no adverse

health impacts. Improvement in the understanding of these priority areas is needed so that effective and timely public health strategies can be developed and implemented to reduce the burden of disease during wildfire events. This will have further implications for setting appropriate air-quality standards, enhancing health care infrastructure, and improving timely risk communication and health advice during wildfires.

The results from our study suggest that $PM_{2.5}$ exposure from wildfire smoke may be an important determinant of out-of-hospital cardiac arrest and IHD (ED visits and hospital admissions) and that susceptible persons such as older adults may be at higher risk during such extreme events. Given the increased incidence and frequency of wildfires recently and the increased number of people at risk of smoke exposure, future research is required to investigate the role of fine particulate matter exposure from wildfire smoke in triggering acute coronary events. The knowledge and evidence resulting from such research will inform policy and practice and help build capacity in the understanding and management of adverse cardiovascular health impacts in vulnerable communities during wildfire episodes.

Sources of Funding

This research was funded by Bushfire Cooperative Research Centre (Bushfire CRC) now a part of Bushfire & Natural Hazards Cooperative Research Centre, Melbourne, Victoria, Australia.

Disclosures

None.

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Measures of forest fire smoke exposure and their associations with respiratory health outcomes

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Purpose of review
Exposure to forest fire smoke is episodic, which makes its health effects challenging to study. We review the newest contributions to a growing literature on acute respiratory outcomes.

Recent findings
Smoke exposure was associated with increases in self-reported symptoms, medication use, outpatient physician visits, emergency room visits, hospital admissions, and mortality. The associations were strongest for the outcomes most specific to asthma.

Summary
Studies with varied approaches to exposure assessment and varied measures of respiratory outcomes were consistent among themselves, and consistent with most previous work.

Keywords
asthma, biomass, forest fire smoke, respiratory

INTRODUCTION

Forest fires occur throughout most of the world, but their frequency, intensity, and size vary according to complex ecologic and human factors [1]. Forest fires also have a complex relationship with the global climate, whereby smoke may contribute to atmospheric warming [2] and cooling [3], but overall warming may increase the fire risk [4,5]. Indeed, there is growing concern about the incidence of so-called mega-fires that resist conventional suppression, transform vast landscapes, and threaten human populations. Such fires also produce large smoke plumes that can affect air quality at local, regional, and global scales [6].

The health effects of forest fire smoke are challenging to assess because large fires are typically sporadic and smoke episodes are typically short-lived. Given that the public health impacts are small, smoke rarely affects populations large enough to support the detection of statistically significant associations. Even so, a growing body of literature indicates that smoke exposure is associated with acute respiratory outcomes ranging from increased reporting of symptoms through to increased risk of mortality. A recent review of the evidence through 2010 [7▪▪] provides an excellent background for this compendium of the newest methods and results. Our review is also limited to studies reporting the respiratory health effects of forest fire smoke in the general population, and it does not cover related work on occupational exposures, periodic agricultural burning, or domestic solid fuel use.

Any epidemiologic study on the health effects of forest fire smoke has two key components: a clear definition of exposure within the population and a clear definition of the measured health outcomes. Here, we summarize eight recent studies (Table 1) [8▪,9▪,10▪▪,11▪▪,12▪,13▪,14▪▪,15▪], classifying the exposure assessment approaches used by each as 'simple' or 'complex' and classifying the health outcomes evaluated as 'mild' or 'severe'. We then generate a matrix of assessment methods and outcome severities to highlight the internal consistency of the results.

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Curr Opin Allergy Clin Immunol 2012, 12:221–227
DOI:10.1097/ACI.0b013e328353351f

KEY POINTS

- Smoke exposure assessment is challenging, but fire databases and remote sensing are facilitating innovation in both simple and complex approaches.
- Respiratory outcomes ranging from self-reported symptoms through to mortality have been evaluated, with administrative databases and surveillance frameworks facilitating larger studies.
- Smoke exposure is consistently associated with respiratory health outcomes, and it is most clearly associated with outcomes specific to asthma.

A RANGE OF SMOKE EXPOSURE ASSESSMENT METHODS

Forest fire smoke is a complex mixture of solids and gases, the composition of which varies with factors such as fuel type and fire temperature [17]. Most epidemiologic studies have used particulate matter measurements to represent this mixture, recognizing that air quality monitoring networks cannot capture the true spatial and temporal variability of smoke plumes. Recent work builds on this conventional approach by exploring innovative methods to assess smoke exposure, particularly in areas with limited air quality monitoring. We have classified the methods used by each study reviewed as 'simple' or 'complex' (Table 1), where simple approaches used existing data to estimate smoke exposure directly and complex approaches required extensive additional analyses.

Simple methods

Kolbe and Gilchrist [13▪] and Vora *et al.* [15▪] took the most straightforward approach, using particulate matter measurements to identify single smoke episodes caused by known fires. In the absence of particulate matter measurements, Caamano-Isorna *et al.* [9▪] simply used the number of known fires as a proxy for smoke episodes. Analitis *et al.* [8▪] used daily burned area as a similar proxy for daily smoke exposure over a 7-year period, although black smoke (a measure of particulate matter light reflectance) concentrations were also available. Henderson *et al.* [11▪▪] assessed daily smoke exposure over a single fire season using PM_{10} measurements and the footprints of smoke plumes visible from satellite images.

Complex methods

In addition to the simple methods, Henderson *et al.* [11▪▪] entered emissions from fires detected by remote sensing into a pollution dispersion model to estimate smoke-specific PM_{10} concentrations throughout the study area [18]. This approach was intended to improve the spatial scale of the exposure estimates and to focus analyses on the health effects specific to smoke (by removing background particulate matter), which was a common objective among the studies using complex methods. Johnston *et al.* [12▪] took a 14-year time series and defined every day with a 24-h PM_{10} concentration over the 99th percentile as an 'extreme air pollution event'. Multiple sources of information (government records, media reports, and remote sensing data) were then used to determine which events were caused by fire smoke [19▪], and analyses were conducted using the binary variable 'smoke event day' instead of the PM_{10} concentrations. Rappold *et al.* [14▪▪] identified 3 days of high exposure using $PM_{2.5}$ measurements, and classified areas as 'exposed' or 'referent' using aerosol optical depth (AOD, a measure of light reflected by particulate matter in the atmosphere) data collected every 30 min by remote sensing. Remote sensing data were also central to the approach used by Delfino *et al.* [10▪▪] for a single fire season. Although their study area had a dense air quality monitoring network, many of the stations only measured particulate matter every 3 or 6 days, and some stations were incapacitated by the smoke or the fires. Satellite images were used to identify areas of 'no smoke', 'light smoke', and 'heavy smoke', and these classifications were combined with meteorological variables to model the available $PM_{2.5}$ measurements. The resulting regression equations were used to generate a complete set of daily $PM_{2.5}$ data for all the stations, and values were assigned to plume footprints (during the fire period) or interpolated (during the prefire and postfire periods) to estimate daily exposures across the study area [16].

A RANGE OF RESPIRATORY OUTCOMES

The respiratory health effects evaluated were as diverse as the approaches used for smoke exposure assessment. We have classified the outcomes reported by the eight studies as 'mild' or 'severe' (Table 1), where the potential long-term health risks of mild outcomes were small when compared with those of severe outcomes.

Mild outcomes

Kolbe and Gilchrist [13▪] assessed the self-reported symptoms including coughing, throat irritation,

Table 1. Summary of the reviewed studies

Author, year	Description	Study population	Smoke exposure assessment method(s)	Respiratory health outcome(s)
Analitis *et al.* 2011 [8▪]	Time-series analysis of all fires seasons between 1998 and 2004 in Athens, Greece	>3 000 000	Simple: every day in the time series was classified as no fire, small fire, moderate fire, or large fire according to area burned	Severe: respiratory mortality
Caamano-Isorna *et al.* 2011 [9▪]	Ecologic assessment of the impacts of the 2006 fire season in Galicia, Spain	~2 040 000	Simple: each of 156 municipalities was classified as having no exposure, medium exposure, or high exposure based on the number of fires in the municipality	Mild: dispensation of drugs for obstructive airway diseases
Delfino[a] *et al.* 2009 [10▪▪]	Poisson analysis of the prefire, during-fire, and postfire season of 2003 in southern California, USA	~20 500 000	Complex: $PM_{2.5}$ estimates for each of 560 postal codes within the study area. Estimates used particulate matter measurements, visibility, meteorological data, and remote sensing data [16]	Severe: respiratory hospital admissions
Henderson *et al.* 2011 [11▪▪]	Population-based cohort followed through the 2003 fire season in British Columbia, Canada	281 711	Simple: PM_{10} measurements Simple: smoke plume footprints Complex: PM_{10} dispersion model estimates [14▪▪]	Mild: respiratory physician visits Severe: respiratory hospital admissions
Johnston *et al.* 2011 [12▪]	Case-crossover analysis of all fire seasons between 1994 and 2007 in Sydney, Australia	~3 862 000	Complex: Validated extreme air pollution event days caused by forest fire smoke [17]	Severe: respiratory mortality
Kolbe[a] and Gilchrist, 2009 [13▪]	Telephone survey of community residents affected by the 2003 fire season in Albury, Australia	389	Simple: known fire smoke event	Mild: respiratory symptoms
Rappold *et al.* 2011 [14▪▪]	Poisson analysis of a high exposure period during a 2008 peat fire in South Carolina, USA	~2 670 000	Complex: 3-day period of high exposure identified from particulate matter measurements. Remote sensing aerosol optical depth was used to classify each of 41 counties during the high exposure period	Severe: respiratory emergency department visits
Vora *et al.* 2011 [15▪]	Case series of eight asthmatics enrolled in other asthma research studies during the 2007 fires season in San Diego, USA	8	Simple: known fire smoke event	Mild: peak expiratory flow and forced expiratory volume Mild: sputum eosinophils Mild: rescue medication usage

Assessment methods defined as 'simple' used existing data to directly estimate smoke exposure, while those defined as 'complex' required extensive additional analyses (available publications on these methods are cited). The potential long-term health risks of 'mild' outcomes were small when compared with those of 'severe' outcomes.

[a]Although these studies were published before the review period, we include them as illustrative examples that have not been described in previous reviews.

shortness of breath, wheezing, asthma, and bronchitis. Vora *et al.* [15▪] measured lung function, sputum eosinophils, and rescue mediation usage in diagnosed asthmatics. Caamano-Isorna *et al.* [9▪] used pharmaceutical billings database to evaluate the use of drugs for obstructive airway diseases. Henderson *et al.* [11▪▪] used an administrative database of outpatient physician visits with respiratory diagnosis codes, including separate analyses for asthma-specific codes.

Severe outcomes

Henderson *et al.* [11▪▪] repeated their analyses for the more severe outcome of hospital admissions with respiratory diagnoses. Delfino *et al.* [10▪▪] also used respiratory hospital admissions, including specific analyses for asthma, chronic obstructive pulmonary disease (COPD), acute bronchitis plus bronchiolitis, and pneumonia. Rappold *et al.* [14▪▪] made use of emergency room data from a real-time public health surveillance system, including specific analyses for

asthma, COPD, pneumonia plus acute bronchitis, and upper respiratory infections. Analitis *et al.* [8▪] and Johnston *et al.* [12▪] both examined mortality with underlying respiratory causes.

CONSISTENT ASSOCIATIONS

To compare the results from these eight disparate studies, we have summarized the reported associations in a matrix of exposure methods and outcome severities (Table 2). The quantitative results of each study are discussed below, ordered by the size of the study population. To further highlight the clearest and strongest associations, we have also summarized the results of all studies reporting on outcomes more specific to asthma (Table 3).

Vora *et al.* [15▪] conducted the smallest study on a convenience sample of eight asthmatic patients who had data collected for a separate research project during a coincidental 5-day smoke event in San Diego, California, USA (~1.2 million). The authors did not describe the criteria used to define asthma, nor the underlying severity of asthma in any of the participants. Lung function was not significantly decreased in any of the patients, but five of the eight did demonstrate increasing use of rescue medication. Sputum testing was only conducted on two patients, and both showed increased sputum eosinophils (a marker of pulmonary inflammation) during the smoke event. Although the findings cannot be generalized to all people with asthma, they are consistent with the previously published evidence about the pulmonary toxicology of forest fire smoke [20] and the clinical responses of people with asthma to air pollution from forest fires [21].

Kolbe and Gilchrist [13▪] randomly sampled 389 households from Albury, Australia (~40 000) following a 38-day smoke event. They documented a high frequency of self-reported respiratory symptoms in all respondents, but particularly in those who self-identified as having previous diagnoses of a respiratory condition. For example, of the 20% of respondents who reported a history of asthma, 44% reported increased shortness of breath in association with the pollution episode, compared with 24% of all respondents. Overall, 70% of respondents experienced symptoms (respiratory and nonrespiratory) that they attributed to smoke exposure, with 5% seeking healthcare as a consequence. The main limitations of these results are the reliance

Table 2. Summary of associations for general respiratory outcomes

Exposure/ outcome	First author	Exposure	Outcome	Association[a]	Notes on age for clear associations (↑↑)
Simple/mild	Vora [15▪]	Known event	Lung function	–	
	Henderson [11▪▪]	Smoke plume footprints	Physician visit	↑	
	Vora [15▪]	Known event	Sputum eosinophils	↑	
	Caamano-Isorna [9▪]	Number of fires	Obstructive airway drugs	↑↑	Pensioners only (age not specified)
	Henderson [11▪▪]	Measured PM_{10}	Physician visit	↑↑	All ages, higher in 20–50 years
	Kolbe [13▪]	Known event	Respiratory symptoms	↑↑	All ages, higher in 40–74 years
	Vora [15▪]	Known event	Medication usage	↑↑	Average age 36 ± 10 years
Complex/mild	Henderson [11▪▪]	Modeled PM_{10}	Physician visit	↑	
Simple/severe	Analitis [8▪]	Measured black smoke	Mortality	–	
	Henderson [11▪▪]	Smoke plume footprints	Hospital admission	↑	
	Analitis [8▪]	Fire size	Mortality	↑↑	All ages, higher in 75+ years
	Henderson [11▪▪]	Measured PM_{10}	Hospital admission	↑↑	All ages
Complex/severe	Johnston [12▪]	Validated smoke event	Mortality	↑	
	Delfino [10▪▪]	Modeled $PM_{2.5}$	Hospital admission	↑	
	Henderson [11▪▪]	Modeled PM_{10}	Hospital admission	↑↑	All ages
	Rappold [14▪▪]	Aerosol optical depth	Emergency visit	↑↑	All ages, higher in <65 years

[a]A dash (–) indicates no association; a single arrow (↑) indicates a suggested association (not statistically significant, where applicable); and double arrows (↑↑) indicate a clear association (statistically significant, where applicable).

Table 3. Summary of associations for outcomes more specific to asthma

First author	Exposure	Outcome	Association[a]	General respiratory measure of association/asthma-specific measure of association
Caamano-Isorna [9▪]	Number of fires	Drugs for obstructive airway diseases	↑↑	N/A
Delfino [10▪▪]	Modeled $PM_{2.5}$	Asthma-specific hospital admission	↑↑	1.03 general/1.05 asthma
Henderson [11▪▪]	Measured PM_{10}	Asthma-specific physician visit	↑↑	1.05 general/1.16 asthma
	Satellite smoke	Asthma-specific physician visit	↑↑	1.08 general/1.21 asthma
	Modeled PM_{10}	Asthma-specific physician visit	↑↑	1.01 general/1.04 asthma
Kolbe [13▪]	Known event	Asthma symptoms in self-reported asthmatics	↑↑	24% overall/44% asthmatics
Rappold [14▪▪]	Aerosol optical depth	Asthma-specific emergency visits	↑↑	1.66 general/1.65 asthma
Vora [15▪]	Known event	Medication usage in diagnosed asthmatics	↑↑	N/A

[a]A dash (–) indicates no association; a single arrow (↑) indicates a suggested association (not statistically significant, where applicable); and double arrows (↑↑) indicate a clear association (statistically significant, where applicable).

on self-reporting and the lack of baseline data to provide context for the findings.

Henderson *et al.* [11▪▪] identified an administrative, population-based cohort of 281 711 individuals residing in eastern British Columbia, Canada (~640 000), who were exposed to forest fire smoke over a 3-month period. Physician visits for general respiratory diagnoses were significantly associated with measured PM_{10} [30 $\mu g/m^3$, odds ratio (OR) = 1.05; 95% confidence interval (CI) = 1.03–1.06], and insignificantly associated with smoke plume footprints (in-plume OR = 1.08; 95% CI = 0.99–1.18) and modeled PM_{10} (60 $\mu g/m^3$ OR = 1.01; 95% CI = 0.99–1.03). Point estimates were higher and significant for asthma-specific diagnoses (Table 3). Hospital admissions for general respiratory diagnoses were significantly associated with measured PM_{10} (30 $\mu g/m^3$, OR = 1.15; 95% CI = 1.00–1.29) and modeled PM_{10} (60 $\mu g/m^3$, OR = 1.11; 95% CI = 1.04–1.18), and insignificantly associated with smoke plume footprints (in-plume OR = 1.60; 95% CI = 0.09–2.81). This is the first study to examine the health effects of forest fire smoke within a cohort. The results are internally consistent within a range of exposure measures and externally consistent with other work [7▪▪,10▪▪,22,23].

Caamano-Isorna [9▪] examined the association between the number of regional forest fires and dispensations of drugs for obstructive airways diseases (we assume these include asthma and chronic obstructive pulmonary disease). The dispensation billings were converted to a metric of defined daily doses per 1000 people in each of 156 municipal regions of Galicia, Spain (~2 million). There were no significant changes in the 'no exposure' (0–3 fires) and 'medium exposure' (4–10 fires) categories, but daily dispensations were increased by 18 and 12 doses for 'high exposure' (11–58 fires) male and female pensioners, respectively, after the fire season. Pharmacy sales have previously been used to monitor the impact of fluctuations in air pollution [24] and pollen counts [25], providing an informative health outcome whether the drugs are primarily used to treat specific, short-term symptoms. They are also useful in smaller populations in which more severe outcomes are too infrequent to be significantly associated with environmental exposures.

Rappold *et al.* [14▪▪] studied the public health effects of peat fire smoke in 41 North Carolina counties (~2.7 million) using emergency department visits reported through a syndromic surveillance system that included data from 111 of 114 civilian emergency rooms. The 18 exposed counties had 65–70% increases in cumulative relative risk of visits for asthma, COPD, pneumonia or bronchitis, and upper respiratory infections (insignificant association) during the 3-day smoke episode and within the following 5 days. No changes were observed in the 23 referent counties. Risks were generally increased in women and in those less than 65 years of age. This is the first comprehensive study on the health effects of peat fire smoke, which is different from forest fire smoke in composition (and possibly in its range and magnitude of health impacts) [26].

Analitis *et al.* [8▪] assessed how respiratory mortality was associated with black smoke and the area burned by 236 fires over a 7-year period in Athens, Greece (~3 million). There was no association between 'small fire' (0.1–1 km^2, $n = 252$) days and mortality, but 'medium fire' (1–30 km^2, $n = 42$) and 'large fire' (30+ km^2, $n = 7$) days were significantly associated with increases of 16% (95% CI = 1.3–33.4%) and 92% (95% CI = 47.5–150%)

in daily respiratory mortality, respectively. This work is challenging to compare with other mortality studies because fire size is a proxy for smoke exposure, and we do not know whether the particulate matter concentrations were elevated on fire days. Although black smoke measurements were also available, the mean (SD) concentration was 45 (21) $\mu g/m^3$ on the 770 'no fire' days and 36 (10) $\mu g/m^3$ on the seven 'large fire' days. This suggests that black smoke (a measure of particulate matter light reflectance that can only be used to estimate particulate matter mass concentration) did not reliably reflect the air quality impacts of forest fire smoke in Athens.

Johnston *et al.* [12▪] more directly assessed how respiratory mortality was affected when forest fire smoke caused 50 extreme air pollution events ($PM_{10} \geq 47\,mg/m^3$) in Sydney, Australia (~3.8 million) over a 14-year period. Although smoke events were significantly associated with all-cause mortality (OR = 1.05; 95% CI = 1.00–1.10), they were insignificantly associated with respiratory mortality (OR = 1.09; 95% CI = 0.88–1.36). The higher point estimate for respiratory mortality compared with all-cause mortality is, however, consistent with the work of Analitis *et al.* [8▪] and others [23,27].

Delfino *et al.* [10▪▪] studied the largest population, associating $PM_{2.5}$ concentrations in 560 postal codes with hospital admissions before, during, and after the 2003 fire season in southern California (~20.5 million). They reported that a 10 $\mu g/m^3$ increase in estimated $PM_{2.5}$ from wildfires was associated with admissions for acute bronchitis (relative risk, RR = 1.10; 95% CI = 1.02–1.18), pneumonia (RR = 1.03; 95% CI = 1.01–1.05), COPD (RR = 1.04; 95% CI = 1.00–1.07), and asthma (RR = 1.05; 95% CI = 1.02–1.08). Similarly to Rappold *et al.* [14▪▪], the association with COPD was largest in 20–65 years age category. Although asthma admissions were increased overall, the largest association was in adults over 65, which is consistent with other reports of asthma outcomes being higher in adults than in school-aged children [11▪▪,21,23].

CONCLUSION

Eight studies have used a wide range of exposure assessment methods in a wide range of study designs to examine the associations with a wide range of respiratory outcomes in a wide range of populations. The overall results are markedly consistent, both internally and externally [7▪▪]. Although readers may place greater confidence in the more rigorous studies, we also want to acknowledge the value of simpler approaches. Forest fire smoke is a challenging exposure to evaluate, and many questions still remain about its health effects. Studies reporting on acute cardiovascular outcomes have been largely null, but recent work has found that out-of-hospital cardiac arrests were increased on smoky days [28], and three studies reviewed here reported other significant associations [8▪,12▪,14▪▪]. These results are consistent with the acute cardiovascular effects of urban particulate matter [29], but we need to build internal consistency within the literature on forest fire smoke. Similarly, there is a dearth of evidence about exposures and outcomes in the equatorial regions more regularly affected by smoke from rainforest clearing. Finally, to the best of our knowledge, there is no evidence on the comparative risks of very acute (1–3 h) and acute (24 h) exposures, nor on chronic outcomes associated with acute exposures. Any contributions that help to address these gaps will be valuable additions to a sparse literature, and we believe that simple studies are preferable to no studies at all. Noonan and Balmes [30] recently articulated some specific ideas for interested investigators.

This is an exciting time for research on the health effects of forest fire smoke. Environmental databases and remote sensing products are facilitating new and innovative approaches to exposure assessment. Administrative health databases are facilitating population-based research, thereby improving the statistical power of many analyses. Furthermore, growing interest in the burden of disease [31▪▪] and health costs associated with smoke exposure [32▪] may help to generate future funding for studies of all shapes and sizes.

Acknowledgements

We would like to thank the authors of the reviewed papers for answering our questions about their work. We would also like to acknowledge everyone who is helping to build this body of literature.

Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:
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A novel use of fire count as a proxy for smoke exposure. The first study to assess the association between forest fires and pharmaceutical use within an entire population.

10. Delfino RJ, Brummel S, Wu J, *et al.* The relationship of respiratory and ■■ cardiovascular hospital admissions to the southern California wildfires of 2003. Occup Environ Med 2009; 66:189–197.

A rigorous study conducted in a very large population. Excellent PM2.5 exposure assessment at a fine spatial resolution. Clear, population-based health outcome with associations reported for the prefire, during-fire, and postfire periods to differentiate the effects of fire-related particulate matter from background particulate matter.

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PLOS | MEDICINE

RESEARCH ARTICLE

The San Diego 2007 wildfires and Medi-Cal emergency department presentations, inpatient hospitalizations, and outpatient visits: An observational study of smoke exposure periods and a bidirectional case-crossover analysis

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OPEN ACCESS

Citation: Hutchinson JA, Vargo J, Milet M, French NHF, Billmire M, Johnson J, et al. (2018) The San Diego 2007 wildfires and Medi-Cal emergency department presentations, inpatient hospitalizations, and outpatient visits: An observational study of smoke exposure periods and a bidirectional case-crossover analysis. PLoS Med 15(7): e1002601. https://doi.org/10.1371/journal.pmed.1002601

Academic Editor: Madeleine Thomson, Africa Program, UNITED STATES

Received: February 22, 2018

Accepted: June 1, 2018

Published: July 10, 2018

Data Availability Statement: Medi-Cal data are available from the State of California Health and Human Services Agency, Department of Health Care Services for researchers who meet the criteria for access to confidential data (http://www.dhcs.ca.gov/dataandstats/data/Pages/default.aspx). Wildfire exposure data were developed by Michigan Technological University and are provided as Supporting information with the article.

Abstract

Background

The frequency and intensity of wildfires is anticipated to increase as climate change creates longer, warmer, and drier seasons. Particulate matter (PM) from wildfire smoke has been linked to adverse respiratory and possibly cardiovascular outcomes. Children, older adults, and persons with underlying respiratory and cardiovascular conditions are thought to be particularly vulnerable. This study examines the healthcare utilization of Medi-Cal recipients during the fall 2007 San Diego wildfires, which exposed millions of persons to wildfire smoke.

Methods and findings

Respiratory and cardiovascular International Classification of Diseases (ICD)-9 codes were identified from Medi-Cal fee-for-service claims for emergency department presentations, inpatient hospitalizations, and outpatient visits. For a respiratory index and a cardiovascular index of key diagnoses and individual diagnoses, we calculated rate ratios (RRs) for the study population and different age groups for 3 consecutive 5-day exposure periods (P1 [October 22–26], P2 [October 27–31], and P3 [November 1–5]) versus pre-fire comparison periods matched on day of week (5-day periods starting 3, 4, 5, 6, 8, and 9 weeks before each exposed period). We used a bidirectional symmetric case-crossover design to examine emergency department presentations with any respiratory diagnosis and asthma specifically, with exposure based on modeled wildfire-derived fine inhalable particles that are 2.5

Funding: NF and MB work conducting the exposure modeling in this research was supported by grant 1-RC1-ES018612 from the National Institute of Environmental Health Sciences, a part of the National Institutes of Health, under the NIEHS Interagency Working Group on Climate Change and Health Initiative. No funding bodies had any role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing interests: The authors have declared that no competing interests exist.

Abbreviations: AIC, Akaike information criteria; AQI, Air Quality Index; CI, confidence interval; COPD, chronic obstructive pulmonary disease; DHCS, Department of Health Care Services; HYSPLIT, Hybrid Single-Particle Lagrangian Integrated Trajectories; ICD, International Classification of Diseases; MIS/DSS, Management Information System/Decision Support System; MLE, maximum likelihood estimate; OR, odds ratio; PM, particulate matter; PM_{10}, inhalable particles that are 10 micrometers and smaller; $PM_{2.5}$, fine inhalable particles that are 2.5 micrometers and smaller; RR, rate ratio; SES, socioeconomic position and status; US EPA, US Environmental Protection Agency; WFEIS, Wildland Fire Emissions Information System.

micrometers and smaller ($PM_{2.5}$). We used conditional logistic regression to estimate odds ratios (ORs), adjusting for temperature and relative humidity, to assess same-day and moving averages. We also evaluated the United States Environmental Protection Agency (EPA)'s Air Quality Index (AQI) with this conditional logistic regression method. We identified 21,353 inpatient hospitalizations, 25,922 emergency department presentations, and 297,698 outpatient visits between August 16 and December 15, 2007. During P1, total emergency department presentations were no different than the reference periods (1,071 versus 1,062.2; RR 1.01; 95% confidence interval [CI] 0.95–1.08), those for respiratory diagnoses increased by 34% (288 versus 215.3; RR 1.34; 95% CI 1.18–1.52), and those for asthma increased by 112% (58 versus 27.3; RR 2.12; 95% CI 1.57–2.86). Some visit types continued to be elevated in later time frames, e.g., a 72% increase in outpatient visits for acute bronchitis in P2. Among children aged 0–4, emergency department presentations for respiratory diagnoses increased by 70% in P1, and very young children (0–1) experienced a 243% increase for asthma diagnoses. Associated with a 10 $\mu g/m^3$ increase in $PM_{2.5}$ (72-hour moving average), we found 1.08 (95% CI 1.04–1.13) times greater odds of an emergency department presentation for asthma. The AQI level "unhealthy for sensitive groups" was associated with significantly elevated odds of an emergency department presentation for respiratory conditions the day following exposure, compared to the AQI level "good" (OR 1.73; 95% CI 1.18–2.53). Study limitations include the use of patient home address to estimate exposures and demographic differences between Medi-Cal beneficiaries and the general population.

Conclusions

Respiratory diagnoses, especially asthma, were elevated during the wildfires in the vulnerable population of Medi-Cal beneficiaries. Wildfire-related healthcare utilization appeared to persist beyond the initial high-exposure period. Increased adverse health events were apparent even at mildly degraded AQI levels. Significant increases in health events, especially for respiratory conditions and among young children, are expected based on projected climate scenarios of wildfire frequency in California and globally.

Author summary

Why was this study done?

- Large wildfires are becoming more frequent and are expected to increase with climate change. Smoke from wildfires can cause health problems, especially for children, older persons, and people who already have respiratory or heart problems.
- Researchers had access to data on emergency department visits, hospitalizations, and outpatient visits from California's Medicaid program, Medi-Cal. This allowed for analysis of the effects of wildfire among a particularly vulnerable population, which included a large proportion of young children. It also provided an opportunity to examine changes in outpatient visits.

- Researchers were able to look at health problems during the time when the wildfire smoke was most intense and also at later periods to see if people had health problems that may take more time to develop. They chose to study a very large wildfire that happened in San Diego County in 2007.

What did the researchers do and find?

- During the peak fire period, emergency department visits for respiratory conditions increased by 34% and visits for asthma by 112%. There was no change in visits for heart-related problems.
- Some healthcare visit types remained high even after the peak fire period. For example, outpatient visits for acute bronchitis were 72% above the usual rate in the 5-day period following the peak fire period.
- Young children had bigger increases in visits during the peak fire period than older age groups. Children aged 0–4 had a 136% increase in emergency department visits for asthma, and very young children aged 0–1 experienced a 243% increase.
- Researchers studied how health visits changed on days with more intense smoke using data from smoke models. Emergency department visits for asthma went up 73% on days following an air quality day designated as "unhealthy for sensitive populations," based on wildfire smoke and using the United States Environmental Protection Agency (EPA)'s Air Quality Index (AQI) air pollution levels as a guide.

What do these findings mean?

- We expect increases in respiratory problems during wildfires, possibly even at mildly degraded levels of air quality. People may continue to seek care for some persisting conditions.
- Young children appear at highest risk for respiratory problems during a wildfire, which is cause for particular concern because of the potential for long-term harm to children's lung development.
- The risk of future wildfires on the health of Californians will continue to be shaped by global climate change, as well as the anticipated growth of vulnerable subpopulations. Planning to protect the health of vulnerable populations is important.

Introduction

Large forest fires have become more frequent in the Western United States since the 1980s [1–3]. Under most future climate scenarios, the frequency and size of wildfires in the southwestern states are expected to increase [4]. Climate models predict up to a 74% increase in area burned in California and a possible doubling of wildfire emissions by the end of the century [5]. Wildfires release large amounts of particulate matter (PM) and other toxic substances into the air, including carbon dioxide, carbon monoxide, and methane [6–7]. In the coterminous US, yearly emissions of fine PM from wildfire smoke are estimated to be between 118,000 and 986,000 metric tons and carbon dioxide emissions between 24 and 134 million metric tons, in

addition to other compounds and gases [6]. In 2012, wildfires contributed 20% of the fine particulate emissions in the US [8].

Smoke from fires can be transported to affect populations far downwind [9]. Projected trends in climate change show that, globally, the number of people who will experience adverse health effects from wildfires is increasing [10–12]. The number of persons who are vulnerable is also expanding because more people live near wildlands [13].

Wildfire smoke exposures have been associated with adverse health outcomes, including premature death and increased inpatient hospitalizations and emergency department presentations [14–16]. Smoke from wildfires produces inhalable particles that are 10 micrometers and smaller (PM_{10}) and fine inhalable particles that are 2.5 micrometers and smaller ($PM_{2.5}$). PM_{10} and $PM_{2.5}$ have consistently been linked to respiratory outcomes, particularly asthma exacerbations [15–17] and in some studies, cardiovascular outcomes [17–20]. Relatively few studies of wildfire smoke have examined the health effects on vulnerable populations. However, the nature and intensity of health impacts are expected to depend on characteristics of the receptor population [16,17,21]. Research on vulnerability to ambient air pollution has identified subpopulations with increased susceptibility to the effects of PM; these include persons with chronic diseases [22], as well as older adults, children, and possibly those with lower education, income, and employment status [23]. Although PM of wildfire origin differs from ambient air pollution in composition and exposure patterns, current research suggests that elderly and young populations will also be especially vulnerable to wildfire-derived PM [16,17,24]. Children warrant particular concern because their lungs are still developing, and exposure to ambient air pollution has been shown to permanently impair lung function [25].

Individual socioeconomic position or status (SES) factors such as personal income and education are accompanied by a broad range of factors that influence health, including prevalent comorbid conditions such as respiratory and cardiovascular diseases, as well as access to healthcare, social stress, and environmental quality of the community [26]. Often, these factors are difficult to isolate.

California's Medicaid program, Medi-Cal, is a public health insurance program covering health services for low-income individuals, including seniors, persons with disabilities, families with children, children in foster care, pregnant women, and childless adults with incomes below 138% of the federal poverty level. These eligibility criteria create a population that tends to be focused on low-income women and children, plus others with varying disabilities. Beginning at age 65, Medicare is available regardless of income, so for this group, Medi-Cal only pays secondarily or for certain services not covered by Medicare.

In this study, we investigated change in healthcare utilization—including differential health responses by age groups and type of health service—related to wildfire smoke exposure from a large complex of fires in San Diego County in 2007 within a vulnerable population, Medi-Cal beneficiaries who resided in San Diego County at the time.

Methods

Study area and design

In late October of 2007, a complex of fires burned nearly 1 million acres in San Diego county, resulting in the evacuation of an estimated 515,000 county residents and numerous road, school, and business closures [27]. San Diego county had a population of 3,095,342 according to the 2010 US Census [28], with the population concentrated along the coastal areas.

Medi-Cal beneficiaries numbered 345,257 in San Diego County in July 2007 [29]. Medi-Cal administrative claims data were obtained from the California Department of Health Care Services' (DHCS) Management Information System/Decision Support System (MIS/DSS) data

warehouse for San Diego County for the period of August 1 through December 31, 2007 to accommodate reference dates surrounding the late-October fire period.

We conducted 2 types of analyses. The first was a county-wide analysis of Medi-Cal claims data, which compared rates for emergency department presentations, inpatient hospitalizations, and outpatient visits during the fires with reference periods. The second was a case-crossover analysis that examined exposures by residential zip code and emergency department presentations with respiratory diagnoses.

For the county-wide analysis, we identified October 22–26 as the peak fire-exposure period (P1) based on a previous study that analyzed this fire using data from the BioSense Platform, an integrated national syndromic surveillance system [30]. We defined 2 following periods, P2 (October 27–31) and P3 (November 1–5), for analysis in order to identify any health outcomes that might be sensitive to cumulative or lagged exposure to wildfire smoke.

For the case-crossover analyses of exposure to varying concentrations of $PM_{2.5}$, the population was limited to those beneficiaries with a valid San Diego County zip code listed for their residential address. Where possible, post office-box–only zip codes were mapped to real-address zip codes in the same subregion, municipality, and neighborhood. Exposures were based on the modeled $PM_{2.5}$ for these 101 real-address zip codes.

Environmental data

Wildfire $PM_{2.5}$ concentrations were estimated through the use of coupled models of wildfire smoke emissions and atmospheric dispersion [31]. Spatially and temporally resolved estimates of wildland fire emissions were computed using the geospatial tool Wildland Fire Emissions Information System (WFEIS); model outputs were then introduced into the meteorological atmospheric transport model Hybrid Single-Particle Lagrangian Integrated Trajectories (HYSPLIT) to produce PM concentration estimates computed to a 0.01-degree grid (approximately 1 km^2) on an hourly basis. Hourly model outputs were used to estimate daily average wildfire $PM_{2.5}$ concentrations ($\mu g/m^3$) by zip code, as described previously [31]. All analyses in this study are based on PM originating from wildfire sources, so all PM in this manuscript refers to wildfire-only PM. We interpolated relative humidity and temperature data from a Remote Automated Weather Station database to county subregional areas for the period of August to November 2007 (environmental data availability period).

Health data

Medi-Cal dataset variables included county of residence and home zip code of the patient, date of the medical visit, general type of service provided, where the visit occurred, classification of the provider (i.e., hospital, emergency department, outpatient, excluding claims related to nursing homes, etc.), and diagnosis that was being treated (by International Classification of Diseases [ICD]-9 code, up to 2 diagnoses per claim). Patient demographic variables included sex and age. A unique, de-identified beneficiary code (beneficiary ID) was provided with the dataset; names were not included. Eligible subjects were San Diego County residents who had a qualifying Medi-Cal fee-for-service claim during the study period. Qualifying claims included those for inpatient hospitalizations, emergency department presentations, and outpatient visits (clinic and physician office visits). The DHCS Data and Research Committee and California's Health and Human Services Agency's Committee for the Protection of Human Subjects approved the study protocol. We performed data management and analysis using SAS version 9.4 (SAS Institute; https://www.sas.com/en_us/home.html) and Excel for Mac version 14.4.3 (Microsoft, https://www.microsoft.com/en-us/).

Identification and description of beneficiaries

The beneficiary ID linked all claims records for each beneficiary. Beneficiaries aged 65 and above were excluded from the study because claims for these beneficiaries were not adequately represented in the Medi-Cal data due to their dual eligibility for Medicare and Medi-Cal.

Identification of episodes of care

Episodes of care ("encounters") were identified from the subset of records with at least one valid diagnosis code. For each beneficiary, inpatient status was assessed for each day from August 1 through December 31, 2007. Inpatient hospitalizations were identified as periods of one or more contiguous days with associated inpatient claims records; the start date of the earliest record was used as the admission date. Emergency department claims records for each beneficiary from the same date were grouped together into a single episode of care. Overnight emergency department presentations were identified, and records from both those dates were grouped into a single episode of care. Physician office and clinic claims records for each beneficiary from the same date were grouped together into a single episode of care, referred to hereafter as outpatient visits. To reduce misclassification of inpatient diagnosis, errors in ascertainment of inpatient status, and errors in date of inpatient admission, the episodes-of-care dataset was limited to episodes with admission during the period of August 16 to December 15, 2007 (encounter data availability period).

Episodes of care were identified as being related to the outcomes of interest based on the primary and secondary diagnoses from any associated claims records, except inpatient hospitalizations, which were limited to claims records from the first 14 days of the hospitalization. Encounters for components of a respiratory index and a cardiovascular index were identified as outcomes for analysis, based on ICD-9 coding in a previous study of a large wildfire event in California (Table 1) [32]. The respiratory index included asthma, acute bronchitis, chronic obstructive pulmonary disease (COPD), bronchitis—not otherwise specified, pneumonia, upper respiratory infections, cystic fibrosis, bronchiectasis, extrinsic allergic alveolitis, respiratory symptoms, and other acute and subacute respiratory conditions caused by exposure to fumes, vapors, or external agents. The cardiovascular index included ischemic heart disease, dysrhythmia, congestive heart failure, cerebrovascular disease including stroke, and peripheral vascular disease. We also examined total visits (all-cause) for each healthcare setting to provide context for results for the outcomes of interest.

Data analysis

County-wide results by exposure periods. For the entire study population (ages 0–64), rate ratios (RRs) were calculated by counting occurrences of the outcomes of interest during the 5-day county-wide exposed periods P1 (October 22–26; highest exposures), P2 (October 27–31; lower exposures and lagged or cumulative effects), and P3 (November 1–5; lagged effects and cumulative effects) and comparing them with occurrences of the same outcome during six 5-day comparison periods, matched on day of week (5-day periods starting 3, 4, 5, 6, 8, and 9 weeks before each exposed period; weeks 1 and 2 were excluded because, for P2 and P3, they would have overlapped with P1, and week 7 was excluded to prevent comparing P1 to the Labor Day holiday). For 5 age groups of interest (0–1 years, 2–4 years, 0–4 years, 5–17 years, and 18–64 years), RRs were calculated by counting occurrences of the outcomes of interest during P1 and comparing them with occurrences of the same outcome during eight 5-day comparison periods, matched on day of week (5-day periods starting 1, 2, 3, 4, 5, 6, 8, and 9 weeks before the exposed period). We calculated Mid-P 95% confidence intervals (CIs) for RRs based on fewer than 10 events (exposed or unexposed) using OpenEpi version 3.01. For

Table 1. ICD-9 codes used to classify respiratory and cardiovascular disorders.

Condition	ICD-9 codes
Respiratory index (all respiratory codes below)	
Asthma	493
Acute bronchitis	466
COPD	491, 492, 496
Bronchitis—not otherwise specified	490
Pneumonia	480–487
Upper respiratory infections	460–464
Cystic fibrosis	277
Bronchiectasis	494
Extrinsic allergic alveolitis	495
Respiratory symptoms	786
Other acute and subacute respiratory conditions caused by exposure to fumes, vapors or external agents	506, 508
Cardiovascular index (all cardiovascular codes below)	
Ischemic heart disease	410–414
Dysrhythmia	426, 427
Congestive heart failure	402–428
Cerebrovascular disease including stroke	430–438
Peripheral vascular disease	450–459

Abbreviations: COPD, chronic obstructive pulmonary disease; ICD, International Classification of Diseases.

https://doi.org/10.1371/journal.pmed.1002601.t001

RRs based on 10 or more events (exposed and unexposed), we estimated 95% CIs using large-sample statistics for person-time RRs [33], with the following formula:

$$95\%\ \mathrm{CI} = e^{[\ln(\mathrm{RR}) \pm 1.96 * \sqrt{(1/A_1 + 1/A_0)}]},$$

where A_1 is the number of events in the exposed period and A_0 is the number of events in the control period.

Statistical significance of increases and decreases in rates were determined from the 95% confidence limits of the RR testing exclusion of 1 (prior to rounding). Changes in the incidence of an outcome in the fire period relative to the control period were calculated using the following formula: $(\mathrm{RR} - 1) \times 100\%$.

Case-crossover analysis of acute respiratory events related to $PM_{2.5}$ concentrations. The bidirectional symmetric case-crossover method [34], a statistical technique suited to examine acute effects of air pollution and effect modification for variables at the individual level, was used for this analysis. The case-crossover study is a matched design in which each case subject (on a different day or days) serves as its own control, thereby adjusting for time-invariant confounders, both known and unknown. The bidirectional symmetric design selects 2 control days from equal distances before and after the event, providing adequate control for both long-term trends and seasonality. Seasonality is of particular concern when examining respiratory health outcomes. To adjust for potential confounding by day of the week, we selected control days on the same day of the week as the case. Based on our analysis of emergency department presentations for respiratory diagnoses and asthma in P3, we expected negligible elevation in these outcomes 10–15 days after exposures. Therefore, we eliminated correlation in the exposure of interest between case days and control days by setting the interval between case days and control days to 14 days, selecting control days 2 weeks before and 2

weeks after cases. Based on the availability of episode-of-care and environmental data, the need to model lagged exposures, and the need to use exposure data from 14 days before and after each event modeled, events spanned the period from September 15 to November 15, and control days spanned the period from September 1 to November 29.

After creating a case-crossover matrix with as many strata as events, we used the SAS procedure LOGISTIC to conduct conditional logistic regressions of 2 outcomes separately—emergency department presentations for respiratory diagnoses and for asthma. $PM_{2.5}$ was scaled to represent increased odds of the inpatient hospitalization per 10 $\mu g/m^3$ increase. Using SAS, we obtained risk estimates in the form of an odds ratio (OR), corresponding 95% CI and Wald *p*-values, and Akaike information criteria (AIC) values for each model.

To determine the best model fit based on the AIC, several exposures were considered, including single-day averages of the same day (24 hour), averages of the same day and the previous day (48-hour), and averages of the same day and 2 previous days (72-hour), all adjusted for temperature and humidity. We evaluated possible effect modification by age by adding an interaction term of PM by age category and assessing significance. We also stratified by sex and re-examined significance of the age interaction.

To investigate the usefulness of existing public health recommendations, we categorized 24-hour average $PM_{2.5}$ concentrations according to the categories of the Air Quality Index (AQI), an index created by the US Environmental Protection Agency (EPA) for communicating daily air quality risks to the public [35]. The AQI values are ranked into categories—good, moderate, unhealthy for sensitive groups, unhealthy, very unhealthy, and hazardous—each with corresponding recommendations for protecting health [36]. For the outcomes of the respiratory index emergency department presentations, we performed conditional logistic regression, adjusting for temperature and relative humidity and calculating ORs relative to the reference category of "good."

In our original data analysis plan (no changes made to the IRB submission, S1 Protocol), we had proposed several additional analyses that were not ultimately conducted. Because we had anticipated finding overall excesses, we had planned to statistically screen multiple diagnosis codes and groupings in order to determine which outcomes were driving the elevations. Based on finding that the excess visits were concentrated among the respiratory diagnoses that we were already evaluating separately, no additional screening was warranted. We also had proposed calculating the cost burden but, due to time considerations, decided not to pursue these additional analyses.

Results

Population

During the health data availability period of August 1 to December 31, 2007, there were a total of 5,454,360 Medi-Cal claims for San Diego beneficiaries, derived from 217,067 residents with at least one claim of any type (not limited to the claim types we examined). We excluded 40,216 residents aged 65 and above. After these exclusions, during the fire period of October 22–26, 2007, there were 26,556 San Diego County residents with at least one Medi-Cal claim (15.0% of beneficiaries). The individuals with at least one claim during the health data availability period and fire period are described by age, sex, and race/ethnicity (Table 2).

Episodes of care

Among our study population and during the period of August 16 to December 15, 2007, we identified 25,000 emergency department presentations, 17,009 inpatient hospitalizations, and 269,842 outpatient visits. Young children aged 0–4 comprised 14.4% of inpatient

Table 2. Age, sex, and race/ethnicity of Medi-Cal beneficiaries under age 65 with fee-for-service claims during health data availability period (August 1–December 31, 2007) and peak fire period (October 22–26, 2007) in San Diego County.

	Data Availability Period (Aug 1–Dec 31, 2007)		Fire Period (Oct 22–26, 2007)	
	N	%	*N*	%
Total	176,851	100	26,556	100
Age				
0–1	24,490	13.8	2,191	8.3
2–4	15,546	8.8	1,197	4.5
5–17	42,548	24.1	4,004	15.1
18–64	94,259	53.3	19,162	72.2
Unknown/missing	8	0.00	2	0.00
Sex				
Female	110,178	62.3	16,099	60.6
Male	66,317	37.5	10,427	39.3
Unknown/missing	356	0.2	30	0.1
Race/ethnicity				
Asian/Pacific Islander	8,969	5.1	1	6.7
Black	13,807	7.8	2,575	9.7
Hispanic	77,447	43.8	9,984	37.6
Native American	821	0.5	136	0.5
White	36,306	20.5	8,014	30.2
Other/unknown	39,501	22.3	4,056	15.3

https://doi.org/10.1371/journal.pmed.1002601.t002

hospitalizations, 15.1% of emergency department presentations, and 28.8% of outpatient visits. Very young children (aged 0–1) accounted for 12.8% of inpatient hospitalizations, 10.8% of emergency department presentations, and 15.8% of outpatient visits.

Exposures

Wildfire-derived $PM_{2.5}$ concentrations are shown in Table 3. During the most intense initial period of the firestorm P1, the mean of the 24-hour average $PM_{2.5}$ concentrations of all the zip codes was 89.1 μg/m^3. The highest of all the zip codes' daily averages occurred during this

Table 3. Summary of modeled wildfire emissions: mean 24-hour, maximum 24-hour, and percentiles of 24-hour wildfire $PM_{2.5}$ concentrations across zip codes and dates during study periods in San Diego County in 2007.

	24-Hour Average $PM_{2.5}$ (μg/m^3) for Zip Codes by Exposure Period		
Measure	**P1 (Day 1–5)**	**P2 (Day 6–10)**	**P3 (Day 11–15)**
Daily mean	89.1	9.33	0.26
Daily maximum	803.1	283.9	5.72
Percentile			
5	0.2	0.0	0.0
25	7.0	0.0	0.0
50	39.9	0.68	0.16
75	131.5	13.17	0.3
95	333.1	40.5	1.05

Abbreviation: $PM_{2.5}$, fine inhalable particles that are 2.5 micrometers and smaller.

https://doi.org/10.1371/journal.pmed.1002601.t003

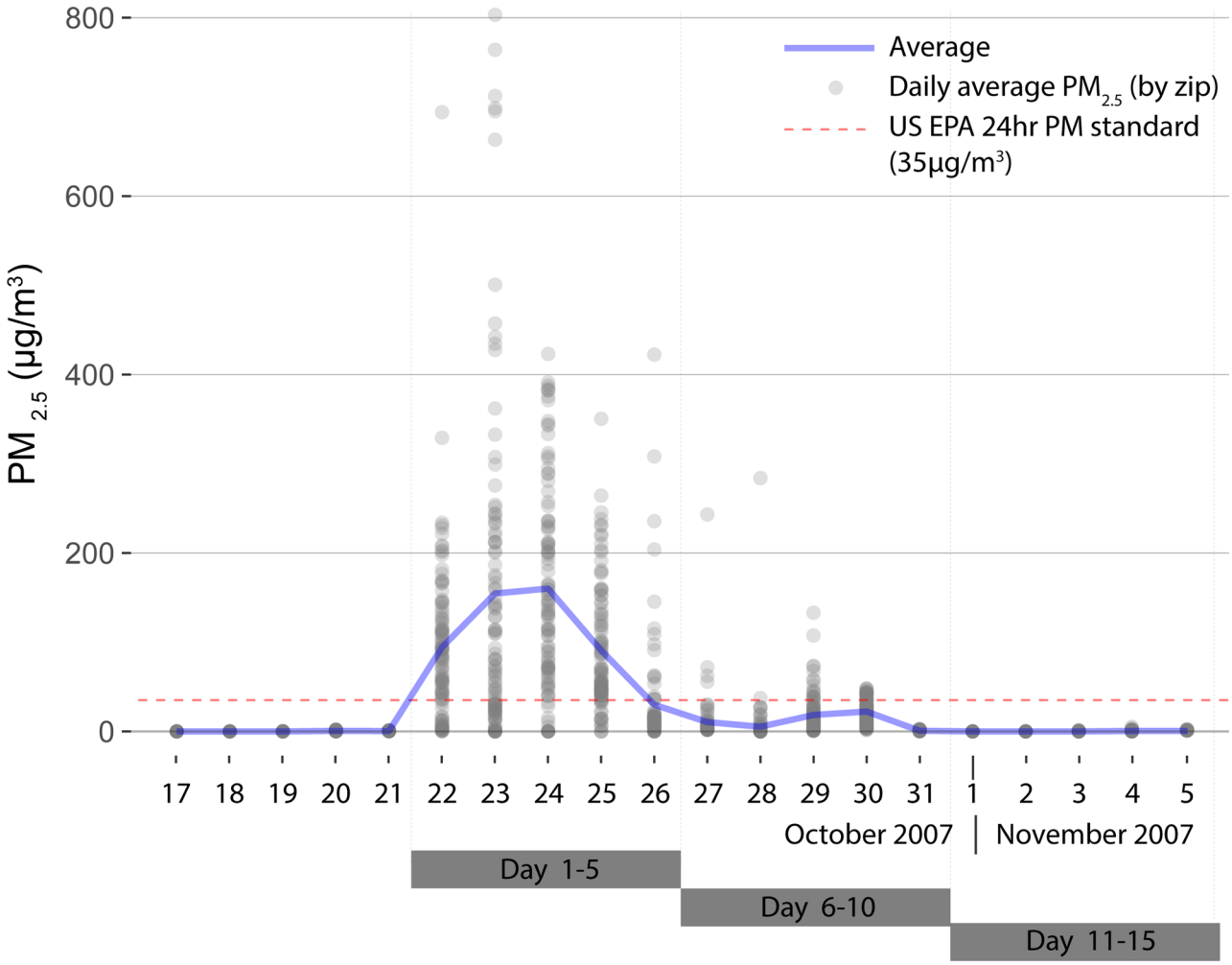

Fig 1. Wildfire $PM_{2.5}$ by day in San Diego County zip codes during 2007 wildfires. Daily average wildfire $PM_{2.5}$ for each of 101 zip codes in San Diego County for a period just prior to the 2007 Firestorm and for the 5-day windows of time following the start of the fires on October 22. The average for all zip codes is shown in blue, and the US EPA 24-hour $PM_{2.5}$ standard is in red. PM, particulate matter; $PM_{2.5}$, fine inhalable particles that are 2.5 micrometers and smaller; US EPA, Environmental Protection Agency.

https://doi.org/10.1371/journal.pmed.1002601.g001

window of time, 803.1 µg/m^3. In comparison, the US EPA 24-hour air quality standard for $PM_{2.5}$ is 35 µg/m^3, and concentrations over 250 µg/m^3 correspond to AQI level "hazardous."

Estimated average daily wildfire $PM_{2.5}$ concentrations by zip code through the course of the fire period are shown in Fig 1. Concentrations spiked sharply on October 22 and continued through the initial 5-day fire period, then declined. The mean $PM_{2.5}$ concentration on the first day of the 5-day fire period was 160 µg/m^3 (AQI "very unhealthy"), which then dropped to 29.9 µg/m^3 on the 5th day (AQI "moderate"). The fire boundaries and daily average $PM_{2.5}$ concentrations by zip code in San Diego County are mapped for the 5-day exposure period (P1) (Fig 2).

County-wide results by exposure period

Total visits. During P1 (October 22–26), there were 1,071 emergency department presentations, 725 inpatient hospitalizations, and 10,822 outpatient visits. RRs for the 5-day

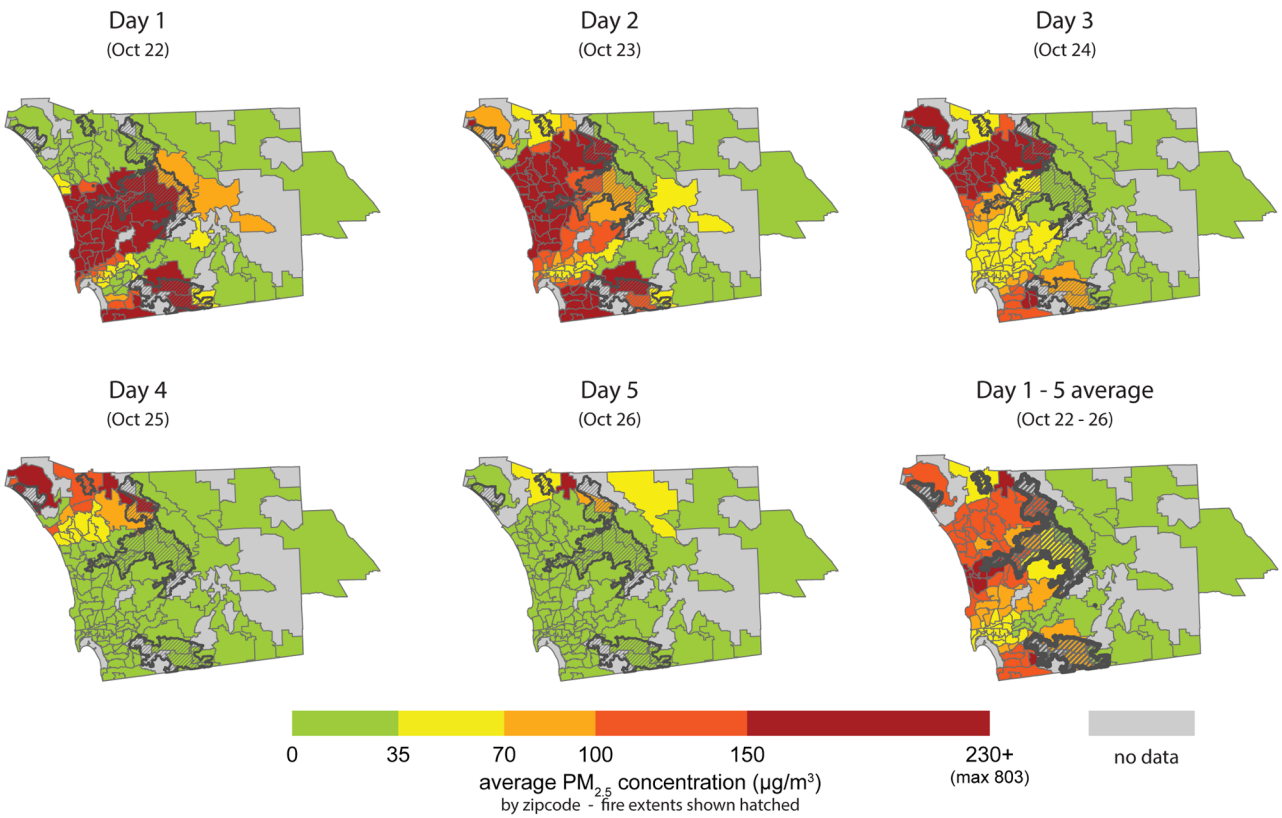

Fig 2. Map of San Diego County wildfire $PM_{2.5}$ by zip code, October 22–26, 2007 fire period. Maps show zip code mean of average daily $PM_{2.5}$ values across the 5-day fire-exposure period. Green indicates satisfactory levels according to the US EPA's 24-hour standard. Fire extent is hatched. $PM_{2.5}$, fine inhalable particles that are 2.5 micrometers and smaller; US EPA, US Environmental Protection Agency.

https://doi.org/10.1371/journal.pmed.1002601.g002

periods P1–P3 for total (all-cause) encounters and encounters for selected respiratory and cardiovascular diagnoses are shown in Fig 3 (S1 Table). Relative to the 6 reference periods, total emergency department presentations did not change significantly during P1 (1,071 versus 1,062.2; RR 1.01; 95% CI 0.95–1.08); inpatient hospitalizations (725 versus 797.8; RR 0.91; 95% CI 0.84–0.98) and outpatient visits (10,822 versus 15,790.7; RR 0.69; 95% CI 0.67–0.70) decreased significantly.

Respiratory outcomes. Despite the overall pattern of no change or deficits in total healthcare encounters, the index of respiratory diagnoses increased across all healthcare settings, with the largest magnitude observed in emergency department presentations (e.g., P1: 288 versus 215.3; RR 1.34; 95% CI 1.18–1.52).

Of the outcomes we studied, the elevations in asthma encounters were the most pronounced. In P1, excess asthma encounters were evident across all healthcare settings, although the relationship was strongest in emergency department presentations (58 versus 27.3; RR 2.12; 95% CI 1.57–2.86).

Infectious respiratory outcomes—upper respiratory infections, bronchitis, and pneumonia—increased in some healthcare settings during P1. Emergency department presentations for upper respiratory infections increased (RR 1.45; 95% CI 1.10–1.89), but not outpatient visits (RR 0.99; 95% CI 0.91–1.07). Outpatient visits for acute bronchitis were also significantly elevated in P1 (RR 1.51; 95% CI 1.23–1.87). Inpatient hospitalizations for COPD increased non-significantly in P1 (RR 1.18; 95% CI 0.79–1.77).

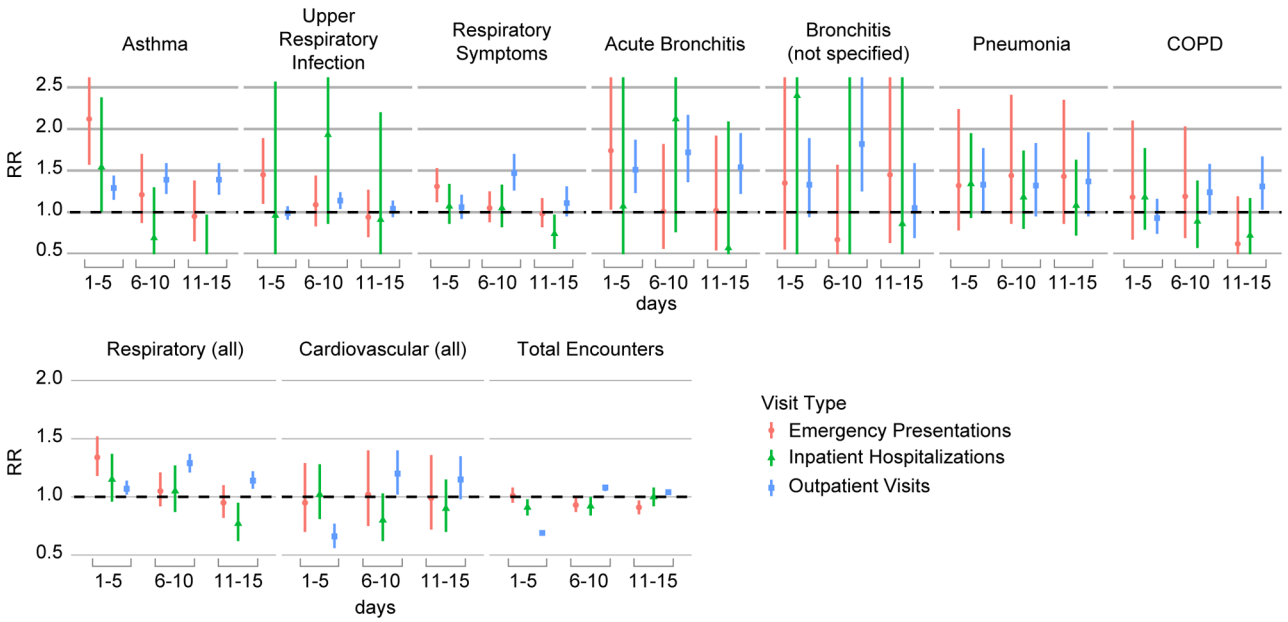

Fig 3. Respiratory and cardiovascular healthcare encounters in San Diego County during 2007 fire period. RRs for the 5-day periods starting from October 22 and for claims related to emergency department presentations (red, circle), inpatient hospitalizations (green, triangle), and outpatient visits (blue, square). The top row shows encounters for specific respiratory diagnoses. The bottom row shows encounters for the respiratory index, cardiovascular index, and total encounters (all diagnoses). COPD, chronic obstructive pulmonary disease; RR, rate ratio.

https://doi.org/10.1371/journal.pmed.1002601.g003

In general, similar types of health conditions were elevated in P2 and P3 as in P1. However, although observed increases in emergency department presentations and inpatient hospitalizations generally decreased after P1, elevations for some respiratory outcomes persisted beyond this initial high-exposure period. For example, although based on small numbers (<50), RRs for pneumonia were elevated in P1–P3 across all settings. However, some outpatient visits increased in the later time frames. Outpatient visit increases for the respiratory index appeared larger in P2 (RR 1.29; 95% CI 1.21–1.37) and P3 (RR 1.14; 95% CI 1.07–1.22) than in P1 (RR 1.07; 95% CI 1.02–1.14). Outpatient visits for individual respiratory diagnoses showed excess visits in P2, which were generally higher than in P1. For example, outpatient visits for acute bronchitis were elevated in P2 (RR 1.72; 95% CI 1.36–2.17). For COPD, we found emergency department presentations to be elevated in P1 (RR 1.18; 95% CI 0.67–2.10) and P2 (RR 1.19; 95% CI 0.69–2.03), although without reaching statistical significance. A reverse pattern was seen for COPD outpatient visits, for which an initial nonsignificant deficit in P1 and P2 turned to a significant excess in P3 (RR 1.31; 95% CI 1.03–1.67), although this could also be due, at least in part, to people making up earlier missed appointments.

Cardiovascular index. RRs for the cardiovascular index tended towards null, although an increase was observed in outpatient visits in P2. Although the numbers of encounters with cardiovascular diagnoses were small, the pattern of the cardiovascular index appeared similar to that of total visits. Although based on very small numbers (<20), the few cardiovascular conditions with RR >1 in the context of emergency department presentations and inpatient hospitalizations included dysrhythmia and stroke.

Young children. Relative risks by age group highlight the vulnerable status of young children (Fig 4; S2 Table). In P1, young children aged 0–4 showed significantly elevated emergency department presentations for respiratory diagnoses (RR 1.70; 95% CI 1.32–2.19), asthma (RR 2.36; 95% CI 1.27–4.39), upper respiratory infections (RR 1.77; 95% CI 1.28–2.45), and respiratory symptoms (RR 1.91; 95% CI 1.29–2.82). Although based on small numbers

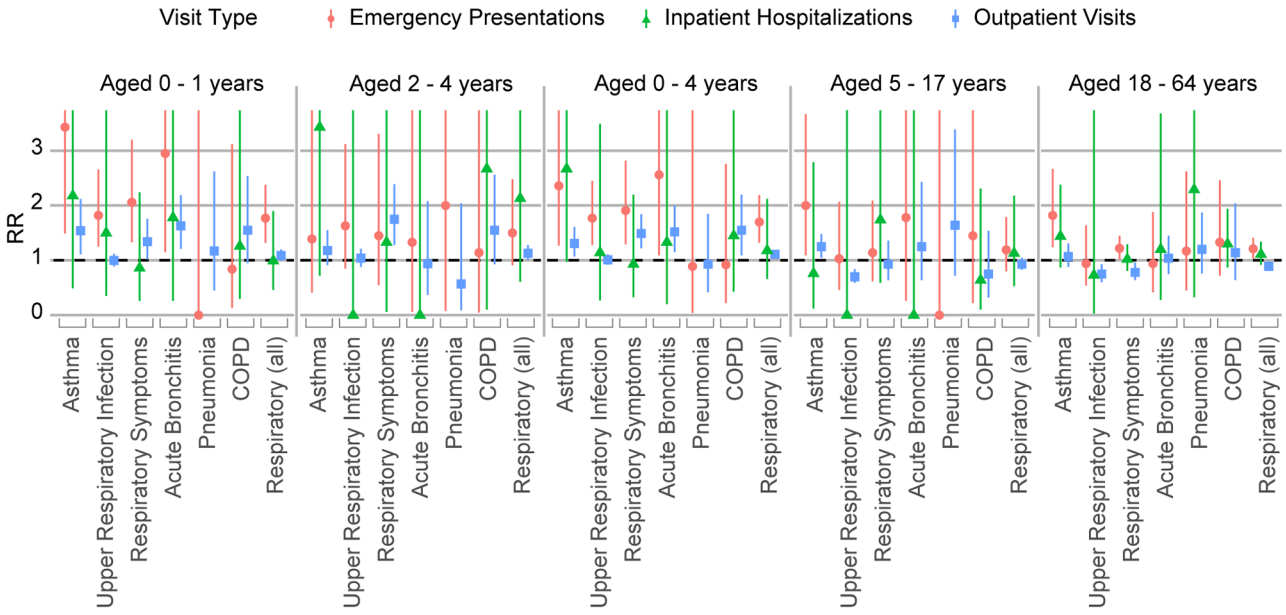

Fig 4. Respiratory healthcare encounters, age-specific results in San Diego County during 2007 fire period. RRs by age group (young children aged 0–1, 2–4, 0–4; older children aged 5–17; and adults under age 65) for the 5-day exposure period starting from October 22 for emergency department presentations, inpatient hospitalizations, and outpatient visits. COPD, chronic obstructive pulmonary disease; RR, rate ratio.

https://doi.org/10.1371/journal.pmed.1002601.g004

(<10), emergency department presentations for acute bronchitis (RR 2.56; 95% CI 1.09–5.54) were elevated in P1 for these young children.

Among children aged 0–4, although there was a deficit in total outpatient visits in P1, outpatient visits for the respiratory index (RR 1.11; 95% CI 1.03–1.19), respiratory symptoms (RR 1.49; 95% CI 1.22–1.84), and acute bronchitis (RR 1.52; 95% CI 1.15–2.00) were significantly elevated. Outpatient visits for pneumonia were also elevated (RR 1.55; 95% CI 1.09–2.20). Although based on very small numbers (<20) and not statistically significant, inpatient hospitalizations for respiratory diagnoses (RR 1.18; 95% CI 0.66–2.12) and for asthma (RR 2.67; 95% CI 0.97–6.53) among children aged 0–4 were elevated in P1.

RRs for children under age 2 (aged 0–1) appeared generally higher than those for young children aged 2–4. The increase in emergency department presentations with respiratory diagnoses appeared greater among children aged 0–1 (RR 1.77; 95% CI 1.32–2.38) than 2–4 (RR 1.50; 95% CI 0.91–2.48). Although based on very small numbers (<10), emergency department presentations for asthma (RR 3.43; 95% CI 1.49–7.38) and acute bronchitis (RR 2.95; 95% CI 1.15–6.85) were elevated among children aged 0–1.

Older children and adults. Unlike younger children, children aged 5–17 in P1 had significantly fewer total encounters across emergency department, inpatient hospital, and outpatient settings versus reference periods. However, for asthma, children aged 5–17 had increased rates of outpatient visits in P1 (RR 1.25; 95% CI 1.05–1.48). Among adults aged 18–64, emergency department presentations for respiratory diagnoses (RR 1.21; 95% CI 1.03–1.41), asthma (RR 1.82; 95% CI 1.24–2.67), and respiratory symptoms (RR 1.22; 95% CI 1.02–1.45) were elevated in P1.

Conditional logistic regression of emergency department presentations for respiratory diagnoses and asthma

In multivariate models adjusted for daily temperature and relative humidity, an increase in the average $PM_{2.5}$ of 10 μg/m^3 for the daily, 48-hour moving, and 72-hour moving averages was

Table 4. Conditional logistic regression of emergency department presentations for respiratory diagnoses and asthma with wildfire $PM_{2.5}$, and ORs adjusted for daily temperature and relative humidity in San Diego County during 2007 wildfires.

	Respiratory Index			Asthma		
$PM_{2.5}$ Measure (10 μg/m^3)	**OR**	**95% Wald CL**	**Wald *p*-value (MLE)**	**OR**	**95% Wald CL**	**Wald *p*-value (MLE)**
Daily average	1.02	1.01–1.04	<0.01	1.03	1.00–1.06	0.03
48-hour moving average	1.03	1.01–1.05	<0.01	1.05	1.02–1.08	<0.01
72-hour moving average	1.04	1.02–1.05	<0.01	1.08	1.04–1.13	<0.01

Abbreviations: CL, confidence limit; MLE, maximum likelihood estimate; OR, odds ratio; $PM_{2.5}$, fine inhalable particles that are 2.5 micrometers and smaller.

https://doi.org/10.1371/journal.pmed.1002601.t004

associated with a 3%, 5%, and 8% increase, respectively, in the likelihood for asthma emergency department presentations, with similar but attenuated increases for respiratory visits (Table 4). ORs were greater when examining moving averages over several days, suggesting that the models were capturing cumulative and lagged effects. Square terms did not reach significance in any of the models, so linear models were selected. We did not find effect modification by age, including after stratifying by sex.

AQI: Respiratory events

Unhealthy AQI levels were associated with increased respiratory conditions in emergency department presentations, adjusting for temperature and relative humidity (Table 5). The AQI models fit best with a 1-day lag compared to same-day– or 2-day–lagged models. The AQI levels "unhealthy for sensitive groups" (OR 1.73; 95% CI 1.18–2.53) and "unhealthy" (OR 1.79; 95% CI 1.30–2.23) both were associated with significantly elevated odds of an emergency presentation the day after exposure versus the AQI level "good." The strongest effect was seen in the same-day model for the highest exposure category, hazardous (OR 2.41; 95% CI 1.39–4.18).

Discussion

By examining multiple respiratory and cardiovascular endpoints across 3 healthcare settings and 3 exposure periods as well as for different age groups, we have compiled a relatively comprehensive view of health events during this significant wildfire complex. While outcomes

Table 5. AQI categories—ORs from conditional logistic regression of respiratory emergency department presentations in San Diego County during 2007 wildfires.

AQI categories $PM_{2.5}$ (μg/m^3)	**OR (95% CI) Same day**	**OR (95% CI) 1-day lag**	**OR (95% CI) 2-day lag**
Good (0–12)	Reference	Reference	Reference
Moderate (12.1–35.4)	1.20 (0.91–1.59)	1.11 (0.84–1.47)	0.80 (0.59–1.08)
Unhealthy for sensitive groups (35.5–55.4)	1.43 (0.96–2.13)	1.73 (1.18–2.53)*	1.51 (1.00–2.28)*
Unhealthy (55.5–150.4)	1.27 (0.97–1.67)	1.79 (1.30–2.23)*	1.50 (1.13–1.98)*
Very unhealthy (150.5–250.4)	1.68 (1.00–2.83)	1.58 (0.93–2.68)	1.87 (1.07–3.27)*
Hazardous (≥250.5)	2.41 (1.39–4.18)*	1.28 (0.70–2.36)	1.74 (1.00–3.03)*
Temperature	1.00 (0.99–1.01)	1.00 (0.99–1.01)	1.00 (0.99–1.00)
Relative humidity	1.01 (1.00–1.01)*	1.01 (1.00–1.01)*	1.01 (1.00–1.01)*
AIC	5,233.2	5,228.9	5,231.8

*Statistically significant (alpha = 0.05).

Abbreviations: AIC, Akaike information criteria; AQI, Air Quality Index; CI, confidence interval; OR, odds ratio; $PM_{2.5}$, fine inhalable particles that are 2.5 micrometers and smaller.

https://doi.org/10.1371/journal.pmed.1002601.t005

such as respiratory conditions were clearly elevated, visits for other outcomes were decreased. These observed results must be viewed in the context of the extensive nature of the fire and the resulting evacuations and other disruptions. These unusual conditions likely altered health-care-seeking behavior; residents may not have accessed healthcare other than for the most urgent conditions. A review of the relationship between the 2007 wildfires and the emergency department of the University of California, San Diego hospital found a 5.8% decrease in admissions during the fires, although the rate of patients with a chief complaint of shortness of breath increased significantly and the rate of patients who left without being seen nearly doubled [37]. Also, an assessment of the 2003 fires in San Diego noted that emergency department presentations initially declined during the fire period, corresponding to days when authorities recommended that students and employees stay home [38].

Our study examined Medi-Cal beneficiaries, a group representing a vulnerable, although fairly substantial, subset of the general population. We would anticipate their response to the health stressor of wildfire smoke to be similar in nature to the general public but possibly increased in magnitude. Asthma, as in other wildfire studies, appeared to be the most sensitive to wildfire smoke exposure [16]. Our findings support a wildfire smoke association with the infectious respiratory outcomes pneumonia, bronchitis, and upper respiratory infections despite inconsistent results from previous studies [16,39]. Airway injury from wildfire smoke exposure could predispose bacterial pneumonia. Previous wildfire studies generally have found positive associations with COPD [16]. Because COPD is a condition more prevalent in the older population, who were excluded from our analysis, this may have limited our ability to study this condition.

Similar to COPD, cardiovascular outcomes are generally more prevalent in older adults, so the absence of this population from our study is relevant here as well. However, our study is not unusual in its null cardiovascular findings for wildfire smoke exposures, despite the scientific relationship between general particulate air pollution and cardiovascular disease [40]. The reasons for this are unclear. The lower prevalence of cardiovascular events in general in comparison with respiratory conditions—along with the possibility that cardiovascular impacts from wildfire smoke may occur at a smaller magnitude than respiratory impacts—may require a larger study to detect an excess. Another factor may be that only certain diagnoses are elevated, and broadly combining all cardiovascular conditions may obscure an association. Moreover, persons with underlying cardiovascular disease may be seen for respiratory rather than cardiovascular conditions (competing diagnoses) during wildfires. Too few studies have examined specific cardiovascular outcomes to have a clear picture of which are related to wildfire exposure [15], although a recent analysis of an extensive California wildfire season provided strong evidence for increased cardiovascular risk [20].

Using sequential exposure periods during and after the peak smoke exposure allowed examination of changes over longer time frames. Studies typically do not detect any increases beyond 3 to 5 lag days. This design allowed us to show some conditions persisting over longer periods of time. Cumulative exposure may be relevant for conditions such as asthma, bronchitis, or pneumonia, which may gradually develop or worsen over time. Inhaled PM may prompt inflammation and alter immune functions, increasing susceptibility to respiratory infections. Also, patients may not seek care until their symptoms become severe.

Our examination of outpatient visits was an exception to the majority of wildfire research studies in the US, which have largely relied on inpatient hospitalization and emergency department data [15]. We noted that patients continued to seek care in outpatient settings while the initial surge in emergency department presentations was declining.

The AQI is a widely used public health tool, yet few wildfire studies have made associations with the AQI categories. The sensitivity of our study population was revealed in its response to

even modestly increased concentrations of PM, as excess adverse health events began to occur at an AQI level designed to represent the first threshold at which susceptible persons are advised to consider limiting their exposure. These results provide evidence for the value of the AQI as a communication tool in conveying health risks of wildfire smoke to the public, especially because the AQI addresses the immediate day, and health events were shown to generally rise with increasing same-day AQI exposure categories.

While children are thought to be more vulnerable to effects of wildfire smoke, the literature has not been conclusive [16]. The mixed results for children may be due to different effects between very young children and older children because null results are often seen in studies that combine all ages or do not include very young children. Wildfire smoke effects among children aged 6 to 18 have been noted in a cohort study of schoolchildren who experienced increased respiratory symptoms [41]. Children's heightened susceptibility to wildfire smoke may be related to their smaller airway size [42]. In our study, this vulnerability was most evident among the very youngest children, aged 0–1, for whom the increase in emergency department presentations during the initial wildfire period (243% increase in asthma) was the highest of any group we evaluated.

Several studies that have stratified on very young children have shown significant associations between increased respiratory admissions and/or visits and wildfire smoke exposures [32,43,44]. However, the magnitude of the association in our Medi-Cal population appears to be greater than what has been found previously in general populations, although results are not directly comparable because methods differ between studies. A study examining 0- to 4-year-olds found a potential 5% increase in the odds of physician visits for asthma, for a 60 $\mu g/m^3$ increase in PM_{10} [41]. Our findings of 236%, 267%, and 131% increases in asthma emergency department presentations, inpatient hospitalizations, and outpatient visits, respectively, suggest a particularly high association among young children (0–4 years). This may be related to underlying vulnerability of the Medi-Cal population. Many factors may contribute to vulnerability, e.g., one study identified increased asthma risks only among children with asthma and obesity [45]. Overall, the very young in our study experienced significantly elevated risks of unusually high magnitude.

The few studies that have examined underlying population vulnerability have tended to use community level analyses that found that various measures of lower SES will confer greater risk from wildfire smoke [15,19,31,46,47]. Although a Canadian study did not, this null finding may be related to Canada's more comprehensive healthcare system [48]. Several studies only detected wildfire health effects in a subgroup with both health and SES vulnerabilities—the indigenous population in Australia—as parallel analyses with the general population failed to detect an effect [48,49]. An analysis of the same San Diego wildfire using Kaiser Permanente health plan members appeared to have possibly lower increases in emergency room visits than our findings, although the analyses are not directly comparable [50]. Our study population of Medi-Cal beneficiaries would encompass multiple susceptibility factors, which may manifest during disasters in ways beyond those directly related to baseline health, e.g., having fewer resources to evacuate, less effective home air filtration, or less control over work schedules.

A limitation of this analysis is that, because Medi-Cal data was used, the study population is not representative of the general population. At the same time, some of the populations most vulnerable to the health effects of wildfires are well-represented among Medi-Cal beneficiaries. For example, over 50% of the state's aged 0–4 population is covered by Medi-Cal [51]. Children are generally more vulnerable to air pollution due to their higher ventilation rate and other factors [52]. A further limitation may be our use of only fee-for-service claims. In 2007, 48% of San Diego Medi-Cal beneficiaries were in managed care [29], and we have no information on differences between the fee-for-service and managed-care populations that could affect

our findings. Medi-Cal data only included a primary and secondary diagnosis code, so any condition not occurring within the first 2 codes would not be identified. There is always a possibility of misclassification in the diagnosis codes or missing data on utilization; however, this should be limited by using medical claims data that are required to be submitted for payment. In addition, the relatively short time frame of this study should reduce any limitations that are a result of changing Medi-Cal eligibility over time.

Our wildfire smoke models allowed geospatially and temporally resolved outputs of particulate concentrations. However, our analysis was based on patient residential zip code, so exposure misclassification would occur because people change location during the day. Wildfire-related disruptions could also have prevented people from seeking care or have caused diversion to facilities outside the area, which would bias our results toward the null. Still, because of the widespread nature of the smoke across much of the populous area of the county, the use of exposure periods defined by sets of wildfire dates appeared to perform relatively well in capturing a broad population risk.

As the population ages and the prevalence of comorbidities increase, the number of persons who are susceptible to wildfire exposures will also grow. Nationally, the proportion of the population over age 65 is anticipated to grow from 15% to 24% by 2060 [53]. Increasing prevalence of diabetes and obesity in the US [54] will also impact cardiovascular health. Unless these trends are reversed, the growing older population will also be less healthy, leading to a greater segment of the population vulnerable to PM from wildfires.

Summary and conclusions

Our study of Medi-Cal beneficiaries identified a significant increase in adverse respiratory events from wildfire smoke exposure and suggested that health risk may persist beyond several immediate days of high–PM exposure. Our findings contribute to growing evidence that, in addition to acute respiratory events such as asthma exacerbation, exposure to wildfire PM may predict infectious conditions, including upper respiratory infections, bronchitis, and pneumonia, which may take longer to manifest. The substantial risk noted among the youngest children is cause for concern because of the potential for long-term harm to children's lung development. The vulnerability of our study population was also shown in its sensitive response to deteriorating air quality because excess adverse health events began to occur at mildly degraded levels of air quality.

The risk of future wildfires to the health of Californians will continue to be shaped by global climate change, as well as the characteristics and anticipated growth of vulnerable subpopulations. The recognition that climate change will increase the burden most severely on disadvantaged communities creates the imperative for public health to help prepare and protect these vulnerable populations.

Supporting information

S1 Protocol. IRB Protocol—Committee for the Protection of Human Subjects, 14-08-1679. IRB, International Review Board.
(PDF)

S1 Table. Respiratory and cardiovascular emergency department presentations, hospital admissions, and outpatient presentations (RRs) for day 1–5, day 6–10, day 11–15 exposure periods; San Diego County, 2007. RR, rate ratio.
(DOCX)

S2 Table. Age-specific RRs for respiratory outcomes for very young children aged 0–1, young children aged 2–4, young children aged 0–4, older children aged 5–17, and adults aged 18–64; October 22–26, 2007, San Diego County. RR, rate ratio.
(DOCX)

S1 Data. Excel dataset of daily average wildfire $PM_{2.5}$ by date and zip code, San Diego County, July 11 through December 31, 2007. $PM_{2.5}$, fine inhalable particles that are 2.5 micrometers and smaller.
(XLSX)

S1 RECORD Checklist. RECORD checklist.
(DOCX)

Acknowledgments

Trang Hoang, Environmental Health Investigations Branch, conducted literature reviews and assisted with development of tables and management of references and final manuscript production.

We would also like to thank the Data and Research Committee of the California Department of Health Care Services, Information and Management Division, for providing data on Medi-Cal claims.

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Writing – review & editing: Justine A. Hutchinson, Jason Vargo, Meredith Milet, Nancy H. F. French, Michael Billmire, Jeffrey Johnson, Sumi Hoshiko.

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Format: Abstract

Environ Res. 2012 Jul;116:44-51. doi: 10.1016/j.envres.2012.04.004. Epub 2012 Apr 26.

Low-level exposure to ambient particulate matter is associated with systemic inflammation in ischemic heart disease patients.

Huttunen K[1], Siponen T, Salonen I, Yli-Tuomi T, Aurela M, Dufva H, Hillamo R, Linkola E, Pekkanen J, Pennanen A, Peters A, Salonen RO, Schneider A, Tiittanen P, Hirvonen MR, Lanki T.

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Abstract

Short-term exposure to ambient air pollution is associated with increased cardiovascular mortality and morbidity. This adverse health effect is suggested to be mediated by inflammatory processes. The purpose of this study was to determine if low levels of **particulate** matter, typical for smaller cities, are associated with acute systemic inflammation. Fifty-two elderly individuals with ischemic heart disease were followed for six months with biweekly clinical visits in the city of Kotka, Finland. Blood samples were collected for the determination of inflammatory markers interleukin (IL)-1β, IL-6, IL-8, IL-12, interferon (IFN)γ, C-reactive protein (CRP), fibrinogen, myeloperoxidase and white blood cell count. Particle number concentration and fine particle (particles with aerodynamic diameters <2.5 μm (PM(2.5))) as well as thoracic particle (particles with aerodynamic diameters <10 μm (PM(10))) mass concentration were measured daily at a fixed outdoor measurement site. Light-absorbance of PM(2.5) filter samples, an indicator of combustion derived particles, was measured with a smoke-stain reflectometer. In addition, personal exposure to PM(2.5) was measured with portable photometers. During the study period, wildfires in Eastern Europe led to a 12-day air pollution episode, which was excluded from the main analyses. Average ambient PM(2.5) concentration was 8.7 μg/m(3). Of the studied pollutants, PM(2.5) and absorbance were most strongly associated with increased levels of inflammatory markers; most notably with C-reactive protein and IL-12 within a few days of exposure. There was also some evidence of an effect of **particulate** air pollution on fibrinogen and myeloperoxidase. The concentration of IL-12 was considerably (227%) higher during than before the forest fire episode. These findings show that even low levels of **particulate** air pollution from urban sources are associated with acute systemic inflammation. Also particles from wildfires may exhibit pro-inflammatory effects.

PMID: 22541720 DOI: 10.1016/j.envres.2012.04.004

[Indexed for MEDLINE]

Publication type, MeSH terms, Substances

LinkOut - more resources

POTASSIUM PERMANGANATE Permanganic acid potassium salt	**ICSC: 0672** **November 2016**
CAS #: 7722-64-7 **UN #: 1490** **EC Number: 231-760-3**	

	ACUTE HAZARDS	PREVENTION	FIRE FIGHTING
FIRE & EXPLOSION	Not combustible but enhances combustion of other substances. Gives off irritating or toxic fumes (or gases) in a fire. Risk of fire and explosion on contact with combustible substances or reducing agents.	NO contact with combustible substances.	In case of fire in the surroundings, use appropriate extinguishing media.

PREVENT DISPERSION OF DUST! STRICT HYGIENE! IN ALL CASES CONSULT A DOCTOR!

	SYMPTOMS	PREVENTION	FIRST AID
Inhalation	Burning sensation. Cough. Sore throat. Shortness of breath. Laboured breathing. Symptoms may be delayed.	Avoid inhalation of dust. Use local exhaust or breathing protection.	Fresh air. Half-upright position. Artificial respiration may be needed. Refer immediately for medical attention.
Skin	Redness. Skin burns. Pain.	Protective gloves. Protective clothing.	Wear protective gloves when administering first aid. First rinse with plenty of water for at least 15 minutes, then remove contaminated clothes and rinse again. Refer for medical attention .
Eyes	Redness. Pain. Severe burns.	Wear face shield or eye protection in combination with breathing protection.	Rinse with plenty of water for several minutes (remove contact lenses if easily possible). Refer immediately for medical attention.
Ingestion	Burning sensation. Abdominal pain. Diarrhoea. Nausea. Vomiting. Shock or collapse.	Do not eat, drink, or smoke during work.	Rinse mouth. If within a few minutes after ingestion, one small glass of water may be given to drink. Do NOT induce vomiting. Refer immediately for medical attention.

SPILLAGE DISPOSAL

Personal protection: chemical protection suit including self-contained breathing apparatus. Do NOT let this chemical enter the environment. Sweep spilled substance into covered containers. Carefully collect remainder. Then store and dispose of according to local regulations. Do NOT absorb in saw-dust or other combustible absorbents.

STORAGE

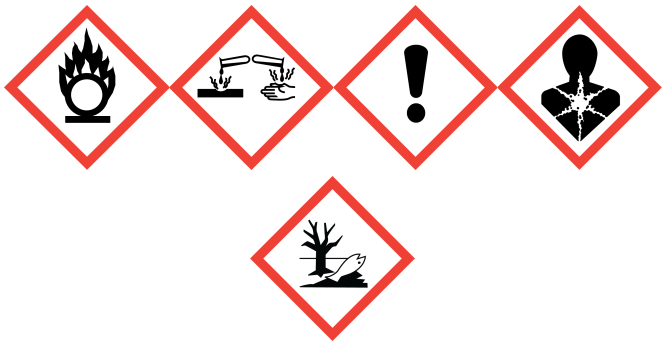
Separated from combustible substances, reducing agents and powdered metals. Well closed. Provision to contain effluent from fire extinguishing. Store in an area without drain or sewer access.

PACKAGING

Marine pollutant.

CLASSIFICATION & LABELLING

According to UN GHS Criteria



DANGER

May intensify fire; oxidizer
Harmful if swallowed

	Causes severe skin burns and eye damage Suspected of damaging fertility or the unborn child Very toxic to aquatic life **Transportation** **UN Classification** UN Hazard Class: 5.1; UN Pack Group: II

Prepared by an international group of experts on behalf of ILO and WHO,
with the financial assistance of the European Commission.

POTASSIUM PERMANGANATE **ICSC: 0672**

PHYSICAL & CHEMICAL INFORMATION

Physical State; Appearance
DARK PURPLE CRYSTALS.

Physical dangers

Chemical dangers
Decomposes on heating. This produces toxic gases and irritating fumes. The substance is a strong oxidant. It reacts with combustible and reducing materials. This generates fire and explosion hazard. Reacts violently with powdered metals. This generates fire hazard.

Formula: $KMnO_4$
Molecular mass: 158
Decomposes at 240°C
Density: 2.7 g/cm³
Solubility in water, g/100ml at 20°C: 6.4 (moderate)
Vapour pressure at 20°C: negligible
Octanol/water partition coefficient as log Pow: 1.73 (calculated)

EXPOSURE & HEALTH EFFECTS

Routes of exposure
Serious local effects by all routes of exposure.

Effects of short-term exposure
The substance is corrosive to the eyes, skin and respiratory tract. Corrosive on ingestion. Inhalation of dust may cause lung oedema, but only after initial corrosive effects on eyes and/or airways have become manifest. The effects may be delayed. Medical observation is indicated.

Inhalation risk
A harmful concentration of airborne particles can be reached quickly when dispersed, especially if powdered.

Effects of long-term or repeated exposure
The substance may have effects on the lungs. This may result in bronchitis and pneumonia. Animal tests show that this substance possibly causes toxicity to human reproduction or development.

OCCUPATIONAL EXPOSURE LIMITS

TLV: (as Mn): 0.2 mg/m^3, as TWA.

EU-OEL: (as Mn, inhalable fraction): 0.2 mg/m^3 as TWA.

EU-OEL: (as Mn, respirable fraction): 0.05 mg/m^3 as TWA.

MAK: (as Mn, inhalable fraction): 0.2 mg/m^3; (as Mn, respirable fraction): 0.02 mg/m^3; peak limitation category: II(1); pregnancy risk group: C; (DFG 2016)

ENVIRONMENT

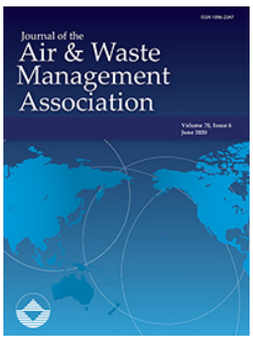
The substance is very toxic to aquatic organisms.

NOTES

Rinse contaminated clothing with plenty of water because of fire hazard.

ADDITIONAL INFORMATION

EC Classification
Symbol: O, Xn, N; R: 8-22-50/53; S: (2)-60-61



Journal of the Air & Waste Management Association

ISSN: (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/uawm20

Wildfire and prescribed burning impacts on air quality in the United States

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To cite this article: Daniel A. Jaffe, Susan M. O'Neill, Narasimhan K. Larkin, Amara L. Holder, David L. Peterson, Jessica E. Halofsky & Ana G. Rappold (2020) Wildfire and prescribed burning impacts on air quality in the United States, Journal of the Air & Waste Management Association, 70:6, 583-615, DOI: 10.1080/10962247.2020.1749731

To link to this article: https://doi.org/10.1080/10962247.2020.1749731

JOURNAL OF THE AIR & WASTE MANAGEMENT ASSOCIATION
2020, VOL. 70, NO. 6, 583–615
https://doi.org/10.1080/10962247.2020.1749731

Taylor & Francis
Taylor & Francis Group

2020 ANNUAL A&WMA CRITICAL REVIEW

Wildfire and prescribed burning impacts on air quality in the United States

Daniel A. Jaffe [a], Susan M. O'Neill[b], Narasimhan K. Larkin[b], Amara L. Holder[c], David L. Peterson[d], Jessica E. Halofsky[d], and Ana G. Rappold[e]

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ABSTRACT

Air quality impacts from wildfires have been dramatic in recent years, with millions of people exposed to elevated and sometimes hazardous fine particulate matter ($PM_{2.5}$) concentrations for extended periods. Fires emit particulate matter (PM) and gaseous compounds that can negatively impact human health and reduce visibility. While the overall trend in U.S. air quality has been improving for decades, largely due to implementation of the Clean Air Act, seasonal wildfires threaten to undo this in some regions of the United States. Our understanding of the health effects of smoke is growing with regard to respiratory and cardiovascular consequences and mortality. The costs of these health outcomes can exceed the billions already spent on wildfire suppression. In this critical review, we examine each of the processes that influence wildland fires and the effects of fires, including the natural role of wildland fire, forest management, ignitions, emissions, transport, chemistry, and human health impacts. We highlight key data gaps and examine the complexity and scope and scale of fire occurrence, estimated emissions, and resulting effects on regional air quality across the United States. The goal is to clarify which areas are well understood and which need more study. We conclude with a set of recommendations for future research.

Implications: In the recent decade the area of wildfires in the United States has increased dramatically and the resulting smoke has exposed millions of people to unhealthy air quality. In this critical review we examine the key factors and impacts from fires including natural role of wildland fire, forest management, ignitions, emissions, transport, chemistry and human health.

Introduction

Large wildfires in the United States are becoming increasingly common, and smoke from these fires is a national concern. Figure 1 shows impacts from large wildfires that burned in the western U.S. in summer of 2017. These fires generated smoke plumes that were transported across North America, resulting in measured $PM_{2.5}$ (particulate matter with aerodynamic diameter ≤2.5 micrometers) concentrations that reached Unhealthy to Hazardous levels in many areas, based on National Ambient Air Quality Standard definitions.

Daniel A. Jaffe

Fires emit PM directly along with hundreds of gaseous compounds. The gaseous compounds include nitrogen oxides (NOx), carbon monoxide (CO), methane (CH_4), and hundreds of volatile organic compounds (VOCs), including a large number of oxygenated VOCs (OVOCs). This chemical complexity makes wildfire smoke very different from typical industrial pollution. A key challenge for understanding fire impacts on air quality is the large variability from fire to fire in both the quantity and composition of emissions. Emissions can vary as a function of the amount and type of fuel (Prichard et al. 2019a), meteorology, and burning conditions. These variations give rise to large uncertainties in the emissions from individual fires (Larkin et al. 2012). Once emitted, wildfire smoke undergoes chemical transformations in the atmosphere, which alters the mix of compounds and generates secondary pollutants, such as ozone (O_3) and secondary organic aerosol (SOA).

Wildland fire is an essential ecological process integral to shaping most North American ecosystems. Wildland ecosystems, broadly, include both forests and rangelands, which are distributed across the

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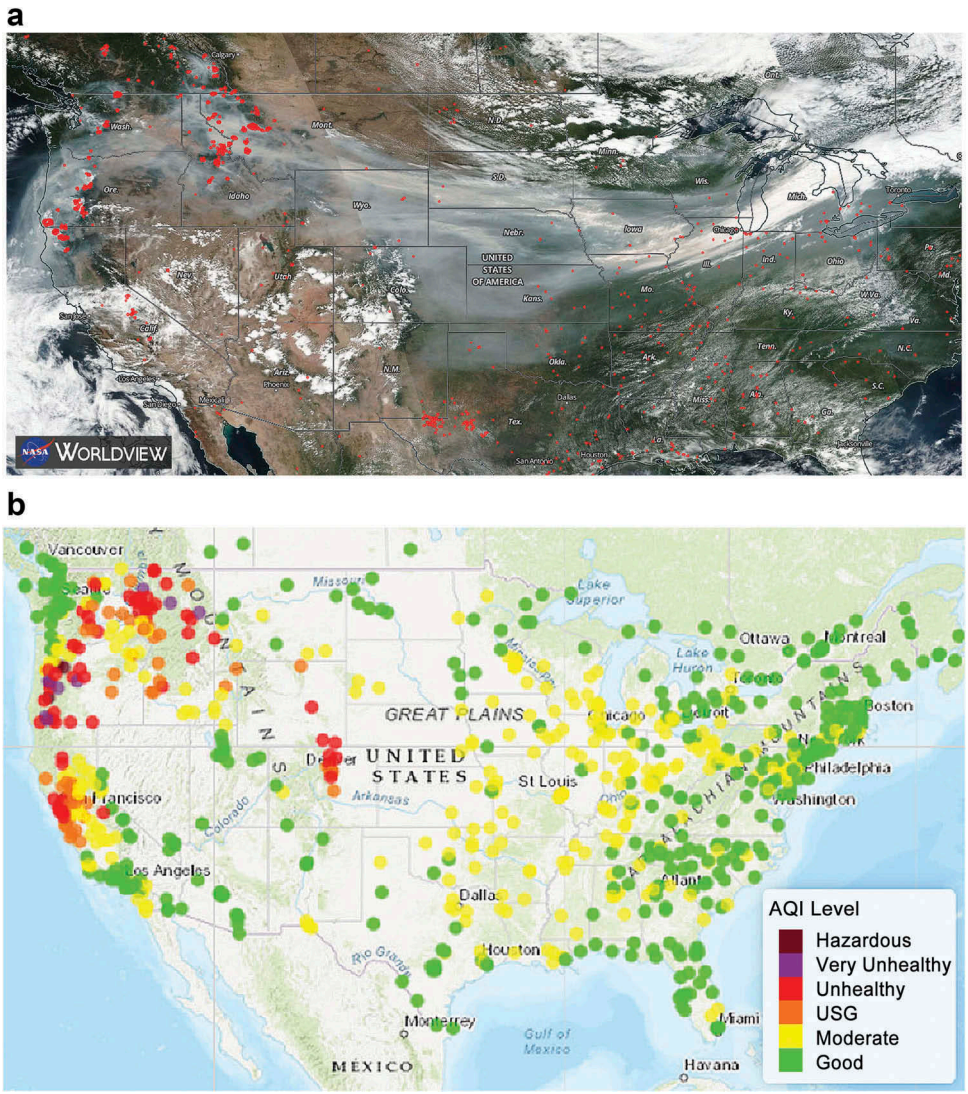

Figure 1. (A) (top) Observed smoke on September 4, 2017. (Top) NASA Worldview (https://worldview.earthdata.nasa.gov/) image showing fire hotspot detections from the VIIRS and MODIS satellite instruments, along with visible satellite imagery from the VIIRS instrument between 1200–1400 local time. Bright white areas are clouds; grayer areas are smoke. (B) (Bottom) 24-hour average $PM_{2.5}$, shown as the corresponding Air Quality Index (AQI) level category colors, based on surface PM sensors collected in the EPA's AirNow system (https://www.airnow.gov/).

spectrum of rural to urban environments; forests cover 360 million hectares (ha) and rangelands cover 308 million ha, 33% and 29% of land in the United States, respectively. The scope and scale of fire within these environments vary widely, with consequences for both emissions and effects of smoke.

Figure 2 shows the progression of fire in the U.S. throughout the year 2017 as seen by satellite detections. In winter, fires are found mainly in the Southeast, typically as prescribed low-intensity understory burns to maintain longleaf pine and other forest savanna systems. As spring approaches, fire detections move north, with increased prescribed fire activity across the central U.S. in many rangelands. In summer, wildfire season peaks, especially in the western U.S. Late fall can also be a time of many fires in California and the Southeast. This progression of fire throughout the seasons and ecosystems across the U.S. has implications for the overall quantity and specific chemistry of the emitted smoke.

Humans have a profound influence on both the use and suppression of wildland fire. It is difficult to separate human influence from the natural occurrence of fire on the landscape (Pyne 1997). For example, Native Americans used fire as a tool for agriculture and to manage wildlife habitat and hunting grounds. These frequent, low-intensity fires may have substantially affected the landscape across the U.S, but modern management practices, especially fire suppression efforts, probably have been more important in

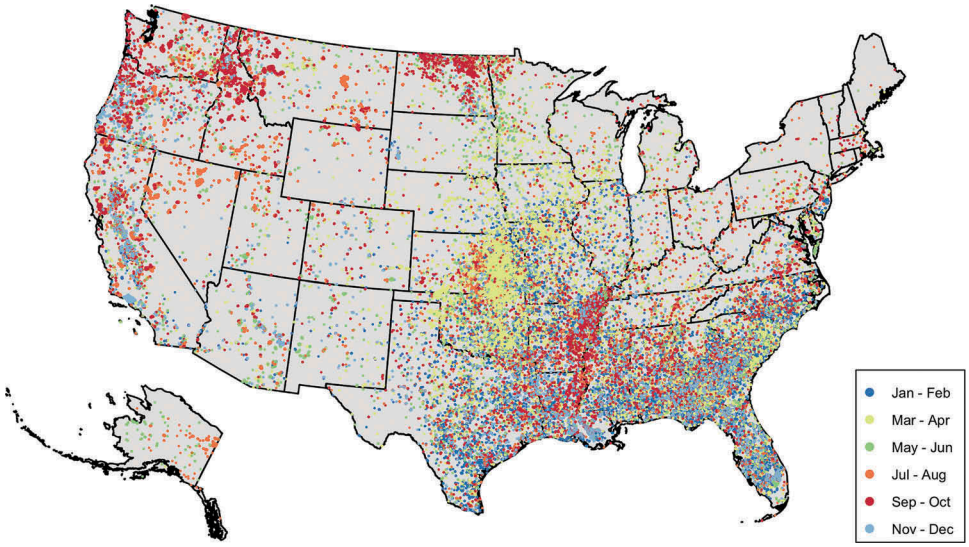

Figure 2. Progression of fires throughout the year using 2017 MODIS hotspot fire detections.
Source: U.S. Forest Service.

changing the forest structure (Ryan, Knapp, and Varner 2013). The result through the 1900 s has been less fire on the landscape than in pre-settlement times (Leenhouts 1998), and therefore, likely less smoke in the air (Brown and Bradshaw 1994). Recent episodes of smoke across extensive landscapes, driven by large wildfires, may therefore to some extent be a return to pre-suppression levels.

A number of studies have documented the importance of climate change on the increasing frequency and size of fires in the western U.S. Large fires are increasing in the West (Dennison et al. 2014). Rising temperatures affect fuel aridity and the length of the fire season (Abatzoglou and Williams 2016), the amount of snow, the timing of snowmelt (Westerling 2016), and relative humidity, which has been related to the increasing trend of area burned in California (Williams et al. 2019). However, the relationship between climate and human influences is complex and not all fires should be attributed to climate change. For example, Mass and Ovens (2019) suggested that the 2017 Wine Country fires in northern California likely had little influence from recent climate change. Littell et al. (2009) found that the effect of climate change on area burned can vary with the ecosystem and fuels.

Complicating the role of climate change are the effects of invasive species (Fusco et al. 2019) and direct human ignitions. These ignitions are estimated to be responsible for over 80% of wildfires, by number, across the U.S., excluding prescribed and management fires (Balch et al. 2017). Human ignition sources include vehicles, construction equipment, power lines, fireworks, camping, arson, and others. However, in the Intermountain West, lightning appears to be the dominant cause for ignitions (Balch et al. 2017). Human ignitions have expanded the length of the wildfire season, but climate and human presence are interrelated factors (Syphard et al. 2017).

Crop-residue burning is common across the U.S. to remove or reduce biomass. Prescribed burning – planned ignition in accordance with applicable laws, policies, and regulations to meet specific objectives (NWCG 2018) – also occurs for multiple reasons, including to reduce fuel loading and ecosystem health. Both crop-residue fires and prescribed burning tend to occur in the non-summer months, and, depending upon the state, they may be permitted under a smoke management program to ensure that smoke exposure will not exceed air quality standards or affect sensitive populations.

Although 98% of wildfires are suppressed before reaching 120 ha (Calkin et al. 2005), the annual area burned by wildfires is increasing. Figure 3 shows the large interannual variability in wildfire area burned and the substantial increase in area burned and federal suppression costs between 1999 and 2018. In those two decades, wildland fires burned an average of 2.8 million ha per year, which is more than double the annual amount that burned in the two decades before 1998 (National Interagency Fire Center [NIFC], 2019). This comparison indicates that a small number of fires are expanding in size and greatly increasing the area burned.

Although the area burned globally appears to be declining (Andela et al. 2017), in the U.S. the area burned by wildfires is on the rise, and federal costs of wildfire suppression have risen substantially along with area burned. In 2018, federal suppression costs were the

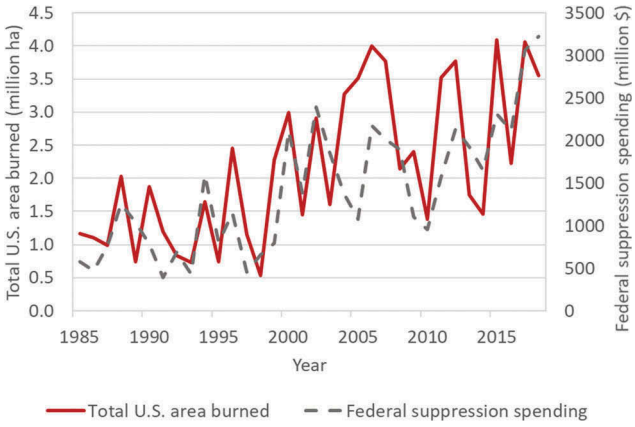

Figure 3. Total U.S. wildfire area burned (ha) and federal suppression costs for 1985–2018 scaled to constant (2016) U.S. dollars. Trends for both wildfire area burned and suppression indicate about a four-fold increase over a 30-year period. Source: National Interagency Fire Center (NIFC) (2019).

highest ever, at over 3 USD billion (NIFC, 2019). As towns and cities have grown and spread deeper into the wildlands, creating a larger wildland-urban interface (WUI), an increasing share of resources for forest management and firefighting effort has gone toward protecting human developments.

In recent years, smoke from large fires has caused extreme concentrations of $PM_{2.5}$ and O_3, especially in the western U.S. (Gong et al. 2017; Laing and Jaffe 2019; Mass and Ovens 2019). The highest $PM_{2.5}$ concentrations ever observed in many western cities were seen in the summers of 2017 or 2018, due to wildfires, with some daily $PM_{2.5}$ values of over 500 μg/m^3 (see box: The relationship between fire activity and smoke, 2004–2018). The U.S. has made steady progress in reducing air pollution from industrial and vehicle emissions, but the recent increase in wildland fires has slowed or even reversed this progress in some parts of the country (McClure and Jaffe 2018).

Although much of the recent attention on wildfires has focused on the western U.S., large fires also burn in the southeastern U.S. In November 2016, large wildland fires burned in Tennessee, North Carolina, South Carolina, and Georgia, generating $PM_{2.5}$ concentrations exceeding 100 μg/m^3 in many cities. The smoke and elevated $PM_{2.5}$ persisted across the region for weeks. Prescribed and crop-residue burning are also common in the Southeast, in some cases with consequences to health (Huang et al. 2019).

As smoke plumes move over populated areas, they can elevate $PM_{2.5}$ and/or O_3 levels over health standards. Large and extended wildfires can be associated with respiratory issues and premature mortality (e.g., Liu et al. 2015a; Reid et al. 2019). The plumes can affect regions directly and/or mix with other urban pollutants. In the U.S., the Clean Air Act of 1963 was enacted to protect public health and welfare. In 1970 the U.S. Environmental Protection Agency (EPA) established the National Ambient Air Quality Standards (NAAQS) for six criteria pollutants. The criteria pollutants most relevant to wildland fire emissions are $PM_{2.5}$, O_3, and CO. For daily average $PM_{2.5}$, the current primary standard is 35 μg/m^3 at the 98th percentile, averaged over three years. For O_3, the current primary standard is 0.070 ppm for the annual fourth-highest daily maximum 8-hour concentration (MDA8), averaged over three years. For CO, the current primary standards are 9 ppm for an 8-hour averaging time, and 35 ppm for a one-hour averaging time, not to be exceeded more than once per year. Although CO from fires is rarely a concern to the public, it can affect wildland firefighters, and recent work analyzes exposure in terms of National Institute of Occupational Safety and Health (NIOSH) standards (Henn et al. 2019). Smoke plumes from wildland fires have caused substantial exceedances of the EPA standards for both $PM_{2.5}$ and O_3, but a state may try to exclude these data from regulatory consideration under the exceptional events rule (See Section 8, Regulatory context for air quality management, for further discussion).

The EPA's National Emission Inventory (NEI) is generated every three years and includes all significant categories of emissions for the major pollutants. The 2011 and 2014 NEI show that wildland fire emissions represented approximately 32% of the total primary $PM_{2.5}$ emissions in the U.S. (Larkin et al. 2020). Liu et al. (2017a) estimated that, in 2011–2015, fires in 11 western states emitted on average twice as much primary $PM_{2.5}$, compared to the annual emissions from all industrial sources in the region. Although prescribed burning remains relatively constant interannually (5.03 million ha in 2011, 4.42 million ha in 2014), wildfires are subject to large interannual variability (4.32 million ha in 2011, 1.72 million ha in 2014) (Larkin et al. 2020). Furthermore, emissions are not necessarily proportional to area burned. The fuel type and amount of fuel consumed are large drivers in determining emissions. For example, in 2011, both Minnesota and North Carolina had relatively moderate area burned, but some of the largest emissions of $PM_{2.5}$ were caused by consumption of deep organic fuels (Larkin et al. 2020). Liu et al. (2017a) found that $PM_{2.5}$ emissions from prescribed burning was lower per kg of fuel consumed.

Most smoke in the U.S. is associated with wildland fires in the U.S., but fires outside the country can also have

major impacts on U.S. air quality. In 2017, high $PM_{2.5}$ in the Pacific Northwest was associated with large fires in British Columbia (Laing and Jaffe 2019). These same fires were associated with smoke transport to Europe and strong thunderstorm-pyrocumulonimbus activity, which injected smoke into the stratosphere (Baars et al. 2019). Large fires in Quebec have significantly affected air quality in the northeast U.S. (DeBell et al. 2004), fires from Mexico and Central American can impact Texas (Kaulfus et al. 2017; Mendoza et al. 2005), and even large fires in Siberia can affect surface air quality in the U.S. (Jaffe et al. 2004; Teakles et al. 2017).

In this review, we examine the current capabilities for observing and quantifying smoke, what is known about wildland fire emissions, the development of models for smoke plumes and transport, and the chemical makeup and transformations of smoke. We also examine current understanding of modeling smoke impacts, understanding of effects of smoke on health, and the state of air quality regulations involving smoke, all with an emphasis on the continental U.S. We conclude by looking at future U.S. national fire patterns and trends and suggest a set of recommendations for future research.

Observations of smoke

In-situ observations

Ground-based smoke impacts are observed by a combination of established permanent in-situ air quality monitoring networks, temporarily deployed monitors and, most recently, low-cost sensor networks. Permanent in-situ measurements include monitoring networks maintained by federal, state, and tribal agencies. The agency monitors use a mix of Federal Reference Methods (FRMs) or Federal Equivalent Methods (FEMs) and other sampling and analysis approaches. Data are generally provided to the EPA AirNow system (for access in near-real time) and the AQS system (for QA/QC'd data). The Interagency Monitoring of PROtected Visual Environments (IMPROVE) network is a permanent network of monitors that measure the major chemical composition of $PM_{2.5}$ every three days (24-hour averages) at remote locations across the U.S. The EPA Chemical Speciation Network (CSN) provides a similar suite of measurements as the IMPROVE system at urban locations. Figure 1 shows an example of $PM_{2.5}$ data from the regulatory network and the relationship to fires.

In addition to the permanent networks, several agencies across the U.S. now deploy ground-based $PM_{2.5}$ monitors to under-sampled areas where smoke impacts are large or anticipated to be so. While not regulatory monitors, these temporary monitors can substantially increase the smoke observations available in affected areas. For example, the U.S. Forest Service's Interagency Wildland Fire Air Quality Response Program (IWFAQRP; https://wildlandfiresmoke.net) maintains and deploys a combination of MetOne Environmental Beta-Attenuation Mass monitors (E-BAMs) and E-Sampler monitors (using light scattering) on both prescribed fires and wildfire incidents, with over 100 such deployments per year. Other agencies, such as the California Air Resources Board, also maintain and deploy such monitors as needed. Deployments are generally made to town and city locations based on need and expected level of impacts and are prioritized where other air quality monitoring is not available. These monitors have found much higher concentrations and a greater frequency of days with $PM_{2.5}$ exceeding 35 ug/m^3, compared to the permanent monitoring networks (Larkin 2019). This pattern suggests that current permanent monitors lack the spatial distribution to fully represent the overall human exposure to wildfire smoke, especially in rural areas.

Increasingly, low-cost sensors are being used by households and businesses concerned with air quality, as well as agencies concerned with cost effectively expanding coverage (Morawska et al. 2018). These sensors, mostly based on light scattering, are less accurate, but they can be highly correlated with regulatory monitors and can be adjusted to regulatory instrument calibrations for typical aerosols to improve accuracy (Mehadi et al. 2019). Reliability, maintenance, and ambient relative humidity concerns are larger than with more systematically setup and maintained permanent networks, and this can cause large biases (e.g., Feenstra et al. 2019; Li et al. 2020; Manibusan and Mainelis 2020; Singer and Delp 2018). Unfortunately, the public usually does not recognize these issues and can misinterpret the results. The number of available low-cost sensors does provide enhanced spatial coverage. For example, the most common such sensor, made by PurpleAir, now has over 4,000 units deployed within the continental U.S. (PurpleAir 2019), compared with approximately 1,100 publicly accessible permanent in-situ $PM_{2.5}$ monitors available in the EPA's AirNow database. The net result is that, in large portions of the continental U.S., the only nearby measurements are from low-cost sensors.

Satellite sensors and products

A wide array of satellite-borne instruments rely on spectral measurements of infrared, visible, or UV light

to detect aerosol plumes and some gaseous pollutants. These instruments provide an important and unique view of fires and their associated air quality impacts. Polar-orbiting satellites can view nearly all of the U.S. every day, at least once per day, whereas geostationary satellites get near-continuous coverage during the daytime, but at lower spatial resolution. Satellite measurements also have specific biases and issues that limit their use. Satellites preferentially detect large, energetic fires and their plumes, but they may miss smaller, less energetic, or obscured fires, resulting in a systemic bias. For air quality, satellite products can provide information where no other observations are available, but most satellite instruments cannot distinguish between impacts at the ground versus impacts aloft. Even with these issues, satellite fire detections are critical inputs for emissions inventories and are used in both real-time air quality forecasts and, retrospectively, for model evaluation and improvement.

Satellite fire detections

Satellite fire detection can be based on thermal anomalies or vegetation changes (e.g., Chuvieco and Martin 1994; Hao and Larkin 2014; Roy, Boschetti, and Smith 2013). Thermal anomaly detection uses the measured energy received across multiple wavelengths to determine both a temperature and a radiative energy per imaged pixel. When the detected temperature and amount of energy is above a non-fire threshold, these are flagged as fire detections, also referred to as hotspot detections. The radiant energy received is used to calculate the fire radiative power (FRP) (instantaneous reading) and fire radiant energy (FRE) (time-integrated measurement) of the pixel. This is the most common satellite fire detection scheme, and it is used by a number of satellite platforms, including the following polar-orbiting and geostationary platforms:

(1) The older Advanced Very-High-Resolution Radiometer (AVHRR; Flasse and Ceccato 1996; Lee and Tag 1990) has been used on various National Oceanic and Atmospheric Administration (NOAA) polar-orbiting satellites since 1978.
(2) The Moderate Resolution Imaging Spectroradiometer (MODIS; Justice et al. 2002, 2011) is carried by NASA's polar-orbiting Terra and Aqua platforms, launched in 1999 and 2002, respectively.
(3) The newer Visible Infrared Imaging Radiometer Suite (VIIRS; Koltunov et al. 2016; Schroeder et al. 2014) is carried aboard the NASA/NOAA Joint Polar-orbiting Satellite Systems (JPSS) satellites. These satellites currently are the Suomi National Polar-Orbiting Partnership (NPP), launched in 2001, and the NOAA-20/JPSS-1, launched in 2017; three additional satellites are planned.
(4) The Advanced Baseline Imager (ABI; Schmit et al. 2017, 2005, 2008) and other radiometers are carried on the various NOAA Geostationary Orbiting Environmental Satellite (GOES) series of geostationary satellites. These include the recently deployed GOES-16 and GOES-17 satellites (Schmidt 2020), launched in 2016 and 2018, respectively.

Polar-orbiting platforms provide once- or twice-daily coverage of an entire region, while geostationary platforms can provide near-continuous measurements. For example, GOES-16 and GOES-17 can image the continental U.S. every five minutes and provide a rapid update of a specific region every minute.

The disadvantage of thermal anomaly detection is that smaller and/or obscured fires (e.g., by clouds) will often be missed. A high percentage of prescribed fires are purposely designed to burn at low intensity and/or as understory burns; consequently, these are harder for satellites to detect (e.g., Nowell et al. 2018). That satellites miss a larger portion of prescribed fires compared to wildfires has been confirmed by comparisons with ground-based prescribed burning databases (Larkin et al. 2020; Larkin, Raffuse, and Strand 2014). Polar-orbiting satellites also need the fire to be active at the time of the satellite overpass, which may not correspond with the period of most active fire behavior. The Terra and Aqua polar-orbiting satellites have daytime overpass times of 10:30 am and 1:30 pm local time, generally ahead of the peak fire energetics that occur later in the afternoon when temperatures are higher, relative humidities are lower, and the mixed layer is more fully developed. Overpass timing can cause even larger fires to be missed if they are short in duration, a problem typical of quick-burning fuels such as grasslands. Geostationary satellites have the advantage of near-continuous daytime coverage, but, due in part to their higher orbits, the resolution (pixel size) reflects larger ground areas compared to polar-orbiting systems, thereby limiting their detection capabilities. Figure 4 shows an example of how different satellite systems can see the same fire, showing the GOES-16 and VIIRS hotspot detections for one day of the 2019 Kincade fire in California.

The NOAA Hazard Mapping System (HMS; NOAA, 2019; Ruminski et al. 2006; Schroeder et al. 2008) is an

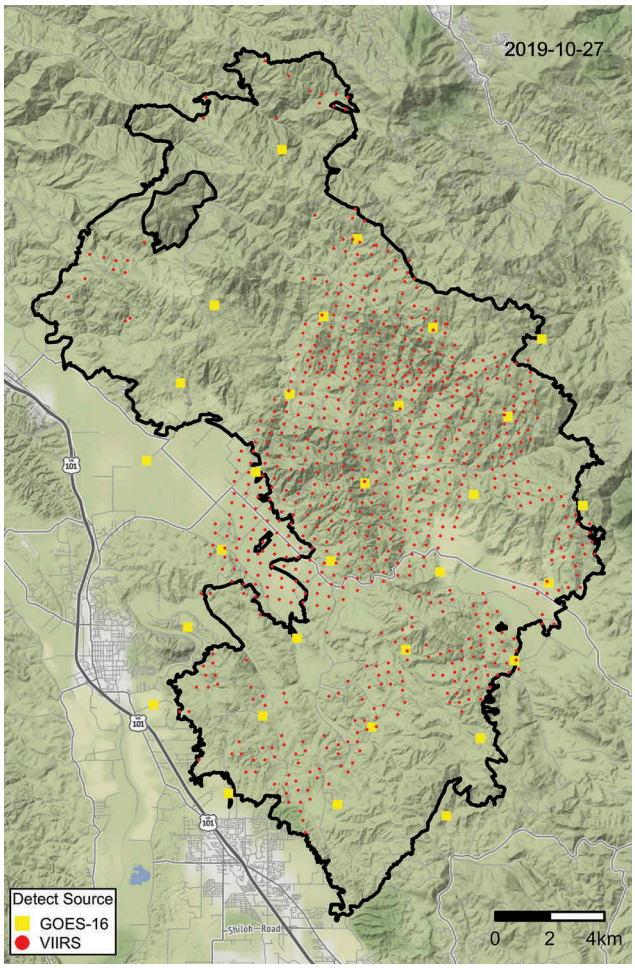

Figure 4. Satellite detections of the Kincade fire in northern California on October 27, 2019 by Geostationary Orbiting Environmental Satellite (GOES) and the polar-orbiting Visible Infrared Imaging Radiometer Suite (VIIRS). Hotspot detections by each are shown at the center points of the sensor pixels (yellow squares: GOES-16; red circles: VIIRS). Black outline: final fire perimeter. The VIIRS detections provide a higher resolution detection (~375 m), but only during overpasses. The geostationary GOES-16 provides a continuous observation but at a lower resolution (~2 km). The size of squares and circles is illustrative and not related to hotspot detection strength or size. Data sources: GOES and VIIRS detections based on NOAA Hazard Mapping System–collected detections; perimeter based on GeoMac data. Image source: U.S. Forest Service.

operational system that aggregates fire and smoke information from across various satellite systems and does quality control to remove identified false detections. Additionally, obscured fires that are not detected are added back in where visible imagery allows for geolocating the source of the plume. HMS fire detections are gridded onto a 1-km grid and are commonly used in smoke forecasting systems (O'Neill et al. 2008).

Burned area also can be detected by comparing satellite imagery on successive passes and identifying areas of vegetative change that are likely due to fire. This is typically done using LANDSAT (Tucker, Grant, and Dykstra 2004), AVHRR, or MODIS imagery. The result is an overall burned area or burn scar estimation (e.g., Kasischke and French 1995; Koutsias and Karteris 1998; Roy et al. 1999). Active hotspot detection can also be folded into the burn scar estimation (Giglio et al. 2009). The amount of change between overpasses at a given pixel reflects the change in biomass due to the fire. This measure is used by the U.S. Forest Service Monitoring Trends in Burn Severity (Eidenshink et al. 2007) project. Although such systems can provide highly detailed maps of specific burns, the process is generally applied only to larger burns, and in specific cases it can also have issues such as extremely large or small area estimations (e.g., Drury et al. 2014). The largest limitation for air quality purposes, however, is that such systems are based on 8-day LANDSAT 30-m resolution imagery, and so are too delayed for air quality forecasting purposes. MODIS-based products are available faster but with lower resolution (approximate 1-km resolution).

Satellite air quality measurements

Satellites provide a number of measurements relevant to air quality (Kahn 2020). The simplest is smoke extent polygons, such as those created operationally by the NOAA HMS (Ruminski et al. 2006; Schroeder et al. 2008). HMS smoke plumes extents are often used as a marker of being in a smoke plume but do not necessarily represent ground smoke impacts (Buysse et al. 2019; Kaulfus et al. 2017). For example, Figure 5 shows the HMS plumes extents for 11/8/2018 for the Camp wildfire (left panel) and surface measurements of 1-hr average $PM_{2.5}$ concentrations overlaid with the visible smoke plume from GOES-16 (right panel). Note that HMS vertically integrated smoke plumes extents may not represent ground-level concentrations: good air quality conditions at the surface (i.e., green) are present in some locations under the thickest visible smoke. Conversely, many monitors show poor air quality conditions (i.e., red) at locations where the visible satellite plume is much less dense. This comparison highlights how the satellite top-down view of the earth may not represent what we experience at the surface. Buysse et al. (2019), for example, found that surface $PM_{2.5}$ was enhanced on 30–80% of days with overhead HMS smoke plumes across 18 western U.S. cities. Locations closer to fire sources are more likely to have ground impacts when inside an identified smoke plume perimeter. In this way, satellite-derived smoke plume extent is a weak marker of ground impacts. However, the shape of the HMS plumes can be used to connect

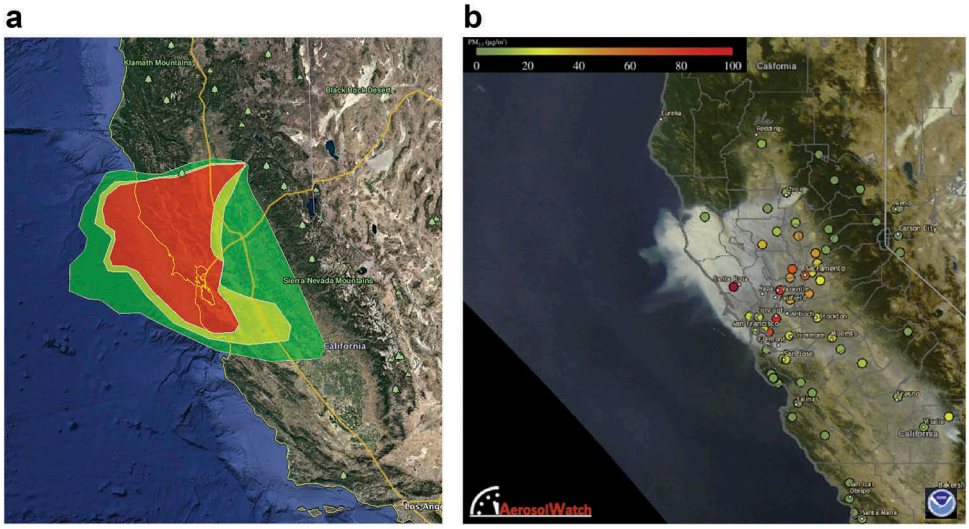

Figure 5. Camp wildfire, northern California, November 8, 2018. A NOAA HMS smoke plume at 12:30:00 PST. Colors are qualitative representation of smoke intensity (green: light, yellow: medium, red: heavy). (b) Visible satellite imagery from GOES-16 overlaid with surface measurements of 1-hr average $PM_{2.5}$ concentrations at 13:02:00 PST. Colors for the $PM_{2.5}$ data are associated with the AQI scale (see Figure 1). The right figure is from the NOAA Aerosol Watch program (https://www.star.nesdis.noaa.gov/smcd/spb/aq/AerosolWatch/).

identified impacts back to fire sources (e.g., Brey et al. 2018) and to validate smoke forecasts (Rolph et al. 2009).

Other smoke characteristics are available from satellites (e.g., Paugam et al. 2016). Plume top height is available from the Multi-angle Imaging SpectroRadiometer (MISR; Diner et al. 1998) instrument aboard the NASA polar-orbiting Earth Observing System (EOS) Terra satellite. MISR uses stereographic imagery to calculate plume top height. This system has been used to identify and evaluate overall fire plume top heights (Val Martin, Kahn, and Tosca 2018) since 1999 and provides the longest history of satellite-observed plume heights. Beyond providing plume top measurements, the vertical structure of plumes can be measured with the Cloud-Aerosol Lidar with Orthogonal Polarization (CALIOP; Hunt et al. 2009) satellite LiDAR system on the NASA Cloud-Aerosol Lidar and Infrared Pathfinder Satellite Observations (CALIPSO) satellite, launched in 2006. With the downward-facing LiDAR, CALIOP provides a vertically allocated measure of backscattering along the track of the satellite. Where this intersects smoke plumes, it can provide measures of the aerosol both at ground level as well as throughout the vertically sampled plume (Liu et al. 2009). Both MISR and CALIOP data have been used to examine plume rise models (e.g., Kahn et al. 2008; Raffuse et al. 2012; Val Martin et al. 2012; Val Martin, Kahn, and Tosca 2018), with modeled plumes showing generally consistent trends compared to the satellites, but with a large amount of variability.

Limitations of the use of these data to constrain modeled smoke plumes include both the timing of the overpass of the fire for MISR and the paucity of the number of times CALIOP, which is not a scanning instrument, intersects major plumes. MISR overpass times are typically in the mid-morning over the continental U.S., but fire plumes continue to grow into the afternoon when humidity, temperature, and development of atmospheric boundary layer typically lead to the highest plume heights. For CALIOP, Raffuse et al. (2012) found only 157 CALIPSO orbit paths (out of 25,000 orbits) intersecting HMS smoke plumes during a three-year period. The recent launch in 2017 of the TROPOspheric Monitoring Instrument (TROPOMI; Veefkind et al. 2012) on the European Space Agency sun-synchronous orbiting (similar to polar-orbiting) Sentinel-5 Precursor satellite, with its Aerosol Layer Height-derived product, offers the potential for daily global coverage and fast retrieval, and examination of this product has only recently started (Griffin et al. 2019). Additionally, Lyapustin et al. (2019) have recently derived a new methodology for determining plume injection height based on thermal differences of the rising plume with the surrounding air based on MODIS observations. Their algorithm is part of the Multi-Angle Implementation of Atmospheric Correction (MAIAC) MODIS collection six products, available daily at a 1-km resolution.

Aerosol optical depth (AOD) is a measure of the integrated amount of aerosol within the full vertical column of the atmosphere, derived from an estimation of the column-integrated attenuation of light due to

scattering and absorption. AOD is available from both the polar-orbiting MODIS, the geostationary GOES, and the sun-synchronous TROPOMI. AOD is also available from VIIRS, where it is called aerosol optical thickness. AOD is useful for showing overall plume extent from major wildfires and, despite being column-integrated, statistical connections with ground-based AERONET measurements and others have allowed for the estimation of surface $PM_{2.5}$ from AOD (Drury et al. 2010; Gupta and Christopher 2009; Hu et al. 2014; Liu et al. 2005a; van Donkelaar, Martin, and Park 2006; Xie et al. 2015).

In addition to aerosols, satellites can detect other atmospheric components that can be used to track smoke plumes, including CO and NO_2. The Measurements of Pollution in The Troposphere (MOPITT) instrument onboard the Terra satellite, launched in 1999, measures column-integrated CO through the use of an array of specific wavelength channels where CO absorbs (Drummond et al. 2010). However, the instrument has non-uniform vertical sensitivity, complicating application and interpretation. Nonetheless, the result is the ability to record column-integrated CO levels across a substantial fraction of the planet each day. MOPITT data have been used to track smoke plumes over large areas (e.g., Lamarque et al. 2003; Liu et al. 2005b; Pfister et al. 2005). TROPOMI (on the Copernicus Sentinel-5 Precursor satellite) also measures CO, as well as CH_4, NO_2, SO_2, and other aerosol properties (Veefkind et al. 2012). Observations from OMI (on the NASA Aura satellite) have been used to understand NO_2 emissions from biomass burning (Mebust et al. 2011; Tanimoto et al. 2015). The upcoming Tropospheric Emissions: Monitoring of Pollution (TEMPO; Zoogman et al. 2014) geostationary mission is designed to augment and enhance current satellite capabilities for measuring atmospheric composition, and it will include a wide array of species, including O_3, NO_2, SO_2, and various aerosol properties of smoke plumes. By combining ultraviolet and visible wavelengths, TEMPO will, for the first time, allow satellite measurement of lower tropospheric (0–2 km altitude), free tropospheric, and stratospheric O_3. TEMPO also offers the promise of observing near-surface O_3, $PM_{2.5}$, and other pollutants at a higher resolution (e.g., 4.4 km x 2.1 km).

Field campaigns

Smoke has received increasing scrutiny from the atmospheric sciences and chemistry community via a number of large field campaigns that include ground-based, airborne, and satellite observations. These include the Department of Energy–sponsored Biomass Burning Observation Project (BBOP) campaign (https://www.arm.gov/research/campaigns/aaf2013bbop) (Briggs et al. 2016; Collier et al. 2016; Zhou et al. 2017), Studies of Emissions and Atmospheric Composition, Clouds and Climate Coupling by Regional Surveys (SEAC4RS) project (Toon et al. 2016), the NOAA-NASA Fire Influence on Regional to Global Environments and Air Quality (FIREX-AQ) campaign (https://www.esrl.noaa.gov/csd/projects/firex-aq/), the NSF-sponsored Western Wildfire Experiment for Cloud Chemistry, Aerosol. Absorption and Nitrogen (WE-CAN) campaign (https://www.eol.ucar.edu/field_projects/we-can), and the U.S. Department of Agriculture–sponsored Fire and Smoke Model Evaluation Experiment (FASMEE) experiment (https://sites.google.com/firenet.gov/fasmee/) (Prichard et al. 2019b). As of early 2020, much of this research has yet to be published, but as this work becomes available we anticipate many new findings and advances in the field, particularly in the areas of better estimations and models of emissions, speciation within smoke, and how smoke chemically ages and interacts with other pollutants in the air throughout the plume.

Emissions

Emissions from wildfires and prescribed fires in 2017

2017 was a major fire year; wildfires burned over 4 million hectares and prescribed fires almost 5 million ha. Tables 1 and 2 show the top five states for annual areas burned in 2017 for wildfires (Table 1) and prescribed fires (Table 2), along with some of the highest monthly areas burned for each state. The tables also show the $PM_{2.5}$ emissions for those months and the maximum observed daily mean $PM_{2.5}$ concentrations at any regulatory monitor for the month.

Table 1. Top five states for annual area burned as wildfires, from the EPA draft National emissions inventory for 2017. Also shown are the peak monthly areas burned (blue shading), peak monthly $PM_{2.5}$ emitted (orange), and the maximum $PM_{2.5}$ concentration measured at any regulatory monitor for the month (green; data from AirNowTech).

State	Annual Area Burned (ha)	Month	Month Area Burned (ha)	Month $PM_{2.5}$ Emitted (tons)	Maximum Daily $PM_{2.5}$ Measured in the Month ($\mu g/m^3$ 24-hr avg)
California	641,440	August	93,388	126,331	310
		October	151,492	106,657	215
Montana	584,527	September	222,497	158,647	550
Nevada	519,250	July	373,169	21,742	135
Oregon	381,294	August	152,505	142,845	314
Idaho	367,205	August	129,799	51,974	125
		September	80,922	93,048	361

Table 2. Top five states for annual area burned as prescribed fires, from the EPA draft National Emissions Inventory for 2017. Also shown are the peak monthly areas burned (blue shading), peak monthly $PM_{2.5}$ emitted (orange), and maximum $PM_{2.5}$ concentration measured at any regulatory monitor for the month (green; data from AirNowTech).

State	Annual Area Burned (ha)	Month	Month Area Burned (ha)	Month $PM_{2.5}$ Emitted (tons)	Maximum Daily $PM_{2.5}$ Measured in the Month (μg/m^3, 24-hr avg)
Texas	632,470	February	143,468	12,807	29
Georgia	465,219	February	92,595	10,217	32
Oklahoma	449,616	March	140,656	18,615	49
Florida	386,518	February	90,367	8,733	30
Alabama	366,899	March	66,059	8,344	38

These tables convey several important results. First, area burned did not correspond to either $PM_{2.5}$ emissions or peak measured concentrations. Rather, the emissions depended strongly on fuel type and density as well as burning conditions. Compared to flaming combustion, smoldering fires emitted more $PM_{2.5}$ per unit of fuel consumed. Heavily forested regions, such as northern California and the Pacific Northwest, had much higher fuel loadings than rangelands (e.g., Nevada) and consequently much higher $PM_{2.5}$ emissions.

Second, even where $PM_{2.5}$ emissions were large, air quality monitors may not have measured high concentrations. This depended on the location of the fires relative to the monitors and transport. For example, the highest wildfire emissions in California were in August, and although the measured $PM_{2.5}$ concentration of 310 μg/m^3 is notable, some state and USFS mobile monitors in several parts of the state reported even higher values on some days. The highest daily mean observed $PM_{2.5}$ in August at a non-regulatory monitor was 745 μg/m3 for a site near Happy Camp, CA, on 8/24/2017. (See https://wildlandfiresmoke.net for near-real time data access. Note that past non-regulatory data is not routinely made available. Contact the authors for more information about accessing this data.) In October, large areas burned in the Napa Wine Country fires. Although the emissions were somewhat lower compared to August, a large population was exposed to unhealthy to very unhealthy levels of $PM_{2.5}$ across the San Francisco Bay area (>200 μg/m^3). These data show the clear signature of wildfires dominating the western U.S. in the summer months and into late fall in California.

In the central and southeastern U.S. (Table 2), prescribed burning peaks in late winter and spring. Although the area burned by prescribed fires was of a similar magnitude as wildfires in the West, the $PM_{2.5}$ emissions were approximately an order of magnitude lower, and the levels of measured $PM_{2.5}$ concentrations were also much lower. This difference is due to both the fuel types (e.g., rangelands) and the management practices in forest systems, where prescribed fires typically do not burn canopy or duff fuels. Thus, these data show that prescribed burning in the southeastern U.S. had much lower emissions per ha, likely due to the fuels and management goals for each fire.

The relationship between fire activity and smoke, 2004–2018

The relationship between the amount of fire in a region and human exposure to $PM_{2.5}$, O_3, and other pollutants is complex. Generally, increases in regional fire result in reduced air quality due to smoke, but this relationship is complicated by smoke transport from other regions and the locations of the fires with respect to in-situ air quality monitors. Figures 6 and 7 show the percentage of monitor-days that exceeded a daily average of 35 μg/m^3 as well as the area burned in 2004–2018 for two states, California and Washington. California (Figure 6) showed a general trend to fewer days over 35 μg/m^3, due to decreasing industrial emissions (McClure and Jaffe 2018), but this number of days clearly increased with the high area burned in 2007, 2008, 2017, and 2018. If the temporal trend is removed, there is a significant correlation between area burned in California and the percentage of monitor-days over 35 μg/m^3 (R^2 = 0.54).

Washington (Figure 7) had fewer days over 35 μg/m^3, but the frequency increased with the large area burned in 2006 and 2015. In 2017 and 2018, the percentage of days above 35 μg/m^3 was much higher than in the previous decade, due not only to fires in Washington but also to transport of smoke from fires in Montana, British Columbia, and Oregon. This reflects the spatial pattern of fires and smoke and the spatial coverage of monitors within each state.

Emissions from fires

About 80–90% of the emissions by mass from biomass fires are of CO_2. Of the non-CO_2 portion, CO represents the largest fraction (~60%), followed by volatile organic compounds (VOC, ~15%), primary $PM_{2.5}$ (~8%), and CH_4 (~2%) (Akagi et al. 2011; Andreae 2019). Other gas phase emissions include inorganic species, including NOx, HCN, NH_3, and HONO. To

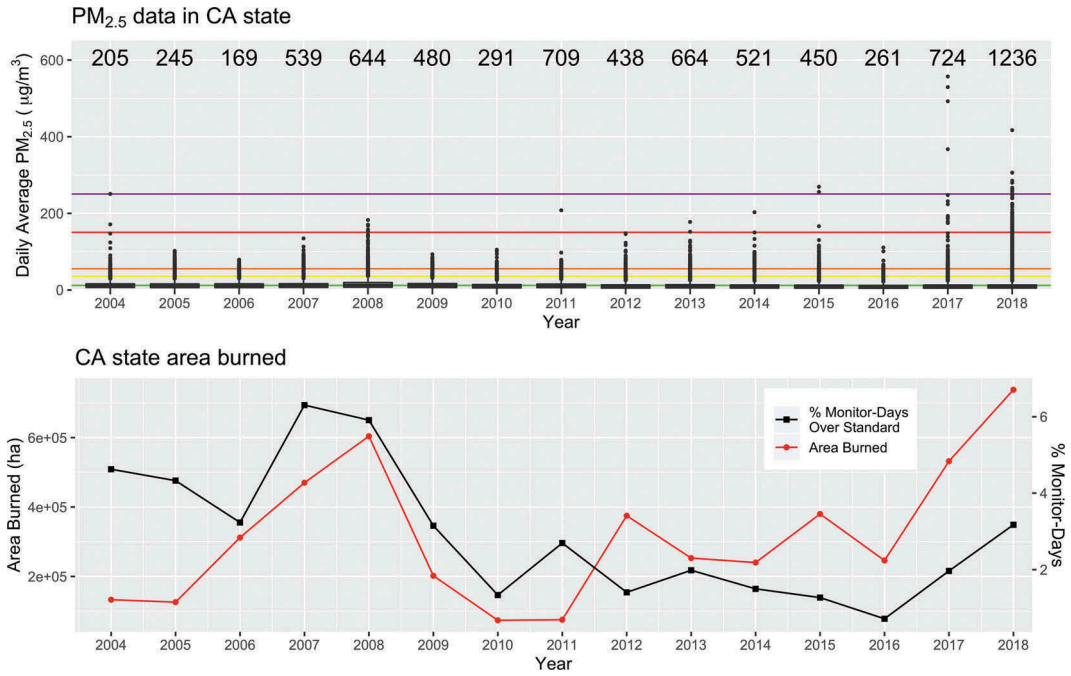

Figure 6. (Top) Box and whisker plots of all daily $PM_{2.5}$ concentrations by year for air quality monitors in California. The numbers at the top of the panel show the total number of monitor-days above the daily $PM_{2.5}$ standard (35 µg/m^3). Colored horizontal lines show the six AQI cut points: Good, <12 µg/m^3; Moderate, <35.4 µg/m^3; Unhealthy for Sensitive Groups, <55.4 µg/m^3; Unhealthy, <150.4 µg/m^3; Very unhealthy, <250.4 µg/m^3; Hazardous, >250 µg/m^3 (see Figure 1 for color key). (Bottom) Annual area burned (left y-axis) and percentage of all monitor-days that exceeded the daily $PM_{2.5}$ standard (right y-axis). All $PM_{2.5}$ data from the EPA AQS system are included (regulatory and non-regulatory). Sources: Burned area for each state is from NIFC, and $PM_{2.5}$ data are from the EPA AQS database.

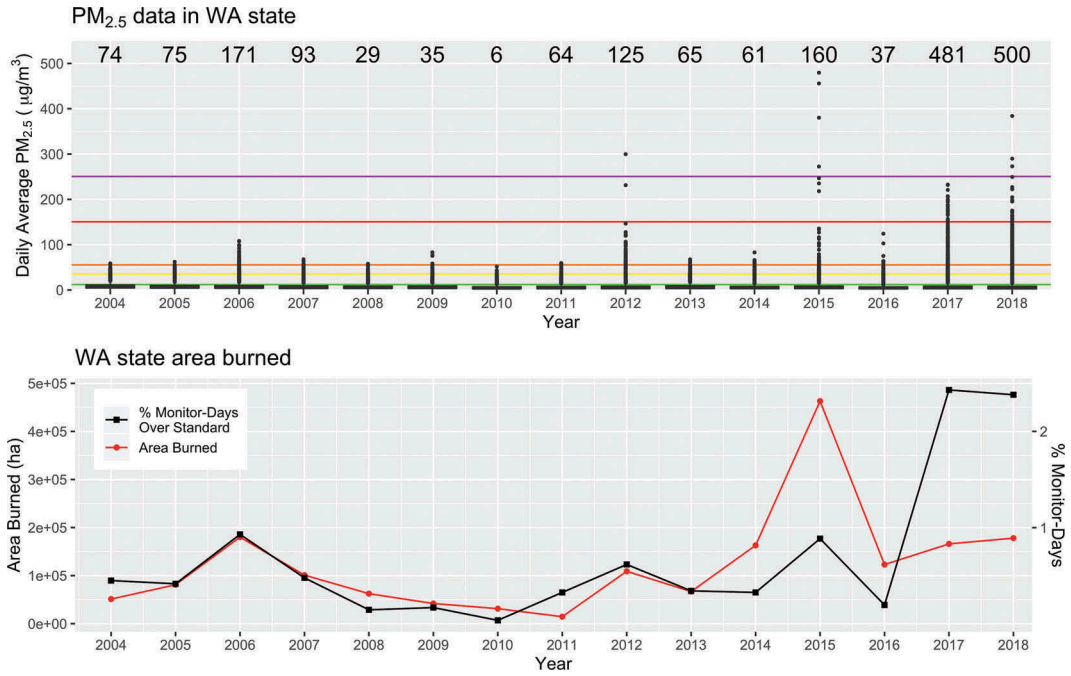

Figure 7. As in Figure 6, but for Washington.

date, more than 500 individual VOCs have been identified in smoke (Hatch et al. 2017), and these compounds are highly reactive with the OH radical (Kumar, Chandra, and Sinha 2018). Although VOCs represent only a fraction of the total gaseous emissions from biomass fires, many are associated with adverse health effects, and the mixture is more reactive than typical industrial emissions, with a high potential for secondary organic aerosol (SOA) and O_3 formation.

Primary smoke $PM_{2.5}$ emissions are composed mainly of organic compounds (>90%), with lesser amounts of elemental carbon (ca 5–10% by mass), NO_3^-, K^+, Cl^-, NH_4^+, and other constituents (Kondo et al. 2011; Liu et al. 2017b; Park et al. 2003; Zhou et al. 2017). Despite their much lower emissions compared to the organic compounds, these and other trace-level elements can be important for biogeochemical cycles and as tracers for source apportionment. For example, fires emit fluorine in globally significant amounts (Jayarathne et al. 2014). Smoke particles are mostly small, with median diameters in the range of 50–200 nm (Carrico et al. 2016; Laing, Jaffe, and Hee 2016), although a few larger particles can extend into the super micron range (e.g., Maudlin et al. 2015). The emissions are variable from fire to fire and depend on fuel type, fuel moisture, fire conditions, temperature, weather, and other factors (Cubison et al. 2011; Hecobian et al. 2011). This variability is a major challenge for understanding the emissions, chemistry, and subsequent impacts of smoke.

Emissions inventories

An emissions inventory (EI) provides a detailed accounting of hectares burned and the pollutants emitted from each fire. An EI is typically used both as an input for air quality models and health assessments and to gauge the relative amounts of different pollutants emitted to the atmosphere. Emissions of species x is often calculated from:

$$E_x = A \times B \times FB \times EF_x \quad (1)$$

Where E_x is the mass of species x emitted, A is the area burned, B is the mass of biomass per unit area, FB is the fraction of biomass consumed, and EF_x is the emission factor per unit fuel consumed for species x (Seiler and Crutzen 1980; Urbanski 2014; Wiedinmyer et al. 2011).

North America has two national EIs: the EPA's NEI (U.S. EPA, 2019a) and the Canadian Air Pollutant Emissions Inventory (APEI; Canada, 2019). At a global scale, there are several EIs for fire emissions, including the Fire Inventory from National Center for Atmospheric Research (FINN; Wiedinmyer et al. 2011), the Global Fire Emissions Database (GFED; van der Werf et al. 2017), the Global Fire Assimilation System (GFAS; Kaiser et al. 2012), and the Integrated System for wild-land Fires (IS4FIRES; Soares, Sofiev, and Hakkarainen 2015).

Unlike the other EIs, which rely solely on satellite fire detects, the NEI uses fire activity data obtained from national, regional, and state reporting (e.g., federal incident reports used to calculate National Interagency Fire Center [NIFC] statistics, Fire Emissions Tracking System [FETS, htto://wrapfets.org]), augmented and reconciled with satellite data (e.g., from NOAA's Hazard Mapping System) (Larkin et al. 2020). The BlueSky emissions modeling framework (Larkin et al. 2009) is then used to generate daily fire emissions, and the EPA applies PM chemical speciation, vertical allocation, and a temporal profile according to the fire type: agricultural, prescribed fire, or wildfire and by season and location (Eyth et al. 2019; Pouliot et al. 2017).

EIs developed from activity reports require considerable effort to develop and are reported retrospectively on a temporally resolved annual (e.g., Canada's APEI) or triennial basis (e.g., NEI); in contrast, EIs based solely on satellite detection can, in principle, be reported in near-real time. Between NEI years, the EPA also develops fire emissions for air quality modeling purposes, using the same data sources but without the extensive review process done for the NEI (Koplitz et al. 2018). By consolidating multiple sources for fire activity, the NEI hectares burned are nearly 20% higher than NIFC–reported wildfire areas and over 100% higher than GFED burned areas, likely due to the inclusion of smaller prescribed fires that may not be reported to NIFC or detected by satellite (Larkin et al. 2020). Emissions can also be estimated by applying smoke emission coefficients to fire radiative power (FRP), avoiding some of the uncertainty in fuel loading and amount consumed (e.g., the NASA Fire Energetics and Emissions Research algorithm; Ichoku and Ellison 2014). However, this approach can miss low-intensity, short-duration, understory fires, resulting in a 54% lower $PM_{2.5}$ emission estimate compared to the NEI (Li et al. 2019a). Comparisons among EIs and the NEI are still sparse.

Emission factors

Emission factors (EFs; see equations 1 and 2) are a critical input parameter in wildland fire EIs and emissions models (e.g., BlueSky Modeling Framework; Larkin et al. 2009). EFs are defined as a mass of species

emitted per unit mass of dry fuel consumed (Andreae 2019). The carbon balance method (Radke et al. 1988; Ward et al. 1982) is the most widely used approach to calculate EFs:

$$EF_x = F_c \frac{\Delta C_x}{\sum_i \Delta C_i} \quad (2)$$

Where EF_x is the EF of species x, F_c is the fraction of carbon in the fuel, ΔC_x is the excess carbon mass concentration of species x (often concentrations are replaced with normalized excess emissions ratio to CO_2 or CO), and the denominator is the sum of the carbon from all carbon-containing species, often limited to CO_2 and CO. The carbon balance method has several assumptions that may introduce error into the EF calculation:

(1) All carbon in the burned fuel is consumed – Carbon remaining in the fuel as char is frequently omitted in the carbon balance, which results, on average, in a 4% overestimate (Surawski et al. 2016).
(2) All major carbon-containing species emitted are accounted for – CO_2 and CO typically account for ~96% of the carbon emissions (Yokelson et al. 1999); ignoring VOCs and particulate carbon results in an overestimate in EFs of about 4%.
(3) Carbon fraction of the fuel is known and approximately constant – Carbon fractions of 0.45–0.50 (Andreae 2019; Yokelson et al. 1999) are commonly used when fuel-specific information is not known, increasing uncertainty in the EF about 10% (Susott et al. 1996).
(4) All species are transported to the measurement location with no losses or deposition – The effect of this assumption is unknown (Hsieh, Bugna, and Robertson 2016).
(5) Background concentrations are accurately accounted for – Background CO_2 enhancement in dilution air underestimates the EFs by about 6% (Hsieh, Bugna, and Robertson 2016). Aircraft measurements downwind encountering background air masses of varying pollutant levels (e.g., at boundary layer vs. free troposphere) can result in a large (>50%) change in normalized excess emission ratios (Chatfield et al. 2019; Yokelson, Andreae, and Akagi 2013). Briggs et al. 2016, see supplemental information) propose a method to compute the uncertainty in these values due to this effect.

These assumptions introduce a positive bias, with added uncertainty from approximating a constant carbon fraction in the fuel. These errors are outside measurement errors, which for some species, like PM, may be sizable as well. However, the uncertainties in the measurement and calculation of EFs are eclipsed by the immense variability of emissions from varying fuels and combustion conditions, as evidenced by the wide range of EFs reported in the literature. Note that the EF equation is similar to one used for enhancement ratios (ERs), but EFs are reserved for cases where fire emissions are observed directly, and ERs are used for downstream measurements, where significant processing of the emissions may have occurred (e.g., Briggs et al. 2016).

The large variability in EFs has been a major driver of research on the emissions from wildland fires. Over the past two decades, a number of EF compilations have been published for global wildland fires and other types of biomass fires (e.g., charcoal making, home biofuel, trash burning; Akagi et al. 2011; Amaral et al. 2016; Andreae 2019; Andreae and Merlet 2001). Other EF compilations have focused on North American wildland fires including wild and prescribed fires (e.g., Lincoln et al. 2014; Prichard et al. 2020; U.S. EPA, 1995; Urbanski 2014; Ward et al. 1989). New emissions studies investigating different fuels, fire types, and emissions characteristics are published frequently, which is why some compilations provide periodic updates. The FINN emission factor compilation is periodically updated with emission factors from recently published studies (http://bai.acom.ucar.edu/Data/fire/). Prichard et al. (2020) developed the Smoke Emissions Reference Application (SERA; https://depts.washington.edu/nwfire/sera/index.php) to be a searchable online EF repository.

Compiling EFs into a cohesive database also facilitates the assessment of data gaps for fuel types/ecoregions, combustion conditions, and pollutants, and it provides a tool for understanding how emissions vary with these parameters. Comparing the EF observations with the average hectares burned in each state from 2006 to 2016 (U.S. EPA, 2019a) reveals that some areas of the U.S. with high fire activity are overlooked in emissions studies (Figure 8). For example, Texas, which has the highest average burned area in the country, has only two EF observations in the SERA database. Other central and southern U.S. states also have high areas burned but few or no EFs in SERA. This limits our understanding of the impact of these fires on air quality.

Of the major species in SERA, 75–90% of the EFs are from laboratory studies, 10–20% are from prescribed

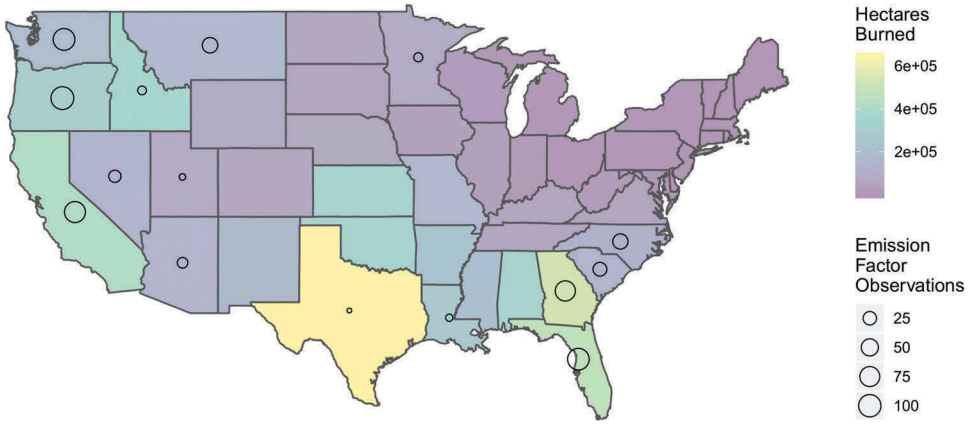

Figure 8. Comparison of the annual average hectares burned for each state in the continental U.S. (2006–2016) with the number of particulate matter emission factor observations for each state in the SERA database.
Source for hectares burned: U.S. Environmental Protection Agency (U.S. EPA) (2019a).

fires, and <5% are from wildfires. The exception is for CO_2 and CO, for which there are ~600 observations, with approximately 15% fromwildfires and the remaining EFs evenly split between lab and prescribed fires. Other pollutants, like NOx, NH_3, or some of the more commonly measured VOCs, like CH_4, have only around 200 EFs across all fuel and ecosystem types (Table 3).

Historically, wildland fire emissions have been modeled using the two basic combustion phases: flaming or smoldering (Prichard, Ottmar, and Anderson 2007). The modified combustion efficiency (MCE) is used as the primary indicator of combustion phase, with MCE > 0.9 considered flaming combustion and MCE < 0.9 as smoldering combustion (Urbanski 2014). MCE is defined as:

$$MCE = \frac{\Delta CO_2}{\Delta CO_2 + \Delta CO} \quad (3)$$

Where ΔCO_2 is the excess CO_2 concentration and ΔCO is the excess CO concentration. EFs for pollutants associated with incomplete combustion (CO, CH_4, and PM) are all moderately to strongly correlated with MCE (r^2 = 0.64, 0.71, and 0.47, respectively; Prichard et al. 2020). Some compounds, like NOx, are poorly predicted by MCE (r^2 = 0.07; Prichard et al. 2020) but have been found to be linearly correlated with fuel nitrogen (Delmas, Lacaux, and Brocard 1995). Elements such as K, Cl, and Ca also appear; these can vary widely among fuel types and depend more on fuel composition, with combustion conditions playing a secondary role.

Prichard et al. (2020) analyzed the SERA EF database to identify conditions with few EF observations. More information is still needed for wildfire EFs, particularly because some studies indicate much higher wildfire EFs, possibly due to the greater consumption of coarse wood, duff, and moist canopy fuels (Liu et al. 2017a). There is also a need for more EFs for smoldering conditions, especially from coarse wood and duff fuel types. Information is limited on how the environmental conditions and the fire behavior affect emissions, which are important considerations for prescribed fire burn plans

Table 3. Comparison of average emission factors (EFs) from non-biomass fuels (e.g., structures, furnishings, vehicles) at the wildland-urban interface (WUI) and from natural fuels from wildland fires, derived from SERA. EF units are g/kg fuel consumed, unless otherwise noted.

	CO_2	CO	HCN	NOx	HCl	SO_2	PM	C_6H_6	Benzo(a) pyrene	Polychlorinated dibenzo-*p*-dioxins (μg/kg)	Polychlorinated dibenzofurans (μg/kg)
Average EF for non-biomass WUI fuels	1514	124	8.8	5.7	153	62.2	66.7	31.4	0.12	0.53	14.0
# EFs observed	143	145	49	21	32	14	97	41	18	4	4
Standard deviation	917	130	41.6	19.4	404	164	84.9	67.2	0.19	1.04	28.0
Average EF for wildland fires	1550	104	0.5	2.2	0.3	1.1	25.1	0.4	0.0003	0.032	0.021
# EFs observed	597	640	188	202	37	125	688	84	11	13	13
Standard deviation	313	58	0.6	2.1	0.9	0.7	34.8	0.3	0.0002	0.020	0.017
WUI/wildland fire EF ratio	0.98	1.2	19	2.6	488	56	2.7	85	366	16	667

(Waldrop and Goodrick 2012). The observations on prescribed fires also presents contradictory results. Bian et al. (2020) reported that prescribed fires in the southeastern U.S. tend toward more-smoldering conditions compared to other parts of the country, which would presumably increase the $PM_{2.5}$ EFs (Prichard et al. 2020). But Liu et al. (2017a) reported lower $PM_{2.5}$ EFs from southeastern prescribed fires compared to western wildfires. A better understanding of the factors and environmental controls associated with prescribed burning is needed to improve our estimates of their emissions.

Primary gas phase emissions

Gas phase emissions are composed of oxidized species associated with flaming conditions, including CO_2, NOx, HONO, SO_2, and more reduced species associated with smoldering conditions, including CO, CH_4, HCN, and NH_3. Both combustion phases are associated with emissions of VOCs, and these have a range of volatilities, oxygenation, heteroatoms (N, F, S, Cl, Br, I), and functional groups (e.g., ketones, carbonyls, alcohols) (Prichard et al. 2020). Most of the VOCs are unsaturated compounds (>80%), and around 60% are oxygenated-VOCs (OVOCs) (Gilman et al. 2015). The most abundant OVOCs emitted from typical U.S. fuels are formaldehyde, formic acid, methanol, acetaldehyde, and acetic acid. Levoglucosan and phenolic compounds (e.g., cresols, guaiacol) are also nearly ubiquitous, but in highly variable amounts (Hatch et al. 2018). Most VOCs vary greatly in their relative abundance across different fuels, and some are unique to specific fuel types (Hatch et al. 2018), demonstrating the difficulties of attempts to simplify emissions models for even the most commonly emitted molecules.

Many of the VOCs correlate only modestly with MCE and are better categorized as products of the initial distillation of fuel or from low or high temperature pyrolysis reaction pathways (Sekimoto et al. 2018). During the brief initial distillation phase, the higher volatility of unburned fuel compounds, like monoterpenes and other biogenic-derived VOCs, are emitted (Sekimoto et al. 2018). Despite contributing minimally to the overall VOC emissions, these biogenic VOCs may have an important role in flammability, by reducing ignition times (De Lillis, Bianco, and Loreto 2009; Owens et al. 1998) and enhancing the rate of fire spread (Chetehouna et al. 2014). The low-temperature pyrolysis products include a greater fraction of low volatility compounds, oxygenates, furans, and ammonia, while the high-temperature products have few low-volatility compounds and are enriched in aliphatic hydrocarbons, PAHs, HCN, HCNO, and HONO (Sekimoto et al. 2018).

Primary particle emissions – chemical, physical, and optical characteristics

Particle emissions from wildland fires are complex, with time-varying size, morphology, chemical composition, and volatility, all of which determine their impact on human health and the environment. PM emissions are composed mainly of organic carbon (50–75%), with 5–10% elemental carbon (EC) or black carbon (BC), and typically less than 5% of inorganic ions (e.g., K, Cl) and metals (Ward and Hardy 1988); the balance of the PM mass is from elements associated with organic carbon (e.g., H, O, N, S). Note that EC and BC are not equivalent and depend on the measurement methodology (Andreae 2019; Petzold et al. 2013). Measurements of complete particle composition are still relatively sparse (Balachandran et al. 2013; Einfeld, Ward, and Hardy 1991; Lee et al. 2005; Ward and Hardy 1988), with many not reporting either the organic fractions (Alves et al. 2019, 2011; Reisen et al. 2018) or the inorganic fractions (Aurell and Gullett 2013; Aurell, Gullett, and Tabor 2015; Holder et al. 2016; Vicente et al. 2013). Toxic metals are also present in PM at very low levels (Alves et al. 2011; Gaudichet et al. 1995; Popovicheva et al. 2016), but they may be enriched in emissions from wildland fires that occur on or near contaminated sites (Kristensen and Taylor 2012; Odigie and Flegal 2014; Wu, Taylor, and Handley 2017).

Organic emissions have a range of volatilities (gas phase, intermediate volatility, semivolatile, low volatility, particle phase), which makes measuring PM difficult, because up to 40% of the PM mass may be lost due to evaporation of the semivolatile compounds (Hatch et al. 2018). The distribution across the volatility range is relatively constant for most combustion conditions and fuels (May et al. 2013). The lowest-volatility fraction consists primarily of anhydrosugars, whereas alcohols and acids dominate the semivolatile range, and phenols dominate in the higher volatility range (Hatch et al. 2018).

Most lab and field studies have demonstrated that BC emissions increase with MCE (e.g., Jen et al. 2019; Selimovic et al. 2018), but other studies have found a weaker relationship (e.g., Hosseini et al. 2013; McMeeking et al. 2009). Laboratory burning studies have suggested larger BC particle mass fractions

compared to field observations under flaming conditions (lab: 15 ± 12%, field: 8 ± 5%) as well as higher inorganic content (lab: 12 ± 13%, field: 8 ± 5%) (Alves et al. 2011; Balachandran et al. 2013; Ferek et al. 1998; Guo et al. 2018; Hosseini et al. 2013; McMeeking et al. 2009; Turn et al. 1997; Ward and Hardy 1988). Both results suggest that laboratory burning cannot fully capture the characteristics of wildland fire emissions.

The composition of PM affects its size, morphology, and hygroscopicity, all of which impact optical properties. The BC fraction is formed during flaming combustion; it is composed of graphitic-like primary particles, with diameters of 20–50 nm that aggregate into larger particles of approximately 200 nm (volume equivalent diameter) (Holder et al. 2016; Sahu et al. 2012; Schwarz et al. 2008) that are hydrophobic (Petters et al. 2009). However, most PM is organic with moderate hygroscopicity (Petters et al. 2009) and, for fresh emissions, has a single size mode with count median diameters (CMD) of ~120 nm and geometric standard deviations (GSD) of ~1.7 (Janhäll, Andreae, and Pöschl 2010; Reid et al. 2005; Virkkula et al. 2014; Wardoyo et al. 2007). Some fuels, like grasses and some shrubs, emit PM with a larger inorganic fraction, resulting in larger hygroscopicity (Carrico et al. 2010; Petters et al. 2009) that may impact light-scattering properties of biomass burning aerosols at high relative humidities (Gomez et al. 2018). Aging causes the PM to converge to a moderate hygroscopicity, likely due to secondary organic aerosol formation (Engelhart et al. 2012; Lathem et al. 2013), making these particles able to serve as cloud condensation nuclei under some conditions. Aging also results in larger particles but with a narrowed size distribution, with CMDs around 175–300 nm and GSDs of 1.3–1.7 (Janhäll, Andreae, and Pöschl 2010); however, wide ranges of CMDs and GSDs have been observed in plumes of various ages and transport histories (Laing, Jaffe, and Hee 2016). PM from flaming emissions are mostly larger than those from smoldering (Janhäll, Andreae, and Pöschl 2010), but mixed results have been seen in the lab from the same fuel (Ordou and Agranovski 2019), and some smoldering fires produce larger particles (Iinuma et al. 2007). PM (both the OC and BC fraction) from grassland fires tends to be smaller than PM from fires of forests or shrublands (Holder et al. 2016; Reid et al. 2005). More field measurements of size and composition of PM emissions from many types of fires and combustion conditions are needed.

Among the organic fraction, tar balls are another distinct particle type that as yet can be conclusively identified only through electron microscopy (Pósfai et al. 2004, 2003). Tar balls are characterized as highly viscous spherical particles (100–300 nm diameter) or aggregates thereof (Girotto et al. 2018; Hand et al. 2005; Pósfai et al. 2004), stable at high temperatures (retaining 70% of tar ball mass at 600 C; Adachi et al. 2019), and composed of amorphous carbon, oxygen, often sulfur, and trace levels of potassium (Adachi et al. 2019). How tar balls are formed is still uncertain (Hand et al. 2005; Sedlacek et al. 2018; Toth et al. 2018), but they appear to increase in number fraction with plume age (Adachi et al. 2019; Sedlacek et al. 2018). Tar ball optical properties and how they relate to other types of organic carbon have yet to be resolved.

Much recent research on smoke PM optical properties has focused on absorption due to the considerable uncertainty in the climate impacts of smoke from wildland fires (Jacobson 2014). Optical properties also affect rates of photolysis (Baylon et al. 2018; Mok et al. 2016) and photosynthesis (Hemes, Verfaillie, and Baldocchi 2020), and they are a critical factor in remote sensing of PM (Li et al. 2019b) and source identification (Schmeisser et al. 2017). Both the BC fraction and the organic fraction contribute to the absorption. BC absorbs across a broad wavelength range, with a weak variation characterized by an angstrom absorption exponent (AAE) of 1 (Bond and Bergstrom 2006). The angstrom absorption exponent is calculated by:

$$AAE = -\frac{\ln abs(\lambda_1)/\ln abs(\lambda_2)}{\lambda_1/\lambda_2} \quad (4)$$

Where abs is the absorption and λ is the wavelength. Some portion of the organic fraction has strong absorption in the UV wavelengths, with AAEs typically >2. This fraction is referred to as brown carbon (BrC) (Andreae and Gelencsér 2006) and is composed of organic compounds such as polycyclic aromatics, nitroaromatics, and humic-like substances (Laskin, Laskin, and Nizkorodov 2015). But rather than being two distinct PM types (BC and BrC), PM may exhibit a continuum of compositions, volatilities, and optical properties from BC to BrC (Adler et al. 2019; Saleh, Cheng, and Atwi 2018).

Emissions from fires in the wildland-urban interface (WUI)

In the wildland-urban interface (WUI), structures, vehicles, and the substances contained within them also burn and contribute to emissions. These "fuels" have very different chemical compositions from natural fuels (soils, grasses, shrubs, and trees) and likely very different emissions. A number of studies have measured emissions from structure and vehicle fires (e.g., Fabian et al. 2014, 2010; Fent et al. 2018; Lecocq et al.

2014). These have shown a wide array of harmful emissions, including irritants (HCl, HF, NO_2, HS, SO_2), asphyxiants (CO, HCN), sensitizers (Isocyanates), carcinogens (formaldehyde, benzene, PAHs, dioxins), and toxic metals (Cd, Cr, Pb). To our knowledge, no EI or model exists that includes emissions from structure or vehicle fires as part of the emissions from wildland fires. Several studies have reported EFs from building materials and furnishings, but few have measured emissions from full-scale fires (Blomqvist, Rosell, and Simonson 2004; Gann et al. 2010; Lönnermark and Blomqvist 2006; Wichmann, Lorenz, and Bahadir 1995). Most studies have measured emissions from small pieces of these materials combusted in a cone calorimeter or tube furnace. Of the studies with EFs, none provides a complete assessment of all such emissions that may impact human health or the environment, for example, inorganic gases (Blomqvist, Rosell, and Simonson 2004; Gann et al. 2010; Kozlowski, Wesolek, and Wladyka-Przybylak 1999; Lönnermark and Blomqvist 2006; Lönnermark et al. 1996; Persson and Simonson 1998; Stec and Hull 2011), PM (Blomqvist, Rosell, and Simonson 2004; Elomaa and Saharinen 1991; Fabian et al. 2010; Lemieux and Ryan 1993; Lönnermark and Blomqvist 2006; Reisen, Bhujel, and Leonard 2014; Valavanidis et al. 2008), VOCs (Blomqvist, Rosell, and Simonson 2004; Durlak et al. 1998; Font et al. 2003; Lemieux and Ryan 1993; Lönnermark and Blomqvist 2006; Lönnermark et al. 1996; Moltó, Font, and Conesa 2006; Reisen, Bhujel, and Leonard 2014), PAHs (Blomqvist et al. 2014; Blomqvist, Persson, and Simonson 2007; Blomqvist, Rosell, and Simonson 2004; Durlak et al. 1998; Elomaa and Saharinen 1991; Font et al. 2003; Lemieux and Ryan 1993; Lönnermark and Blomqvist 2006; Moltó, Font, and Conesa 2006; Reisen, Bhujel, and Leonard 2014; Valavanidis et al. 2008), dioxins (Blomqvist, Rosell, and Simonson 2004; Lönnermark and Blomqvist 2006), and toxic metals (Lemieux and Ryan 1993; Lönnermark and Blomqvist 2006; Valavanidis et al. 2008). Thus, extrapolation across studies is necessary to obtain a complete picture of emissions, which is needed, for example, to understand health impacts or to model fire chemistry for exceptional event demonstrations.

Table 3 compares the average EFs from the combustion of non-biomass WUI fuels (structures, vehicles, furnishings, and structural materials) and biomass fuels, derived from the SERA database. The EFs from the primary combustion products – CO_2, CO, and NOx – are similar for WUI and natural fuels. However, most WUI VOC EFs were far greater than those from natural fuels, with WUI/natural ratios ranging from 4 (propene) to over 2,000 (Diebenzo(a, h)anthracene). These EFs are highly variable, with relative standard deviations of 200–500%. In contrast, the EFs for aldehydes (formaldehyde, acetaldehyde, and acrolein) had much lower WUI/natural ratios (0.12–-0.9). The large WUI/natural ratios for the most toxic compounds suggest that fires in the WUI may present a substantial hazard to firefighters and nearby communities, despite the far lower "fuel" consumption in the WUI. However, estimates of emissions including structures and vehicles are still needed to accurately determine the impacts of smoke from fires in the WUI. This variability and the uncertainty in emissions from an individual fire propagate into uncertainties in forecast air quality impacts.

Transport

Once emitted, gases and particles interact with, and modify, the atmosphere in terms of physical processes such as airflow, heating of surrounding atmosphere, and radiative properties. Emissions associated with flaming combustion typically get injected higher into the atmosphere than emissions associated with smoldering combustion. On a micro scale these processes occur individually, but on a macro scale they occur simultaneously as a fire progresses across the landscape. Computational fluid dynamic (CFD) systems such as the Wildland-urban interface Fire Dynamics Simulator (WFDS) (Mell et al. 2007, 2009; Mueller, Mell, and Simeoni 2014) and FIRETEC (Linn et al. 2002, 2005) explicitly simulate these physical processes, with a focus on simulating the detailed combustion and propagation of the fire.

During combustion, energy is released in the form of radiation and latent and sensible heat. Radiant heat is transferred through the atmosphere and is largely responsible for the preheating of fuels. Sensible heat, in the form of conduction and convention, heats the surrounding atmosphere. Latent heat from the condensation of water vapor in the plume releases additional energy. The combination of these processes is responsible for lofting fire emissions vertically into the atmosphere.

As the emissions are injected, the plume entrains cooler air and mixes with the surrounding environment. In one of few studies that provide insight into the entrainment structures in a wildfire convective plume, Lareau and Clements (2017) used lidar to measure how this entrainment dilutes and expands the plume as it rises. Fires are often composed of multiple plume updrafts (Achtemeier et al. 2011), which have smaller ascending velocities and are more affected by

entrainment (Liu et al. 2010) than a single plume. The flaming front will also pull air in at its boundaries to fuel the combustion process. These phenomena represent the coupling of the fire with the atmosphere, which happens when the heat supplied by the fire is sufficient to overcome the kinetic energy of the ambient flow (Clements and Seto 2015) and results in modifications to the wind and temperature fields. Coupled fire-atmosphere modeling systems, such as the Weather Research and Forecasting (WRF) WRF-SFire (2014; Mandel, Beezley, and Kochanski 2011) and the Coupled Atmosphere-Wildland Fire-Environment (CAWFE) (Clark, Coen, and Latham 2004; Coen et al. 2013), compute fire spread using the Rothermel algorithm (Andrews 2018; Rothermel 1972), which is less computationally intense than the CFD approaches of WFDS and FIRETEC.

The plume injection height is controlled by the thermodynamic stability of the atmosphere and surface heat flux released from the fire (Freitas et al. 2007). The initial maximum height that the smoke plume reaches is referred to as the plume rise. Many methods have been developed to estimate this parameter, ranging from the traditional empirical approach by Briggs (1975), originally developed for power plant stack emissions, to 1-D models that include cloud microphysics and other boundary layer conditions (Freitas et al. 2007). Some methods rely upon radiant heat measured from space by remote-sensing instruments (Sofiev, Ermakova, and Vankevich 2012). Both ground-based and remote sensor-based studies have been conducted to evaluate various plume injection height schemes. Cunningham and Goodrick (2013) and Lareau and Clements (2017) found that their single plume measurement cases compared well to those of Briggs (1975). Raffuse et al. (2012), using data from the Multi-angle Imaging SpectroRadiometer (MISR) onboard the Terra satellite, found that the Briggs scheme was systematically low for smaller fires and high for large fires. Val Martin et al. (2012) evaluated parameterizations developed by Freitas et al. (2007) with MISR data and found that this approach tended to underestimate plume rise and did not perform well at identifying when plumes were injected into the free troposphere. Paugam et al. (2016) provide a comprehensive review of plume rise performance in chemical transport models along with the atmospheric and fire parameters governing plume rise.

An important corollary to plume injection height is the concept of how gases and aerosols are initially injected in the vertical, which is critical to atmospheric modeling of smoke plumes. The assumption is that emissions are distributed equally from either the ground to plume top or from an assumed plume bottom to the plume top. Mallia et al. (2018) found that model results were improved when fire emissions were distributed vertically below the plume top in a Gaussian manner. Systems such as the BlueSky Smoke Modeling Framework (Larkin et al. 2009) attempt to address this vertical allocation question by distributing smoldering emissions near the surface and flaming emissions aloft. Lidar data from both satellites and ground-based measurements can help track the vertical distribution of emissions (Banta et al. 1992; Clements et al. 2018; Lareau and Clements 2017). For example, Lareau and Clements (2017), in their measure of the turbulent structure of a plume using ground-based lidar, found a Gaussian distribution of backscatter (and thus smoke) in their single-plume study. Remotely sensed lidar data from the Cloud-Aerosol Lidar with Orthogonal Polarization (CALIOP) instrument gives vertical cross-sections of the atmosphere, and, if the swath occurs over a fire emission point, the data can inform the vertical injection distribution. The data also illustrate the stratification of smoke plumes – how layers may travel at different heights in the atmosphere, remain aloft, or mix at the surface. From downwind swath data, back-trajectory analyses using the methods of Soja et al. (2012) give information about the initial vertical distribution of emissions as well as contributions from multiple fires, if present.

Studies using MISR data found that emissions from most fires (>80%) are injected into the boundary layer, and the remaining smaller percentage of fires inject above the boundary layer (Paugam et al. 2016; Sofiev, Ermakova, and Vankevich 2012; Sofiev et al. 2009; Val Martin, Kahn, and Tosca 2018). Emissions emitted near the surface are subject to local and regional flow regimes (e.g., up-valley and down-valley drainage flows in complex terrain). Emissions injected above the atmospheric boundary layer, although fewer, have longer atmospheric lifetimes and are more available for long-range transport.

A special case where emissions are transported very high into the atmosphere is when large buoyant plumes develop cumulus clouds, releasing latent heat and further enhancing vertical transport. These pyrocumulus (pyroCu) clouds can, in rare cases, develop into thunderstorms, known as pyrocumulonimbus (pyroCb). PyroCb activity and buoyant plumes can inject gases and aerosols into the upper troposphere or lower stratosphere, where they can persist for weeks and months; these emissions can then be transported on a hemispheric scale (Fromm and Servranckx 2003; Fromm et al. 2006; Peterson et al. 2017; Sofiev, Ermakova, and Vankevich 2012). The exact

mechanisms of pyroCb formation are still an active area of debate (Peterson et al. 2017) and research (Lareau and Clements 2016). Although pyroCb are a special subset of smoke plumes, the scope and scale of their emissions and the height of injection have been likened to that of a volcano, and a single event can reduce surface temperatures on a hemispheric scale. Fromm et al. (2010) suggest that some stratospheric aerosol layers previously assumed to be from volcanic eruptions were, in fact, due to pyroCb events.

Once emissions reach a point of neutral buoyancy, transport occurs similar to other atmospheric constituents. Diurnal processes, such as surface heating and cooling, along with regional winds, fronts, and topography, control smoke concentrations near the surface and within the mixed layer. Daytime heating of the surface creates an unstable boundary layer that can dilute smoke concentrations or entrain smoke aloft. At higher wind speeds, the atmosphere becomes more stable, which reduces vertical mixing. Smoke emitted in these conditions can be stratified, tending to transport within the layer it was emitted. As night approaches, the ground cools faster than the atmosphere, creating a near-surface stable layer. Smoke emitted at night near the ground will often stay near the ground. Smoke emitted earlier in the day will remain above in the middle portion of the nocturnal atmospheric boundary layer. In complex terrain such as mountain valleys, daytime heating will create up-valley winds, and then at night, surface cooling will cause the winds to shift and flow down valley (Whiteman 2000). Population centers are often located within valleys, and these nighttime down-valley flows can transport smoke into town, resulting in high concentrations, especially if fuels up valley continue to emit through the night (Miller et al. 2019). In the humid southeastern U.S., smoldering fire emissions along with the higher atmospheric water content (both emitted from the fire and the surrounding atmosphere) can form a thick fog with near-zero visibility conditions (Achtemeier 2006; Bartolome et al. 2019). This smoke can travel along fine-scale topographical depressions (Achtemeier 2005) and has been attributed to catastrophic vehicle collisions (Bartolome et al. 2019).

Smoke and the physical atmosphere are highly coupled. Smoke modifies the radiative properties of the atmosphere by blocking the sun from reaching the surface and absorbing heat and re-emitting that heat to the surrounding atmosphere. This increases atmospheric stability within the mixed layer, makes temperatures cooler near the surface, and reduces both the height of the mixed layer and mixing of smoke through the layer; it may reduce wind speeds as well. In theory, these processes will increase surface concentrations, although there is currently no experimental evidence of this. Absorption of solar radiation by the smoke will also delay the breakup of the nighttime stable layer, maintaining the subsidence inversion much later into the day.

Vant-Hull et al. (2005), Markowicz, Lisok, and Xian (2017), and McKendry et al. (2019) discussed these feedback mechanisms for North American cases. These phenomena have large implications for concentrations, transportation safety, and visibility. For example, during 2017 in the Willamette Valley in Oregon, stagnant air maintained high PM concentrations from nearby fires as lower wind speeds reduced smoke mixing and transport. Other unique examples are smoke-induced density currents that form from differential solar heating between smoke-filled and smoke-free portions of the atmospheric boundary layer. These density currents are relatively common near large wildfires. Lareau and Clements (2015) conducted the first measurements of these density currents, which can spread smoke counter to the ambient wind and over large distances (~30 km), thereby contributing to rapid wind shifts, reductions in visibility, and delayed inversion breakup.

To properly account for these phenomena, the transport model needs to account for radiative effects on the meteorology due to the presence of smoke. Some examples of systems that do this are GEOS-Chem (Bey et al. 2001), WRF-Chem (Grell et al. 2005), and WRF-Community Multi-scale Air Quality (WRF-CMAQ) (Wong et al. 2012). Two operational implementations of these systems are the High Resolution Rapid Refresh (HRRR) forecasting system and recent work modifying the Northwest Regional Modeling Consortium WRF forecast system (Vaughan et al. 2004) to ingest GEOS-5 AOD to modify surface temperatures.

Special cases of transport that have large impacts on fire behavior and downwind air quality are Santa Ana winds (Kolden and Abatzoglou 2018; Langford, Pierce, and Schultz 2015; Mensing, Michaelsen, and Byrne 1999; Westerling et al. 2004), Diablo winds (Mass and Ovens 2019), and Sundowner winds (Blier 1998). Santa Ana winds are strong northeasterly winds with low relative humidity that occur in Southern California. Diablo winds (Smith, Hatchett, and Kaplan 2018) are north winds occurring in northern California, typically overnight, characterized by high wind speeds and low relative humidity but not necessarily higher temperatures. These winds promoted the rapid spread of the 2017 northern California Wine Country fires (Mass and Ovens 2019). Santa Ana and Diablo conditions set up when high pressure over the intermountain west produces an offshore pressure gradient (Mass and Ovens 2019).

Sundowner winds are another case of strong downslope flows that enhance fire behavior. They occur in the Santa Ynez mountains near Santa Barbara, CA, typically in the late spring, with onsets in the late afternoon to early evening, giving them their name (Blier 1998; Hatchett et al. 2018). They are characterized by high wind gusts and low relative humidity, and one notable result of these conditions is that they promote fire growth that is different from typical or expected fire activity. Wildfire activity is assumed to be greatest mid-afternoon when temperatures peak, solar radiation is maximized, and atmospheric instability is greatest. This translates to the rule of thumb that the greatest fire emissions occur mid-afternoon. Sundowner wind transport processes show this is not always the case: Sundowner winds promote increased fire activity and emissions in the evening hours. Although Sundowners might seem regionally specific, they have been responsible for some of the biggest wildfire losses in terms of lives and property in recent history, with widespread smoke impacts affecting millions of people (e.g., Mass and Ovens 2019). Mass and Ovens also point out that high-resolution meteorological forecasting can help identify high fire risk conditions in these situations. The GOES-16 satellite data, which includes fire detection data every 5 minutes for the continental U.S., can show fire progression for large wildfires; for the 2017 and 2018 northern California wildfires, they demonstrated that typical diurnal fire patterns do not hold. These data can be applied to create more accurate fire diurnal profiles.

Chemical processing of smoke

Once released, the gases and particulate matter in smoke evolve through a multitude of complex chemical processes. A key challenge for understanding this processing is the large variability in emissions. No two fires are the same, and thus the chemical evolution is also different.

Changes in aerosol mass and composition during smoke aging

Once released, organic aerosol can lose mass, through evaporation or volatilization, or gain mass, through formation of secondary organic aerosol (SOA). SOA formation occurs due to oxidation of VOCs. Oxidation adds organic functional groups, which lowers the vapor pressure of the compounds, or it can cleave C-C bonds, which can increase the vapor pressure of the existing aerosol compounds (Kroll et al. 2009). SOA production from biomass burning aerosols can also occur in the aqueous phase, when aerosols deliquesce or are associated with fog, although a clear mechanistic understanding is presently lacking (Gilardoni et al. 2016). As the aerosol moves with a smoke plume, we can monitor the enhancement ratio (ER) as $\Delta X/\Delta CO$ to identify physical or chemical production or loss of components (e.g., ΔX). CO is typically used in the denominator of this ratio, because CO concentrations are strongly enhanced in the smoke plume compared to background concentrations, and CO undergoes only slow loss by reaction with OH (the CO lifetime with respect to loss is ~2 weeks). Thus, CO can act as a relatively inert indicator for dilution. For plumes with no production or loss of component X, dilution affects both compounds similarly, and the enhancement ratio remains constant.

However, aerosol/CO ratios are highly variable. Some observations suggest aerosol production and others suggest aerosol loss (e.g., Briggs et al. 2016). Hodshire et al. (2019a) summarized an extensive dataset of field and lab observations on SOA enhancements. The field observations suggest, on average, that aerosol loss appears to be largely balanced by SOA production. In contrast, the laboratory data suggest that SOA production dominates (increasing the aerosol/CO ratio over time). May et al. (2014) discussed the lab/field discrepancies and attributed some of these differences to dilution, which can increase the organic aerosol evaporation.

Chemical changes in the smoke aerosol can also give information on its processing and evolution. A key tool for this is high resolution aerosol mass spectrometry (HR-AMS), which can resolve molecular fragments from the biomass burning aerosol (Zhang et al. 2018). The molecular fragments at a mass to charge (m/z) ratio of 60 are thought to be associated with leuvoglucosan, a tracer of biomass smoke, along with other similar compounds. The peak at an m/z of 44 is due to the "C-O-O" molecular fragment. The ratio of the peak areas at m/z values of 60 and 44 to the cumulative peak areas in the mass spectra are termed F60 and F44, respectively. Aiken, DeCarlo, and Jimenez (2007) showed that F44 is correlated with the O/C ratio of the aerosol. Observations indicate that, with aging of biomass burning aerosol, F60 tends to decrease while F44 increases, and these go along with changes in the O/C ratio (Garofalo et al. 2019; Zhou et al. 2017). Fresh smoke aerosols have O/C ratios of ~0.35, whereas aged, highly oxidized smoke aerosols have O/C ratios greater than 1 (Zhou et al. 2017). Liu et al. (2016) also found rapid changes in the O/C ratios for prescribed burns, with values increasing from around 0.4 to 0.6 in less than an hour. So, even if the mass of smoke PM shows

relatively little change during aging, the composition moves toward a more oxidized aerosol. This more oxidized aerosol may have greater health impacts (Tuet et al. 2017; Wong et al. 2019). The simultaneous loss and production of biomass PM can coexist due to the combined processes of primary aerosol evaporation and SOA production (Hodshire et al. 2019b).

A number of studies have identified organic carbon from biomass burning as a dominant component of summertime $PM_{2.5}$ in rural areas of the western U.S., and this can explain the large interannual variability in $PM_{2.5}$ concentrations (Holden et al. 2011; Jaffe et al. 2008; Schichtel et al. 2017; Spracklen et al. 2007). In the southeastern and midwestern U.S., fires make a significant, albeit smaller, contribution to particulate organic carbon. Here, the seasonality is slightly different, with spring the highest period and prescribed fires the dominant fire type (Nowell et al. 2018; Schichtel et al. 2017; Zeng et al. 2008).

Ozone production in smoke plumes and urban areas

Ozone (O_3) is a secondary pollutant that is formed from the oxidation of VOCs in the presence of nitrogen oxides and UV light. Since fires emit NOx and VOCs, in variable amounts, O_3 may be formed in a smoke plume, but this will depend on emissions, temperature, UV light, and many complex interactions within the plume. The many factors involved give rise to large variations in the O_3 production found in smoke plumes. Under warmer conditions, O_3 can form in a matter of hours (Akagi et al. 2013; Baylon et al. 2015; Hobbs 2003), whereas in cooler environments, O_3 production takes longer and may not be apparent for several days (e.g., Alvarado et al. 2010). Rapid O_3 production is likely driven by several sources of oxidants, including OH from HONO (nitrous acid) photolysis. HONO can be either emitted directly (Burling et al. 2010; Veres et al. 2010) or produced from heterogeneous reactions (Alvarado and Prinn 2009; Ye et al. 2017). One important control on O_3 production is the amount of NOx emitted and subsequently removed by chemistry (Mauzerall et al. 1998). NOx in boreal smoke plumes is rapidly sequestered as peroxyacetyl nitrate (PAN) (Alvarado et al. 2010; Jacob et al. 1992). A similar result was found for smoke plumes at the Mt. Bachelor Observatory in central Oregon, at 2.8 km above sea level (Baylon et al. 2015; Briggs et al. 2016). In a review of more than 100 studies, Jaffe and Wigder (2012) found that O_3 is usually enhanced downwind from fire plumes, and the production increases with plume age. Tropical and sub-tropical fires generally make greater amounts of O_3 and make it faster than do temperate/boreal fires. This arises because tropical and sub-tropical fires emit more NOx per unit of fuel, and the higher temperatures discourage PAN formation. Nonetheless, PAN is only a temporary reservoir; subsequent thermal decomposition will regenerate the original NOx back and distribute O_3 production further downwind.

When a smoke plume mixes into an urban area, it will mix in all the components of the plume, but it will also change the local photochemical environment. Thus, urban O_3 from smoke could be due to upwind O_3 production or through new O_3 production in the urban environment, since optimum O_3 production occurs at a VOC/NOx molar ratio of around 8 (Qian et al. 2019). Most urban areas are near this or have lower ratios (e.g., if NOx rich). Fire emissions typically have high VOC/NOx molar ratios (e.g., ~10-30) (Akagi et al. 2011; Andreae 2019), so when smoke mixes into an urban area, it can move the region closer to this optimum O_3 production regime. There are large variations in this behavior by region, fire emissions, meteorology, and other factors. Buysse et al. (2019) showed that enhanced O_3 in urban areas (due to wildland fires) is most pronounced at $PM_{2.5}$ concentrations below about 60 ug/m^3. At higher $PM_{2.5}$ concentrations, O_3 levels appear to be suppressed, due either to reduced photolysis rates (Alvarado et al. 2015) or to heterogeneous chemistry on smoke particles (e.g., Konovalov et al. 2012). High $PM_{2.5}$ could also indicate insufficient reaction time. Photolysis can be complex, because there can be multiple scattering influences, and photolysis rates will depend on the location within the plume (Alvarado et al. 2015). At high smoke levels, photolysis will be diminished, but at moderate smoke levels and with high scattering amounts, photolysis may not be significantly reduced inside a smoke plume (Baylon et al. 2018).

Multiple approaches have been used to estimate O_3 production in smoke plumes. Many studies have compared concentrations in and outside a plume. Lindaas et al. (2017) documented enhancements in O_3 of around 15 ppb in Colorado associated with transported smoke plumes. Liu et al. (2016) found that O_3 can be produced downwind of southeastern U.S. agricultural fires. Significant impacts on surface O_3 via intercontinental transport wildfire emissions can also occur, for example, from Siberian smoke reaching the western U.S. (Jaffe et al. 2004; Teakles et al. 2017) or Alaskan smoke reaching the north Atlantic (Real et al. 2007). Canadian wildfires have been found to enhance O_3 in the southeastern U.S. (McKeen et al. 2002), Maryland (Dreessen, Sullivan, and Delgado 2016), and New

England (DeBell et al. 2004). Using a statistical approach, Gong et al. (2017) found that smoke raises the O_3 MDA8 by 3–6 ppb on average, with a maximum enhancement of up to 40 ppb for 6 cities in the western U.S. Using a similar approach, Gao and Jaffe (2020) found an average enhancement in the MDA8 of 7–10 ppb for 5 cities in the Pacific Northwest, with a maximum enhancement of 50 ppb during the large 2017 smoke events. The western U.S. fires in 2017 and 2018 led to the highest MDA8 values seen in the last few decades in Enumclaw, WA, Portland, OR, and Sacramento, CA. During an especially smoky summer in Boise, ID, smoke increased the O_3 MDA8 by an average of ~15 ppb (McClure and Jaffe 2018). The smoke also increased the number of days over the 8-hour 70 ppb air quality threshold.

While O_3 production is driven by UV photolysis in the daytime, chemical processing can still occur at night, although much less is known about this. From other (non-smoke) studies, we know that NO_2 and O_3 will react to form the NO_3 radical, which can oxidize many organic species and further react to form N_2O_5 (nitrogen pentoxide). Ahern et al. (2018) found that nighttime processing in smoke generates both N_2O_5 and $ClNO_2$ (nitryl chloride), both of which regenerate NO_2 through photolysis; $ClNO_2$ can also generate reactive Cl radicals, which are important oxidants in some circumstances. Finewax, de Gouw, and Ziemann (2018) and Decker et al. (2019) demonstrated several nighttime reactions, mostly through the NO_3 radical, which can significantly modify the overall reactivity of aerosols, VOCs, and O_3. At present, the full suite of nighttime chemistry is not understood and therefore not well represented in models.

An important question is whether the most common regulatory measurement of O_3, made using UV FEM monitors, exhibits interferences during major smoke events. This was suggested by laboratory studies on possible interferences in the UV method (Payton 2007). However, Gao and Jaffe (2017) compared the UV method with the FEM approach for O_3 (nitric oxide chemiluminescence) and found that these gave nearly identical results in smoke plumes with up to 134 μg/m^3 of $PM_{2.5}$ and O_3 concentrations up to 83 ppb.

Smoke modeling

Accurate modeling of primary emissions and secondary pollutants is desirable to understand the chemical processing and the impacts on human health (Brown et al. 2014). Smoke forecasting systems have been built to predict air quality impacts. These include both statistically based systems that use observations, and historical air quality relationships and dynamic models that simulate the underlying physics and chemistry of the fire, plume, and atmosphere. Forecasts usually project forward 1 to 3 days into the future, similar to short-term weather forecasts, with a few systems extending further out. Inputs to such systems are generally satellite fire detections and predictions from weather forecast models.

Statistically based forecast models that predict daily average PM concentrations are run daily by the British Columbia Center for Disease Control for western Canada (Yao and Henderson 2014) and the USFS Interagency Wildland Fire Air Quality Response Program (IWFAQRP) for the western U.S. (Marsha and Larkin 2019). These models rely on empirically derived relationships between ground monitoring data (typically $PM_{2.5}$) and other measures of nearby fires (satellite fire detections) and smoke (satellite-derived smoke plume extents and AOD). Statistical models generally show good performance for locations with an existing history of observations on which to train the statistical relationship. For example, Lightstone, Moshary, and Gross (2017) showed that a trained neural network outperformed a 3-D chemical transport model (CTM) for the state of New York and responded more rapidly, especially during transient events such as wildfires.

Dynamical modeling systems require simulating a chain of logic that implicitly or explicitly identifies where the fires are, what the available fuels are, how much of these fuels will burn, how high up in the atmosphere the plume will rise, and then where the plume will be transported (Goodrick et al. 2013; Strand et al. 2018). In certain cases, such as emissions estimates calculated directly from fire radiative power, several of these steps are combined into a single parametric relationship. Some systems focus solely on the smoke plume, using a particle or puff modeling system such as HYSPLIT (Stein et al. 2015). Some also include the chemical transformation of the plume as it reacts with other pollutants in the atmosphere, typically by the use of the CMAQ (Byun and Schere 2006) or the WRF-Chem (Grell et al. 2005) or WRF-CMAQ (Wong et al. 2012) models. WRF-Chem and WRF-CMAQ can be run in a coupled mode that includes feedbacks between the meteorology and atmospheric chemistry, including explicitly treating smoke's effect on the radiative process that can influence the overall atmospheric structure (Grell et al. 2011). Other models, such as the WRF-SFire (Mandel et al. 2014), resolve the coupling between the meteorology and the fire and the development of the close-in buoyant smoke plume, but these

models are usually run at fine scales (meters to tens of meters) in limited domains that preclude modeling of the full smoke plume for air quality purposes. However, fully coupled atmosphere-fire-chemistry models such as WRF-SFire-CHEM (WRFSC; Kochanski et al. 2015) hold promise as future operational forecasting models as computing power and model development continues (Prichard et al. 2019a).

Over the U.S. and Canada, daily smoke forecasts are generated by a number of agencies and universities, with each system having different setups, strengths, and designed uses. Official air quality forecasts are generated by Environment Canada using the FIREWORK system (Chen et al. 2019; Pavlovic et al. 2016), which uses a photochemical model that includes emissions from fires and industrial sources to forecast across a North American grid at 10-km resolution. In the U.S., NOAA's National Air Quality Forecast Capability (https://www.weather.gov/sti/stimodeling_airquality) generates operational smoke forecasts using CMAQ on a 12-km resolution, which covers all of North America (Lee et al. 2017; Stajner et al. 2012). NOAA also produces an experimental High Resolution Rapid Refresh-Smoke model (HRRRS; Grell et al. 2011), which uses WRF-Chem at a 3-km resolution over the continental U.S.; HRRRS is updated hourly, but treats smoke as a passive tracer. Washington State University runs the regional AIRPACT-5 CMAQ forecasts at resolutions down to 1.33 km over the Pacific Northwest, and Georgia Tech runs a CMAQ forecast system down to a 4-km resolution for the southeastern U.S. The USFS IWFAQRP runs over 30 smoke models aimed at public health, transportation safety, and firefighter safety, using the BlueSky Smoke Modeling Framework (Larkin et al. 2009) and HYSPLIT or CMAQ, at resolutions down to 1 km; these runs can also incorporate specific incident decision scenarios. The result is that locations across the U.S. fall within at least three and potentially over eight different smoke forecast model domains. Additional tools, such as NOAA's Air Research Laboratory HYSPLIT website (https://www.ready.noaa.gov/HYSPLIT.php), the USFS IWFAQRP's BlueSky Playground web tool (https://tools.airfire.org), and the Canadian BlueSky Playground web tool (http://firesmoke.ca) allow for customization of emissions and parameters before computation of a customized trajectory and dispersion model result, typically using the HYSPLIT model.

The large number of smoke forecasting systems exemplify both the difficulties in developing the input information needed and the myriad ways to process emissions, plume rise, dispersion, transport, and chemistry. Higher resolutions typically result in better results for wind forecasts in areas of complex topography (e.g., Mass et al. 2002), but more defined meteorology beyond a 3-km resolution is available only for specific regional domains. Full chemistry CTMs may provide better PM results by including all sources (e.g., fires, anthropogenic emissions, and natural sources) and by including the formation of SOA and ozone. But CTMs require substantially more computing power per modeled grid cell than smoke-only models. Inclusion of coupled mechanisms between the atmosphere and smoke plume, or between the atmosphere and fire plume, exacerbates the need for more computing power. Model differences also occur due to large uncertainties in fire emissions. The choice of fire information sources is one of the largest differentiators in the overall computation of emissions (Larkin et al. 2020; Larkin, Raffuse, and Strand 2014), which in turn sets the overall level of smoke within the model.

There have been relatively few analyses examining smoke forecasting system performance for predicting ground-level $PM_{2.5}$ concentrations. A few analyses have looked at overall performance, with mixed results, and at specific processes that may contribute to large uncertainty (Larkin et al. 2012). Herron-Thorpe et al. (2014) reported on performance of the AIRPACT modeling system for $PM_{2.5}$ and found that it gave both overestimates, near fires, and underestimates further away. These discrepancies were likely due to inadequate SOA production in the chemistry model; errors in fire detections, assigned fire sizes and fuel loadings; and the large uncertainty associated with the vertical distribution of emissions. In a hindcast case study examining the Wallow fire in Arizona and rangeland fires in South Dakota, Baker et al. (2016) found a model overestimation bias up to approximately 20 $\mu g/m^3$ for $PM_{2.5}$, but performance was limited by the fire inputs and the chemistry representation used. Zhou et al. (2018) found that higher estimates of buoyancy heat flux produced plume rise values similar to measured plume top data from aircraft sampling plumes from crop-residue burning in the northwestern U.S. Yang et al. (2011), Garcia-Menendez, Hu, and Odman (2013), and Miller et al. (2019) found that errors in the weather forecast data are critically important in affecting overall smoke model performance. Small errors in geolocation of fires and/or the vertical distribution of emissions can significantly affect model performance (Garcia-Menendez, Hu, and Odman 2014). Larkin et al. (2012) found that diurnal timing (e.g., hourly allocation of emissions) was also an important factor in determining smoke forecasting system performance. While all these processes are fundamentally important to smoke system performance, if transport processes (as simulated by the

meteorological dataset) do not carry the smoke in the correct direction, then smoke modeling systems may not provide useful information, even if all other components are estimated perfectly.

An additional challenge for modeling future air quality is knowing how a fire will behave in the near term. Most smoke forecast systems use a simple persistence assumption for fire occurrence and growth, assuming that fire emissions in the next few days will be similar to the current day. Development of a reliable fire growth model for predicting actual area burned is still an active area of study within the fire community.

The current plume rise calculations used in smoke forecasting models have also been identified as major sources of uncertainty (Stein et al. 2009; Larkin et al. 2012; Raffuse et al. 2012; Val Martin et al. 2012; Zhou et al., 2018; Liu et al. 2019). Using more resolved modeling techniques, such as found in coupled fire-atmosphere models, can more accurately model the plume structure and dynamics and may lead to significant improvements in smoke forecasting. This area, and the need for a robust observational dataset of the myriad of fire and atmospheric variables related to the complex plume dynamics at work, have been identified as a major need (Liu et al. 2019; Prichard et al. 2019b). Despite these obstacles and limits on quantitative forecasts, smoke prediction models generally do well in modeling overall plume extent and shape compared with satellite-derived plume extents (e.g., Chen et al. 2008; Rolph et al. 2009; Strand et al. 2012), and they are important tools for community preparedness.

Data fusion techniques combine satellite data, surface observational data, and modeling outputs to produce an improved estimation of pollutant exposure and human health impacts. These techniques capitalize on the strengths of each tool and seek to reduce the limitations associated with the individual datasets. For example, observational data give the best available estimate of $PM_{2.5}$ at a few locations but are sparse across large portions of a domain. Satellite AOD are regionally coherent but do not indicate what is at the ground, and they have issues at night or when clouds obscure the measurement. CTMs provide 4-D output but are based on model assumptions and inputs, which may or may not represent reality. Data fusion methods range from linear regression relationships between AOD and surface $PM_{2.5}$ (e.g., Engel-Cox, Hoff, and Haymet 2004; Wang and Christopher 2003) to statistical algorithms that incorporate meteorological data (e.g., Gupta and Christopher 2009), land use information (e.g., Hu et al. 2014), and CTM outputs (e.g., Liu et al. 2004; van Donkelaar et al. 2010). Several datasets of surface $PM_{2.5}$ concentrations from fusion methods are publicly available (Diao et al. 2019). Recently, data fusion techniques have been specifically applied to improve estimates of wildfire smoke impacts (Gan et al. 2017; Lassman et al. 2017; Reid et al. 2015; Yuchi et al. 2016; Zou et al. 2019). These approaches used a combination of surface $PM_{2.5}$ observations, satellite AOD, meteorological and land use data, and CTM outputs. Yuchi et al. (2016) used forecast model output from the Canadian FireWork and BlueSky systems, while the other wildfire data fusion studies used retrospective CTM simulations.

Chemical modeling: Chemical transport models, Lagrangian plume models, and statistical modeling

The discussion above focused on modeling the emissions and transport of smoke. In this section, we focus on various strategies used to model and understand the chemical interactions during smoke transport.

Multiple approaches have been used to model chemical interactions in smoke plumes: gridded CTMs (described above), Lagrangian plume (or box) models, and statistical methods. Each has some advantages but also presents a unique set of challenges. CTMs characterize the chemical environment in three dimensions over time. Modeling O_3 and SOA production in a CTM first depends on accurately knowing the flux, timing, and location of the primary emissions (e.g., PM, NOx, HONO, CO, VOCs). Modeling the resulting concentrations requires spatial and temporal knowledge of the injection heights, 3-D wind fields, and other meteorological parameters (e.g., temperature and RH; Cai et al. 2016; Garcia-Menendez, Hu, and Odman 2013, 2014; Herron-Thorpe et al. 2014; Kochanski et al. 2015; Koplitz et al. 2018; Pfister, Wiedinmyer, and Emmons 2008). For secondary PM and O_3, the model must also include a detailed chemical mechanism and UV radiation fields.

A key component in CTMs is the grid resolution. Smaller grid size means greater spatial resolution, but this also increases the computational demands due to the increased number of grid cells horizontally. For a primary pollutant, even if the spatial distribution is not well described, the integrated flux downstream can still reflect the emission flux, assuming no loss or production; thus, we expect that model calculations of column-integrated quantities will be better than point comparisons. But this does not hold for secondary species, especially O_3 and possibly SOA. Grid size is especially important for wildfire O_3 production, since this is known to be non-linear with NOx and VOCs (Wu et al. 2009). Here, secondary production is non-linearly related to the concentrations.

Accurate modeling of O_3 using CTMs is particularly challenging. Wildland fires are known to have large emissions of acetaldehyde, a PAN precursor, and this results in rapid sequestration of NOx. The degree to which a model captures this process will depend critically on its spatial resolution and, of course, the accuracy of its emissions. Models that over-predict the NOx emissions and/or under-predict acetaldehyde will probably over-predict O_3 close to the fires, and this is a common pattern seen in CTM predictions of O_3 production from fires (e.g., Baker et al. 2016; Jaffe et al. 2013; Zhang et al. 2014).

Other important nitrogen species are HONO and NH_3. Direct fire emissions of HONO (e.g., Burling et al. 2010; Veres et al. 2010) will be a source of OH radicals, through daytime photolysis, and this provides an early-morning oxidant to stimulate VOC loss and O_3 production. Recent observations from the WE-CAN experiment show that, on average, western fires' emissions of NH_3 were larger than NOx (Lindaas et al. 2019). Further, some fires have large emissions of HONO, which can contribute to rapid O_3 production (Palm et al. 2019). Both observations challenge our current understanding of the EFs and O_3 production for western wildfires.

An additional challenge for CTMs is the large number of VOCs and oxygenated VOCs that are emitted by wildland fires; the vast majority of these compounds are not included in standard chemical mechanisms. For example, it has been calculated that furans (5-carbon aromatic compounds) are important sources of SOA and can be responsible for 10% of the O_3 production in smoke plumes (Coggon et al. 2019), but furans are not included in most chemical mechanisms. Given the enormous number of VOCs identified in biomass burning plumes – more than 500 so far (Hatch et al. 2017) – it is necessary to simplify the reaction scheme, but at present the implications of these simplifications are not understood. Despite the many challenges in modeling O_3 from wildland fires, one important advantage of CTMs is that all sources (e.g., multiple fires, industrial emissions) can be modeled simultaneously for all receptor locations, and the contribution from each source can, in theory, be teased out of the results.

To overcome the challenges of grid resolution and accurately simulating transport, a number of studies have successfully used box models (e.g., Wolfe et al. 2016). In this approach, a hypothetical box (or airmass) is identified whereby detailed chemistry is simulated in the box as it moves downwind with the prevailing wind in a Lagrangian framework. Usually the concentrations in the box can be initialized with observations and dilution rates. There are several variations in this approach, but these generally do better at simulating O_3 production compared to CTMs (e.g., Alvarado et al. 2015; Coggon et al. 2019; Mason et al. 2006; Müller et al. 2016). One advantage of box models is that a more complex chemical scheme can be incorporated, since only one grid cell need be simulated. An additional advantage is that by simulating the emissions from a single fire plume, more accurate representation of the emissions can be incorporated, and transport is essentially removed as an uncertainty (the box follows the prevailing plume direction). In the future, box models for individual plumes could be embedded in CTMs as a means to carry out higher-resolution chemistry simulations, which can then pass this information on to the larger scale CTM (Karamchandani et al. 2014).

Statistical models take a completely different approach. These attempt to model or "predict" the O_3 concentrations (hourly or 8-hour average) using machine learning tools. A variety of meteorological indicators are used to predict O_3 concentrations (e.g., daily maximum temperature, vector winds, 24-hour backward trajectories, relative humidity, 500 mb geopotential height). This approach uses either multiple linear regression (e.g., Jaffe et al. 2013; Lu et al. 2016) or Generalized Additive Models (GAMs; e.g., Camalier, Cox, and Dolwick 2007; Gao and Jaffe 2020; Gong et al. 2017; Jaffe et al. 2018). A typical method splits the available data into a non-smoke training dataset, an evaluation or cross-validation dataset, and a smoke dataset. The difference between the prediction from the non-smoke training set and the actual observation then gives an indication of the contribution to O_3 due to the fire emissions. In practice, these models can give predictions for the O_3 MDA8 for non-smoke days with R^2 values of between 0.5 and 0.8; they suggest that, for urban environments, the average contribution on smoke days to the MDA8 is 3–10 ppb (depending on the city), with a maximum contribution in some extreme cases of up to 50 ppb. These models have the advantage of being simpler to apply then the CTM approach and give statistically robust predictions that have been used to support EPA exceptional event designations (see discussion on regulatory impacts in Section 8). On the other hand, a statistical model does not clearly indicate cause and effect.

Health effects of smoke

Smoke from fires is a health concern in the communities near and downwind from the source (Larsen et al. 2018). For the continental U.S., a health burden assessment estimated that, for 2008–2012, 3900–6300

respiratory hospitalizations and 1700–2800 cardiovascular hospitalizations could be attributed annually to short-term smoke exposures (Fann et al. 2018). Since 2012, the U.S. has experienced smoke levels that exceeded any previously recorded seasons, thus likely increasing the health burden.

Smoke is composed of many harmful components, but $PM_{2.5}$ is usually considered the most important concern for public health, and most epidemiological and toxicological studies have focused on this pollutant. The scientific literature on the health impact of smoke is still limited compared to studies of exposure to general ambient and indoor air pollution. Studies of urban pollutants provide valuable insights into the biological mechanisms that play a role in developing adverse health outcomes. However, during wildfire events, concentrations are substantially higher and mixtures contain different air pollutants. During wildfires, exposures are typically an order of magnitude greater than in typical ambient settings, while during prescribed burning events, exposures are closer to typical ambient exposures. Further, there is evidence that smoke PM is more toxic than typical urban PM (Wegesser, Pinkerton, and Last 2009). Both short-term and long-term exposures have been associated with health risks.

The scientific literature related to wildfire health effects is rapidly growing. Much of the current evidence has been synthesized in recent reviews (Adetona et al. 2016; Black et al. 2017a; Liu et al. 2015a; Reid et al. 2016a; Youssouf et al. 2014) and quantitative meta-analyses (Borchers Arriagada et al. 2019; Fann et al. 2018). Substantially less research has been done on the health impacts arising from prescribed burning. This is an important gap in knowledge, because increased burning is a key land management strategy for reducing the risk of wildfires and maintaining ecosystem benefits. By its nature of being planned, prescribed burning may provide an opportunity to reduce the health risks of smoke, but without fully understanding the health impacts, these risks cannot be quantified.

Many studies have shown the relationship between wildfire smoke exposure and adverse respiratory effects. The most consistent evidence is documented in the analysis of administrative data, through increased respiratory-related emergency department visits, physician visits, and hospitalizations (Chen, Verrall, and Tong 2006; Delfino et al. 2009; Henderson et al. 2011; Ignotti et al. 2010; Johnston et al. 2014; Lee et al. 2009; Martin et al. 2013; Moore et al. 2006; Morgan et al. 2010; Mott et al. 2002; Rappold et al. 2011; Tham et al. 2009; Thelen et al. 2013; Yao, Eyamie, and Henderson 2016). These studies are population-based with a good representation of the affected population and have been replicated in multiple locations.

Particularly strong evidence links smoke exposure to exacerbations of asthma and chronic obstructive pulmonary diseases. There is also growing evidence of other respiratory outcomes, including acute bronchitis, pneumonia, and upper respiratory infections several days following exposure (Reid et al. 2016b; Tinling et al. 2016). Gan et al. (2020) examined asthma-related outcomes in the out-of-hospital setting and reported increased usage of medication and visits to emergency department, ambulatory care, and outpatient clinics. Studies of health impacts in out-of-hospital settings are rare, but they provide important evidence on the extent of the health burden in the population, and they signify that the extent of health outcomes currently documented likely underrepresents the total health burden.

Cardiovascular health

Outcomes related to the circulatory and cardiovascular system are of significant concern during smoke episodes because of their known causal link with $PM_{2.5}$ exposure. In the presence of environmental irritants such as wildfire smoke, existing circulatory diseases can more easily trigger ischemic events such as heart attacks and stroke, worsening heart failure, or abnormal heart rhythms. These conditions are serious health events that lead to emergency department visits, hospital admissions, and even death. Early systematic reviews called the evidence of cardiovascular effects mixed or inconsistent, but this evidence has been rapidly increasing in recent years. For example, all 10 studies reviewed for evidence in all-cause cardiovascular outcomes in Reid et al. (2016a) found no statistically significant changes in risk; however, when the associations were examined by specific cardiovascular outcomes, approximately half of these studies reported an increased risk of congestive heart failure, ischemic heart disease, hypertension, and/or acute myocardial infraction, and two-thirds reported an increased risk of cardiac arrest and apnea. Additional evidence for all-cause and cause-specific cardiovascular outcomes was reported by Wettstein et al. (2018), DeFlorio-Barker et al. (2019), and Yao et al. (2019). This growing body of evidence could be attributed to the use of more comprehensive exposure metrics (e.g., air quality chemical transport models, satellite data, dispersion models, data fusion) and the increasing ability to examine cause-specific outcomes from administrative databases (e.g., myocardial infraction, congestive heart failure).

Risk of mortality from smoke exposure

Studies on short-term smoke exposures have consistently found a positive association for all-cause mortality and, to lesser extent, a positive association with

cardiovascular and respiratory causes (Liu et al. 2015a; Reid et al. 2016a; Youssouf et al. 2014). The strongest evidence is found in time-series and multi-city studies whose results have been replicated in locations around the world, including Australia (Johnston et al. 2011; Morgan et al. 2010), Europe (Analitis, Georgiadis, and Katsouyanni 2012; Faustini et al. 2015; Kollanus et al. 2016; Linares et al. 2018, 2015), Canada (Yao et al. 2019), and the U.S. (Doubleday et al. 2020).

Evidence for association with mortality due to respiratory and cardiovascular causes is less consistent than for all-cause mortality. Among the studies that examined all-cause, respiratory, and cardiovascular effects on mortality (Analitis, Georgiadis, and Katsouyanni 2012; Faustini et al. 2015; Johnston et al. 2011; Kollanus et al. 2016; Linares et al. 2018; Morgan et al. 2010), only one study (Analitis, Georgiadis, and Katsouyanni 2012 found positive associations with both causes). Among the other five studies, none found associations with respiratory causes of mortality, and three reported significant associations with cardiovascular causes (Faustini et al. 2015; Johnston et al. 2011; Kollanus et al. 2016). Kollanus et al. (2016) found evidence for the effects of long-range transport of smoke plumes on daily mortality in the city of Helsinki over a 10-year period. In another long-term study of daily mortality rates, Doubleday et al. (2020) reported significant changes in risk for all-cause mortality and respiratory mortality over a 12-year period in the state of Washington.

Other health outcomes and exposures

The acute effects of long-term exposure to smoke, as well as the chronic effects of both short- and long-term exposures, have not been characterized, even though considerable evidence exists on ambient and indoor air pollution. Chronic effects such as birth outcomes, neurological effects, diabetes, and the progression of various diseases are best studied in cohort designs, where individuals are enrolled and followed through time. However, such studies have not yet been established to monitor long-term smoke impacts on health.

Psychological effects of wildfires have been documented (Caamano-Isorna et al. 2011; Papanikolaou et al. 2011), but few studies have focused on psychological effects of smoke exposure. In a review by Reid et al. (2016a), only two smoke-specific studies were evaluated and both yielded largely null findings (Duclos, Sanderson, and Lipsett 1990; Moore et al. 2006). More recently, Dodd et al. (2018) examined effects of smoke on the mental, emotional, and physical well-being of a community in the Northwest Territories, where a prolonged episode of smoke led to evacuations and disruptions of daily lives. Fear, stress, and uncertainty contributed to acute and long-term negative impacts on mental health. As smoke in communities increases, it becomes more important to understand the emotional and social toll on individuals and communities to be able to build successful responses.

The effects of maternal exposure to $PM_{2.5}$ during pregnancy have also been reported, but they have not been studied extensively in ambient or wildfire smoke exposure settings. The strongest evidence of adverse birth outcomes is linked to studies of indoor exposure to biomass burning (e.g., cooking, heating); however, those exposures are typically both longer and more acute than wildfire smoke in populations. Only a handful of epidemiologic studies on prenatal exposure to $PM_{2.5}$ have been conducted. Holstius et al. (2012) found a small reduction in average birth weight among infants exposed to $PM_{2.5}$ in utero, and Abdo et al. (2019) reported a positive association between $PM_{2.5}$ exposure and both the incidence of pre-term birth and lower birth weight. The 2008 northern California wildfires led to an unintended experiment in which a cohort of infant primates in the California National Primate Research Center were exposed to a prolonged episode of smoke, while another cohort lived indoors in the same research facility with filtered air. Three years after the exposures, the exposed primates had lower lung volumes compared to age-matched primates who were not exposed. Follow-up studies in this cohort have provided valuable evidence that prolonged smoke exposure can result in chronic effects (Black et al. 2017b).

Communities and individuals of lower socio-economic status have been reported as more vulnerable to higher personal exposure and increased risk of adverse health outcomes from both urban air pollution and smoke (Rappold et al. 2012; Reid et al. 2016b). Increased exposures have been attributed to lack of financial means to reduce exposure (e.g., installing all-house air conditioning, purchasing a HEPA filter unit), differential occupational exposure based on type of employment, and differential indoor exposure due to housing characteristics. The largest wildfires tend to occur in rural areas, where air conditioning and airtight housing is not prevalent, so the exposure differential with respect to socio-economic position may be even larger than in urban settings. However, assessment of personal exposure is time-consuming and expensive; thus, limited data exist on levels of exposure indoors and the ability to improve indoor air quality during wildfires through interventions for different socio-

economic groups. Socio-economic factors also lead to increased susceptibility to adverse health effects during wildfire exposure because of reduced access to health care, cumulative stress, and insufficient control of underlying health conditions (e.g., asthma, diabetes, heart failure).

Exposure in occupational settings (e.g., firefighters, outdoor workers) is often greater than in the general population because of proximity to the fires, prolonged periods of exposure, and increased exertion rates, which increase the total deposition of air pollutants in lungs. High levels and exceedances of permissible occupational exposure limits have been reported during work shifts with respect to particulate matter, gases, diesel, and hazardous air pollutants (HAPs: acrolein, benzene, formaldehyde, and polycyclic aromatic hydrocarbons) (Broyles 2013; Naeher et al. 2007; Reinhardt and Broyles 2019; Romagnoli et al. 2014). Several studies of occupation exposure reported acute phase effects, such as declines in lung function, increased urinary metabolites of HAPs, and indicators of systematic inflammation in blood (Adetona et al. 2017, 2019). Semmens et al. (2016) surveyed wildland firefighters and examined the association between the duration of their careers and self-reported health outcomes; many reported physician-diagnosed heart arrhythmia. However, neither acute nor chronic health effects in occupational exposure have been characterized systematically enough to understand the total burden of such occupational exposure to smoke.

In addition to $PM_{2.5}$ (Naeher et al. 2007; U.S. EPA, 2009), smoke contains HAPs (Reinhardt and Ottmar 2004), isocyanic acid (Roberts et al. 2011), VOCs, O_3, and other pollutants that have been associated with health risks. Carbon monoxide inhibits the body's ability to transfer oxygen to the heart, brain, and other organs, and HAPs are known carcinogens. However, these pollutants are rarely measured at the population level; consequently, their contribution to the overall health burden is not quantified in epidemiology or risk assessment. Structural fires can result in particularly toxic smoke and ash due to the burning of household items such as plastics, metals, and other synthetic materials, which can also generate water quality concerns if toxics in ash enter drinking water supplies. The potential for long-term exposures resulting from structural fires varies greatly by site, and the hazards are not well quantified.

Although several hypotheses have been established regarding the mechanisms by which $PM_{2.5}$ exposure leads to adverse health outcomes, smoke exposure may present unique concerns due the level of exposures and co-pollutants. Current and future research efforts related to spatially and temporally resolved exposure maps, indoor levels of exposure, and a better understanding of internal dose in occupational settings will continue to add relevant information to establish health-protective recommendations and practices and to identify populations at risk. The largest gap in scientific evidence is related to long-term effects, such as birth outcomes, progression of chronic disease, incidence of chronic disease related to wildland fire smoke exposure, and the effects of chronic and repeated exposures in population and occupation settings.

Smoke-ready communities

Annual health costs of wildland fire episodes from 2008 to 2012 were estimated at 11 USD billion to 130 USD billion (Fann et al. 2018), far exceeding fire suppression costs. Intervention strategies can reduce exposure to smoke, and local communities can play an important role in informing residents. The EPA, in partnership with other agencies, has led the development of community guidance on smoke with a publication "Wildfire Smoke: A Guide for Public Health Officials" (U.S. EPA, 2019b). This article provides state, tribal, and local public health officials with information needed to prepare for smoke events and, when wildfire smoke is present, to communicate health risks and take measures to protect the public. It provides specific procedures (e.g., operation of air cleaners, proper use of masks or respirators) and recommendations (e.g., avoiding strenuous activity). These proactive measures can substantially reduce hospital admissions, mortality, and community impacts from wildfire PM (Fisk and Chan 2017).

Regulatory context for air quality management

Smoke causes many days above the daily NAAQS thresholds for $PM_{2.5}$ (>35 $\mu g/m^3$) and O_3 (MDA8 > 70 ppb). In an analysis of how smoke affects regulatory standards for $PM_{2.5}$, McClure and Jaffe (2018) showed that although most regions of the country have declining $PM_{2.5}$, the annual 98^{th} percentile of daily averages is increasing in many parts of the western U.S., where wildland fires are increasing. However, using the exceptional events rule (U.S. EPA, 2016) smoke-influenced air quality data can be excluded from regulatory consideration (e.g., designation of areas as not attaining the NAAQS). This process can be complex and resource-intensive, requiring states to submit extensive supporting documentation. In the case of $PM_{2.5}$, wildland fires frequently cause large

exceedances of the $PM_{2.5}$ daily standard, making the documentation less complex. But for O_3, smoke events can increase the MDA8 values by modest amounts (e.g., 5–30 ppb; Gao and Jaffe 2020; Gong et al. 2017), and the chemistry is not well understood; thus, documenting the influence of fire on O_3 is more challenging (e.g., see discussions in Gong et al. 2017,; Jaffe et al. 2018).

The U.S. EPA in the 1999 Regional Haze Rule (RHR; 40CFR 51.308) calls for state and federal agencies to work together to improve visibility in 156 Class I areas, which include national parks and wilderness areas. The goal is to eliminate human-made visibility impairment by 2064 in these areas. Wildland fire can contribute to visibility impairment. Under the RHR, wildfires are considered natural events. Regarding prescribed fires, the EPA recognizes the need for healthy and resilient forests, rangelands, and other federal lands, which can include the use of prescribed fires. Thus, the EPA requires states to consider basic smoke management practices applicable to prescribed fires as they consult with federal land managers about how best to improve visibility in Class I areas (U.S. EPA, 2019c).

Smoke management programs are regulatory tools for protecting public health and safety and natural resources in both long-term (e.g., with the Regional Haze Rule) and short-term (e.g., daily NAAQS) horizons (Long, Tarnay, and North 2017). These are typically used to manage prescribed and/or agricultural burns, but smoke management programs vary widely by state.

Given this regulatory context, it is important to identify specific chemical tracers that can help identify the contribution of smoke to local air quality (e.g., $PM_{2.5}$ and O_3). Past studies have used aerosol potassium (K), levoglucosan ($C_6H_{10}O_5$), gas phase hydrogen cyanide (HCN), and/or acetonitrile (CH_3CN, ACN). Levoglucosan is known to be emitted by wildfires but is readily oxidized (Hennigan et al. 2010) and emitted in widely varying amounts (Bhattarai et al. 2019). Potassium is emitted by wildfires, but it is also emitted by many other sources (Pachon et al. 2013). Acetonitrile has been used in many previous studies as a tracer of biomass burning and is relatively stable during transport. ACN has a low background mixing ratio (0.1–0.3 ppbv) and an atmospheric lifetime on the order of months, and other emissions sources are much less significant (de Gouw et al. 2003; Singh et al. 2012), making it the most suitable tracer. While past studies have measured ACN in the field using proton-transfer mass spectrometry (e.g., Warneke et al. 2011), a recent study has used the much simpler approach of thermal desorption gas chromatography-mass spectrometry (GC-MS) to identify ACN and OVOCs in urban areas influenced by biomass burning (Chandra et al. 2020). In this approach, continuous samples from a field site can be collected relatively easily, with GC-MS analysis occurring back in the laboratory. Both ACN and some of the OVOCs are highly specific indicators for biomass burning sources that could be used to support exceptional event designations.

National fire patterns and trends

Forests on public and private lands provide benefits to the natural environment, as well as economic benefits and ecosystem services (e.g., water, recreational opportunities, and carbon storage). The ability of U.S. forests to continue to provide clean air is potentially threatened by climate change and associated increases in extreme weather events and wildfire. Spatial and temporal patterns of wildland fire vary across the U.S. (Table 4), so inferences about fire emissions, the effects of climate change, and other issues are appropriate only at the regional to sub-regional scale.

Wildland fire is a component of a broader stress complex of extreme weather events, insect outbreaks, pathogens, and invasive species (McKenzie et al. 2014), which can pose long-term risks to forests (Trumbore, Brando, and Hartmann 2015; Vose et al. 2018). An example of interactions occurred recently in the Sierra Nevada of California, where 102 million trees died during a five-year drought ending in 2017 (U.S. Forest Service 2016), with much of the mortality attributed to beetle outbreaks in drought-weakened trees. This rapid change in stand structure and composition has increased the likelihood of large, intense fires in the short term and altered hydrology in the long term (Adams et al. 2012; Hicke, Meddens, and Kolden 2016; Pfeifer, Hicke, and Meddens 2011).

Several decades of fire suppression in fire-prone forest ecosystems in the U.S. (especially in the West) have created landscapes of dense forests with high flammability and heavy surface and canopy fuel loads, especially at lower elevations (Keane et al. 2009). Over the past two decades, a warm, dry climate has increased the area burned across the U.S. (Abatzoglou and Kolden 2013). Wildland fire burned at least 1.5 million ha nationwide in 17 of the years from 2001 to 2019, including over 4 million ha each year in 2015 and 2017 (Figure 3) (National Interagency Fire Center (NIFC) 2019). Large, intense wildfires in some locations (Barbero et al. 2015) have been difficult to suppress, increasing risk to property and lives as well as increasing smoke production (Liu et al. 2015b; Stavros et al. 2014). The cost of fire suppression has also increased over time – ranging from 240 USD million

Table 4. Summary of wildland fire for different regions in the U.S.

Region*	Typical fire season	Wildfire characteristics	Role of wildland-urban interface (WUI)	Management actions
Alaska	May–Jun	Mostly lightning-caused; high interannual variability in fire depending on dry weather; largest fires >100,000 ha.	WUI is usually not important.	Although most wildfires are suppressed, it is difficult to limit fire spread in remote landscapes; prescribed burning is rarely used.
Western contiguous states, minus California and Southwest (Arizona and New Mexico)	Jun–Sep	Mostly lightning-caused in mountains; high fuel loadings in many dry forests can facilitate intense fires; largest fires may be 1,000 km^2.	WUI expanding in many areas, resulting in human ignitions and challenges for fire suppression.	Most wildfires are suppressed when small; emphasis on WUI protection; prescribed burning is used in dry conifer forests.
California	Oct–Nov** Jun–Sep	Many lightning-caused in Sierra Nevada, mostly human-caused elsewhere; high fuel loadings in many dry forests can facilitate intense fires; largest fires >100,000 ha.	WUI is pervasive in most areas, resulting in human ignitions and challenges for fire suppression.	Most wildfires are suppressed when small except for those caused by Diablo and Santa Ana winds; emphasis on WUI protection; prescribed burning is used in dry conifer forests in the Sierra Nevada.
Southwest (Arizona and New Mexico)	May–Jun	Combination of lightning- and human-caused; fires often driven by interannual variation in fuel production (e.g., grasses); largest fires >100,000 ha.	WUI is important mostly for smaller communities near mountains.	Most wildfires are suppressed when small; prescribed burning is used in dry conifer forests.
Great Plains	Apr–Jul	Mostly human-caused, some lightning-caused; largest fires are rarely >10,000 ha.	WUI is sometimes important.	All wildfires are suppressed; prescribed fire and livestock grazing are used in some areas to reduce grass fuels.
Midwest and Northeast	Apr–Jun	Mostly human-caused; dependent on dry spring weather; fires are small.	WUI is very important due to high population density.	All wildfires are suppressed; prescribed fire is sometimes used on small areas of hardwood and pine forests.
Southeast	Feb.–Sep	Mostly human-caused, some lightning-caused; largest fires are rarely >10,000 ha.	WUI is increasingly important as population expands.	All wildfires are suppressed; prescribed fire is extensively and routinely used in pine forests.

*Hawai'i and U.S.-affiliated areas are not included here because they comprise a very small portion of fire and smoke occurrence.
**Fire occurrence varies from north to south. Diablo winds (northern California) and Santa Ana winds (southern California) typically occur in the fall, but other fires occur in summer.

in 1985 to 3.1 USD billion in 2018 (National Interagency Fire Center (NIFC) 2019) – partially driven by the high cost of protecting property at the wildland-urban interface (WUI) (Figure 3).

The duration of the wildfire season has increased by 80 days in some parts of the western U.S. as a result of increased temperature (McKenzie and Littell 2017; Westerling 2016), earlier snowmelt (Gergel et al. 2017; Luce, Lopez-Burgos, and Holden 2014), and altered precipitation patterns (Holden et al. 2018). By the mid-21st century, the annual area burned in the U.S. could increase 2–3 times from the present, depending on the geographic area, ecosystem, and local climate (Halofsky, Peterson, and Harvey 2020; Litschert, Brown, and Theobald 2012; Ojima et al. 2014). As the spatial extent of wildfires increases, burned areas may provide fuel breaks that influence the pattern, extent, and severity of future fires (Parks et al. 2015).

In the southeastern U.S., landscapes are dominated by private lands and relatively high human populations, so changes in social behavior (e.g., human-caused ignitions), policy (e.g., fire suppression), and climate can affect the frequency and extent of wildland fire (Balch et al. 2017). Data from Florida indicate that in drought years, less prescribed burning is conducted (Nowell et al. 2018). Modeling studies suggest that the southeastern U.S. will experience increased fire risk and a longer fire season in the future (Liu, Goodrick, and Stanturf 2013).

Although projections vary by state and ecoregion, by 2060, the annual area burned by lightning-ignited wildfire is expected to increase by at least 30% in the Southeast (Prestemon et al. 2016). More frequent and larger wildfires, combined with increasing development at the WUI, portend increasing risks to property and human welfare. For example, a prolonged dry period in the southern Appalachian region in 2016 resulted in widespread wildfires that caused 15 deaths and damaged or destroyed nearly 2,500 structures in Gatlinburg, TN. In a warmer climate, increased fire frequency will further degrade pollution levels and damage local economies in the Southeast.

Topography, fuel accumulation, stress complexes, a patchwork of previous fires, and past efforts to suppress and prevent fires provide a biogeographic and social context for future wildland fire regimes (Abt et al. 2015; Butry et al. 2010). Currently, 95–98% of all U.S. fires are controlled in the initial attack phase (i.e., before they expand beyond 40 ha of forest or 120 acres of grassland or shrubland), but the remaining 2–5% of fires that cannot be controlled early are

increasingly demonstrating extreme fire behavior (U.S. Department of Agriculture and Department of the Interior 2015). Higher temperatures, lower summer precipitation, and increased frequency and intensity of drought are expected to create longer periods during which surface fuels are sufficiently dry to burn. This will drive rapid (months to years) and persistent changes in forest structure and function across large landscapes. Other changes, resulting from gradual climate change and less severe disturbances, will alter forest productivity and vigor and the distribution and abundance of species at longer time scales (decades to centuries) (Vose et al. 2018).

Public land managers are acutely aware that increasing human population and climate change will alter fire regimes and ecosystem conditions. Expansion of the WUI has already altered fire suppression tactics and costs, as well as when and where fuel treatments are applied. Fuel treatments, including forest thinning, mechanical removal of surface fuels, and prescribed burning, have been used for decades to reduce hazardous fuels in dry forest landscapes (Peterson et al. 2005), including in the WUI (Johnson, Kennedy, and Harrison 2019). However, concerns about the health effects of smoke on residents in the WUI and exurban locations often limit the extent of fuel treatments. Miller, Field, and Mach (2020) describe some of the barriers to prescribed burning in California, which include liability concerns, resource limitations and regulations. The widespread use of prescribed burning in southern forests is highly effective in reducing fuels across large landscapes, but effectiveness in western landscapes is limited due to inadequate budgets for treating vast landscapes with elevated fuel loading.

The effects of periodic prescribed burning on long-term emissions and air quality are poorly quantified. A synthesis of studies in the western U.S. determined that carbon emitted per ha from prescribed burning over many decades is similar to or slightly higher than what would have been emitted by wildland fires over the same time period (Restaino and Peterson 2013). If we assume that total emissions are proportional to carbon flux into the atmosphere from fire, we can cautiously infer that total emissions per ha for prescribed burning are similar to those of wildland fire. However, the periodic pulses of emissions produced by prescribed burning have lower concentrations of particulates and other pollutants for a shorter duration than in a large wildland fire. Prescribed burning can also be timed to minimize population exposure to $PM_{2.5}$ using forecast models.

Over the past decade, assessments of climate change effects on fire have been developed for many locations in the western and southern U.S. (e.g., Halofsky et al. 2018a; Prestemon et al. 2016). These assessments and adaptation responses to the effects of climate change are now being incorporated into resource management plans, environmental assessments, and monitoring programs of public agencies (Halofsky et al. 2016; Halofsky, Peterson, and Prendeville 2018b; Timberlake and Schultz 2019). Many ongoing practices that address existing forest management needs – stand density management, surface fuel reduction, and control of invasive species – are considered "climate smart" because they reduce risk by creating resilience to increased temperature, drought, and disturbances (Peterson, Halofsky, and Johnson 2011a, Peterson et al. 2011b; D'Amato et al. 2013). Resource managers are evaluating how these practices can be modified and implemented to address future climate risks (Halofsky et al. 2016). For example, forest managers are considering reductions in stand density to increase forest resilience to fire, insects, and drought (Sohn, Saha, and Bauhus 2016). Allowing more wildland fires to burn without suppression (but with observation) in remote mountainous locations would reduce fuels, but they may enhance emissions in the short term compared to aggressive suppression activities.

Summary and recommendations

Wildland fires are a natural occurrence, but the area burned has increased dramatically in the last few decades due to past forest management practices, climate change, and other human factors. As a result, millions of people in the U.S. have been exposed to extremely high concentrations of air pollution in the recent decade. As our population has expanded into the wildland-urban interface (WUI), the costs for fire suppression and consequences of wildland fires have risen dramatically. Based on our review, we conclude with the following recommendations:

(1) Multiple factors have led to the significant increase in the area burned by wildland fires in recent decades. Research is needed to better understand the effects of various biophysical characteristics on past and future trends in wildland fire, including human land use and ignitions, insect outbreaks, invasive species, and climate change (including increasing temperatures, drought, and other factors). The respective roles of these factors will vary regionally, so data will be needed at a variety of spatial scales. Long-term monitoring and frequent reevaluation will be needed to refine

quantitative relationships as the climate continues to warm.

(2) As the risk of wildfires increases, the use of prescribed burning to protect human and ecosystem health also increases. Developing strategies to minimize adverse impacts on air quality requires improved understanding of emissions from wildfires and prescribed fires. More research is needed to link emissions to fuels, fire behavior, and other factors. In particular, research is needed on differences between wildfires and prescribed fire emissions, including on various burn strategies that could be used to minimize impacts on air quality.

(3) Satellite data provide critical information about fire detections, smoke transport, and impacts. However, ease of access to the data and an understanding of how best to use the satellite information needs improvement, particularly data from the rapidly evolving suite of newer and more sensitive satellite systems. Additional research is needed to examine best approaches for using fire intensity (e.g., fire radiative power) to calculate emissions, and to link fire radiant energy to the fire type and quantity of vegetation on the landscape. Improved tools to derive the vertical distribution of smoke from satellite observations would substantially improve our understanding of impacts at the surface.

(4) Smoke forecast and modeling systems are important tools to understand impacts from wildland fires and provide advance warnings to affected communities. Smoke prediction systems rely on various meteorological forecasts; however, although meteorological forecasts are typically analyzed as an ensemble to produce probabilistic forecasts, this has not occurred to date with smoke forecasts. Future smoke forecasting research should focus on generating ensemble/probability smoke forecasts, thus accounting for multiple potential outcomes due to uncertainty in model inputs and algorithms as well as the natural variability and heterogeneity of the fuels and ecosystems.

(5) Once released, the gas and particle emissions undergo substantial chemical processing in the atmosphere. In some cases, this processing may lead to compounds with greater health implications (e.g., more oxidized aerosols). But the large number of compounds, many of which are not found in typical urban air, makes it difficult to understand the chemistry. Research is needed to improve understanding of the chemical processes that form secondary pollutants (e.g., secondary organic aerosol, O_3, and their precursors), especially as smoke plumes mix into population centers. Embedded "plume in-grid cell models" may be needed to address non-linear chemical processes such as O_3 or SOA production. A related need is for easily measured smoke tracers that can provide a quantitative measure of smoke in urban areas.

(6) $PM_{2.5}$, O_3, and other compounds in smoke have clear and demonstrated human health impacts. But the episodic nature of smoke exposure and the variable mix of compounds make health studies even more challenging than traditional air pollution studies. Future research is needed to provide better data on exposure, including indoor and occupational exposure, to improve our understanding of the resulting health effects, and to establish exposure guidelines. The largest gap in scientific evidence is related to long-term consequences, such as birth outcomes, neurological and cognitive effects, and progression and incidence of chronic disease related to wildland fire smoke exposure.

(7) Field campaigns need to be integrated across the wide spectrum of disciplines involved in fuel combustion, fire behavior/growth, fire emissions, plume dynamics, and atmospheric chemistry. Experiments should relate ground-based information from fuels and how the fire spreads, to what the satellites see from space, and everything in between. Recent campaigns, such as WE-CAN, FIREX-AQ, and FASMEE, provide a starting point for such work, but additional studies that both build upon and learn from these successes are needed to sample across the wide range of fire types and conditions that lead to smoke impacts.

(8) Fire-prone communities need to identify approaches to protect lives and property, build resilience, and develop response plans to minimize health and socio-economic impacts. On the health side, these could include, for example, communication in advance with the most at-risk citizens, creation of community clean air spaces in public buildings, workshops on creating clean air spaces at home and in workplaces, and distribution of filtration equipment to those most in need, such as those with limited mobility or particular sensitivities. All of these methodologies are now being tested and/or implemented by communities in the western U.S. This work needs to be continued,

expanded, and funded, and communities would benefit from working together to develop a framework for sharing the best strategies.

Acknowledgments

The authors acknowledge editing, analysis and/or graphics assistance from Dee Ann Lommers-Johnson (UW Bothell), Amy Marsha (USFS), Patti Loesche (USFS), Jonathan Callahan (Mazama Science), and Aranya Ahmed (Oak Ridge Associated Universities). Partial support was provided by the NASA Health and Air Quality Applied Sciences Team (HAQAST) (grant #NNH16AD18I) for remotely-sensed data processing under the direction of S. O'Neill.

The views expressed in this publication are those of the authors and do not represent the policies or opinions of any U.S. government agency.

Supplmental materials

The full citation for all references cited in this document are in the online supplemental materials section. These can be accessed at https://doi.org/10.1080/10962247.2020.1749731

Disclosure statement

No potential conflict of interest was reported by the authors.

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Estimated Global Mortality Attributable to Smoke from Landscape Fires

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Background: Forest, grass, and peat fires release approximately 2 petagrams of carbon into the atmosphere each year, influencing weather, climate, and air quality.

Objective: We estimated the annual global mortality attributable to landscape fire smoke (LFS).

Methods: Daily and annual exposure to particulate matter ≤ 2.5 μm in aerodynamic diameter ($PM_{2.5}$) from fire emissions was estimated globally for 1997 through 2006 by combining outputs from a chemical transport model with satellite-based observations of aerosol optical depth. In World Health Organization (WHO) subregions classified as sporadically affected, the daily burden of mortality was estimated using previously published concentration–response coefficients for the association between short-term elevations in $PM_{2.5}$ from LFS (contrasted with 0 μg/m^3 from LFS) and all-cause mortality. In subregions classified as chronically affected, the annual burden of mortality was estimated using the American Cancer Society study coefficient for the association between long-term $PM_{2.5}$ exposure and all-cause mortality. The annual average $PM_{2.5}$ estimates were contrasted with theoretical minimum (counterfactual) concentrations in each chronically affected subregion. Sensitivity of mortality estimates to different exposure assessments, counterfactual estimates, and concentration–response functions was evaluated. Strong La Niña and El Niño years were compared to assess the influence of interannual climatic variability.

Results: Our principal estimate for the average mortality attributable to LFS exposure was 339,000 deaths annually. In sensitivity analyses the interquartile range of all tested estimates was 260,000–600,000. The regions most affected were sub-Saharan Africa (157,000) and Southeast Asia (110,000). Estimated annual mortality during La Niña was 262,000, compared with 532,000 during El Niño.

Conclusions: Fire emissions are an important contributor to global mortality. Adverse health outcomes associated with LFS could be substantially reduced by curtailing burning of tropical rainforests, which rarely burn naturally. The large estimated influence of El Niño suggests a relationship between climate and the burden of mortality attributable to LFS.

Key words: air pollution, biomass burning, carbon cycle, deforestation, global burden of disease, landscape fire smoke, mortality. *Environ Health Perspect* 120:695–701 (2012). http://dx.doi.org/10.1289/ehp.1104422 [Online 18 February 2012]

Landscape fires (encompassing wild and prescribed forest fires, tropical deforestation fires, peat fires, agricultural burning, and grass fires) release approximately 2 petagrams (2×10^{12} kg) of carbon into the atmosphere annually (van der Werf et al. 2010). These emissions affect planetary processes such as radiative forcing (which influences average global temperatures) and hydrological cycles (which influence regional cloud formation and rainfall) (Bowman et al. 2009; Cochrane and Laurance 2008; Fargione et al. 2008; Langmann et al. 2009; Tosca et al. 2010; Yokelson et al. 2007). Most emissions originate from fires set in tropical rainforests and savannas, where they cause recurrent episodes of severe pollution that affect some of the poorest regions of the world (van der Werf et al. 2010). Despite extensive literature describing the harmful effects of air pollution, the health impacts of landscape fire smoke (LFS) are rarely highlighted in discussions about fires and their role in the earth system (Lohman et al. 2007).

Smoke from the combustion of biomass is composed of hundreds of chemicals, many of which are known to be harmful to human health (Naeher et al. 2007). The most important risk-related measure of smoke is particulate matter (PM) with an aerodynamic diameter ≤ 2.5 μm ($PM_{2.5}$). This PM primarily consists of organic carbon and black carbon components, along with smaller contributions from inorganic species (Naeher et al. 2007; Reid et al. 2005). PM is also produced by the combustion of fossil fuels, and most health evidence for $PM_{2.5}$ comes from studies in urban environments (Pope and Dockery 2006). Urban PM has been associated with a wide range of adverse health outcomes including all-cause, neonatal and cardiorespiratory mortality, exacerbations of respiratory and cardiovascular conditions, and pathophysiological changes such as inflammation, oxidative stress, and procoagulation (Pope and Dockery 2006). The effects of PM derived from burning biomass have been less extensively investigated, and much of the evidence comes from studies of air pollution from household solid fuel use (Naeher et al. 2007). A handful of toxicological studies suggest that biomass smoke particles elicit pathophysiological effects similar to those of urban PM (Barregard et al. 2006; Danielsen et al. 2009; Kocbach et al. 2008). Although there are relatively few epidemiological studies on smoke-related PM, they also report outcomes consistent with those elicited by urban PM, including increased all-cause mortality and exacerbations of respiratory conditions (Delfino et al. 2009; Hänninen et al. 2009; Johnston et al. 2007, 2011; Morgan et al. 2010; Sastry 2002). However, evidence concerning cardiovascular outcomes of smoke-related PM remains scarce and inconclusive (Naeher et al. 2007; Sanhueza et al. 2009). Results from several studies of the extensive rainforest and peat fires in Southeast Asia in 1997 through 1998 suggest substantial health and economic impacts of LFS (Jayachandran 2009; Mott et al. 2005; Sastry 2002; Schweithelm et al. 2006). Further, fires are becoming more widespread and frequent in some regions (Turetsky et al. 2011; Westerling et al. 2006), and this source of air pollution is likely to continue to grow in magnitude and consequent health impacts (Confalonieri et al. 2007; Denman et al. 2007; Langmann et al. 2009). Because fire emissions contribute to radiative forcing, there is potential for the development of a positive feedback between a warming climate and increasingly severe fire events in several biomes (Bowman et al. 2009). In this context, a global assessment of the mortality impacts of LFS is required.

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Supplemental Material is available online (http://dx.doi.org/10.1289/ehp.1104422).

This project was supported in part by grants from the Australian Research Council (LP LP0882048), the University of Tasmania (J0018237), and the U.S. National Aeronautics and Space Administration (NNX08AF64G). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

The authors declare they have no actual or potential competing financial interests.

Received 30 August 2011; accepted 13 February 2012.

Materials and Methods

Studying the magnitude of health impacts from LFS presents several technical challenges, including estimation of the exposure to smoke-specific PM for each spatial unit of analysis, selection of the most appropriate concentration–response functions, and consideration of what theoretic minimum (counterfactual) exposure values to apply. Moderate to high levels of uncertainty are associated with many of these steps, so our objectives were to provide a reasonable principal estimate given the available data and then to evaluate the sensitivity of the principal estimate to the assumptions used in the principal analysis. The World Health Organization (WHO) Global Burden of Disease (GBD) Comparative Risk Assessment framework provides a standard set of methods for this and has previously been used to evaluate the annual mortality attributable to urban air pollution and to indoor air pollution from household solid fuel use (Ezzati et al. 2002; Lopez et al. 2006a). Methods for estimating the global mortality associated with particulate air pollution are being revised in the light of new epidemiological evidence and exposure assessment methods, and new cause-specific results are expected in 2012 (Institute for Health Metrics and Evaluation 2010). However, the epidemiological evidence concerning LFS remains limited, and evidence concerning LFS and cause-specific mortality is not currently available. For this reason, our analyses evaluate all-cause mortality.

Input data. **Exposure estimates.** We combined information from satellite-derived observations of global fire activity, geographic area burned, and type of vegetation burned in a global atmospheric three-dimensional (3-D) chemical transport model. We then combined output from that model with satellite-based measurements of aerosol optical depth (AOD) to estimate annual $PM_{2.5}$ emissions from landscape fires. For a detailed description of the exposure estimates, see Supplemental Material, pp. 3–8, Table 1, and Figures 1 and 2 (http://dx.doi.org/10.1289/ehp.1104422). A summary is presented below.

Monthly resolved emissions estimates were obtained from the Global Fire Emission Database (Global Fire Data 2012; van der Werf et al. 2006), which combines satellite observations of burned area (in square kilometers) with estimates of fuel loads obtained from a biogeochemical model (Giglio et al. 2006). These emissions estimates were used in the GEOS-Chem global 3-D chemical transport model (Bey et al. 2001), which simulates the transport, transformation, and deposition of organic carbon and black carbon aerosols. The model had a 2° (latitude) × 2.5° (longitude) horizontal resolution ~ 222 × 278 km at the equator and 30 vertical layers (Bey et al. 2001). We performed two sets of GEOS-Chem simulations spanning a 10-year period (1997 through 2006). The first included all aerosol emission sources (fossil fuel, biofuel, landscape fires, natural dust, and sea salt), whereas the second excluded landscape fire emissions to separate the contribution from this source.

Finally, we scaled the modeled $PM_{2.5}$ estimates using two sets of AOD observations from the Moderate Resolution Imaging Spectroradiometer (MODIS) and the Multiangle Imaging Spectroradiometer (MISR) aboard the U.S. National Aeronautics and Space Administration (NASA) *Terra* satellite (Martonchik et al. 2009; Remer et al. 2005). We maintained the same seasonal, regional, and vertical aerosol distributions as predicted by the GEOS-Chem simulations. Our best estimate of surface $PM_{2.5}$ (1997 through 2006 average shown in Figure 1) combined information from the model estimates along with the two satellite AOD-scaled estimates:

$$\text{LFS } PM_{2.5} = [(2 \times \text{MODEL}) + \text{MODIS} + \text{MISR}]/4, \quad [1]$$

where MODEL is the estimate of $PM_{2.5}$ from LFS derived from GEOS-Chem and MODIS and MISR are the two satellite AOD-scaled estimates. We multiplied the model contribution by 2 so that our best estimate gave equal weight to the *a priori* atmospheric model distribution and the sum of the two satellite-scaled estimates. The total aerosol emissions from fires used in the model simulations was 23.5 teragrams (Tg; 1 Tg = 10^9 kg) per year averaged over 1997 through 2006. Comparable estimates for the MISR and MODIS AOD-based optimizations were 55.0 and 45.5 Tg/year, respectively [see Supplemental Material, Table 1 (http://dx.doi.org/10.1289/ehp.1104422)] and were within the range of previously published estimates (see Supplemental Material, Table 2). Our best estimate, defined according to Equation 1, was 36.9 Tg/year.

Evaluation of exposure estimates. Surface measurements of $PM_{2.5}$ are not available for most regions with high fire emissions. To evaluate the quality of the global exposure estimates, we used ground-based AOD from National Aeronautics and Space Administration's (NASA) Aerosol Robotic Network (AERONET; NASA 2012) (Holben et al. 1998), $PM_{2.5}$ measurements from the U.S. Environmental Protection Agency IMPROVE (Interagency Monitoring of Protected Visual Environments) program (Chow and Watson 2002), and visibility data in tropical regions from the National

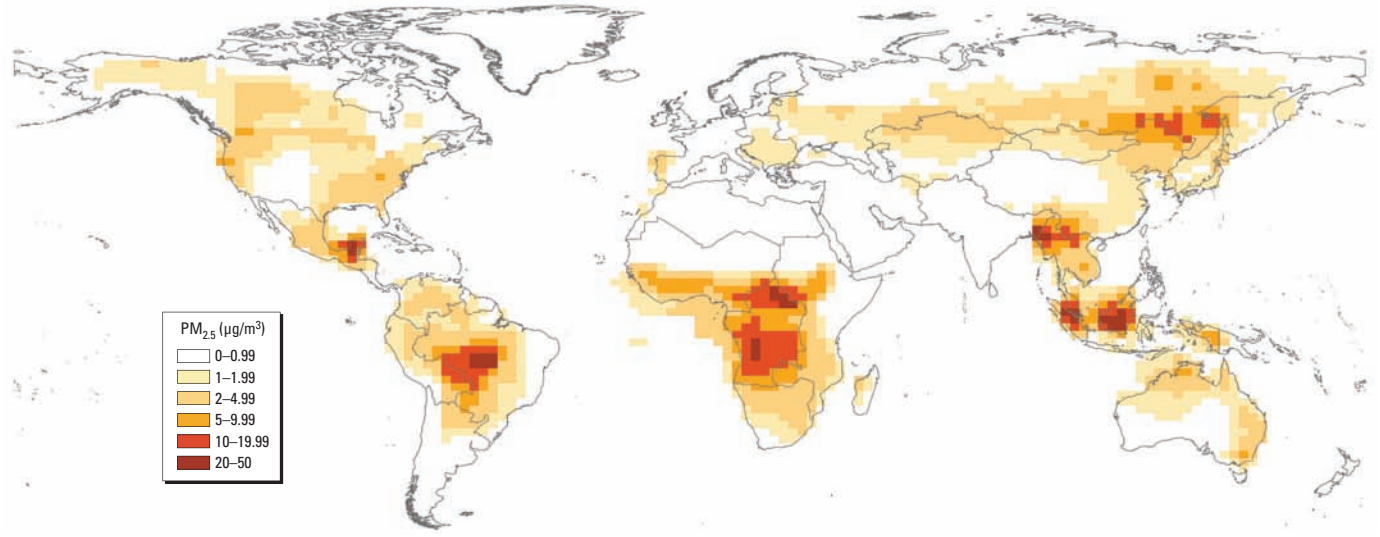

Figure 1. Estimated annual average (1997–2006) $PM_{2.5}$ concentrations from landscape fires, combining estimates from the GEOS-Chem model with the MODIS and MISR optimizations.

Climatic Data Center Global Summary of the Day (National Oceanic and Atmospheric Administration 2009). Our exposure estimates correlated well with these other measures in regions with high fire activity [see Supplemental Material, Figures 3–6 (http://dx.doi.org/10.1289/ehp.1104422)]. Correlations (Pearson's *r*) of estimated AOD with monthly mean AODs from AERONET were 0.81 in southern Africa (n = 119), 0.90 in northern Africa (n = 74), and 0.76 in Southeast Asia (n = 148; see Supplemental Material, Figure 4). Median correlations between $PM_{2.5}$ and visibility were 0.57 for sub-Saharan Africa (n = 58), 0.60 for South America (n = 47), and 0.68 for Southeast Asia (n = 13; see Supplemental Material, Figure 6).

Gridded mortality estimates. Country-specific estimates for all-cause all-age mortality in the year 2002 were obtained from the WHO Global Health Observatory (2011). Estimates from the Gridded Population of the World (GPW; version 3) project were used to map country-specific mortality onto the 2° × 2.5° exposure cells (Sociodemographic Data and Applications Centre 2011). The spatial resolution of the GPW data is 2.5 arc-min (~ 4.6 × 4.6 km at the equator), meaning that each exposure cell encompassed 2,880 population cells. To distribute mortality between the population cells, we assigned each cell to the underlying country that contained most of it, summed the GPW population for each country and calculated the percentage of the total population in each cell, and then assigned that percentage of the national mortality to the cell. In the < 1% of cases where population cells were assigned to countries that do not belong to the WHO, we followed the same steps for the 21 WHO subregions and assigned those values instead. The mortality in each exposure cell was estimated by summing the mortality in the 2,880 underlying population cells.

Global burden calculations. **Pattern of exposure: subregions of sporadic and chronic impact.** Fire activity varied widely across the globe during the 1997 through 2006 period. Some areas were affected sporadically, with a limited number of smoky days in any given year; some areas were affected chronically, with whole seasons being smoke-affected in multiple years. Our principal analysis treats these areas as fundamentally different because acute and chronic PM exposures have independent health effects (Pope and Dockery 2006; Schwartz 2000). We began by classifying each of the 21 WHO subregions as sporadically affected or chronically affected.

The complete set of smoke-specific $PM_{2.5}$ estimates (12 months × 10 years × 4,208 exposure cells = 504,960) was log-normally distributed with a 90th percentile value of 3 μg/m^3. When concentration estimates were rounded to integers, most exposure cells had a value of zero in most months (331,035 of 504,960), indicating low smoke-specific $PM_{2.5}$. An exposure cell with a 1-month smoke-specific $PM_{2.5}$ estimate > 3 μg/m^3 was classified as being smoke affected during that month. Exposure cells with ≥ 3 smoke-affected months in ≥ 5 of the years were classified as chronically affected (732 of 4,208). Exposure cells that were not chronically affected were classified as sporadically affected (3,476 of 4,208). A WHO subregion was classified as chronically affected if > 50% of its population and/or > 50% of its land area was covered by chronically affected exposure cells (7 of 21; Figure 2). All other WHO regions were classified as sporadically affected (14 of 21; Figure 2).

Burden for sporadically affected subregions. For sporadically affected subregions, we estimated effects of short-term (daily) fluctuations in smoke-specific $PM_{2.5}$ concentrations on mortality. Daily output from GEOS-Chem was used to estimate the number of days per year that $PM_{2.5}$ concentrations exceeded a set of threshold values (300, 200, 100, 50, 40, 30, 20, 10, 5, 4, 3, 2, and 1 μg/m^3). These threshold values were chosen to provide a range of possible concentrations for sensitivity analyses, and because they reflect clinically relevant increments (10 μg/m^3, 100 μg/m^3) reported in the literature.

The annual mortality attributable to LFS in each sporadically affected 2° × 2.5° exposure cell was calculated as

$$\text{Sporadically affected attributable mortality} = \sum_{\text{PM}}^{n}\left[D_{\text{PM}} \times \frac{M}{365} \times \left(\text{RR}_{\text{SI}}(\text{PM}) - 1\right)\right], \quad [2]$$

where PM is one smoke-specific $PM_{2.5}$ threshold concentration out of n possible threshold values (see above), D_{PM} is the number of days between PM and the next highest concentration, M is the annual number of deaths in the exposure cell, and RR_{SI} is a relative rate estimate for all-cause mortality due to short-term PM exposure. Although annual mortality is not evenly distributed among the 365 days of the year, there are insufficient data to estimate seasonal mortality on a global scale.

For the principal analysis, a linear RR_{SI} estimate of 0.11% [95% confidence interval (CI):

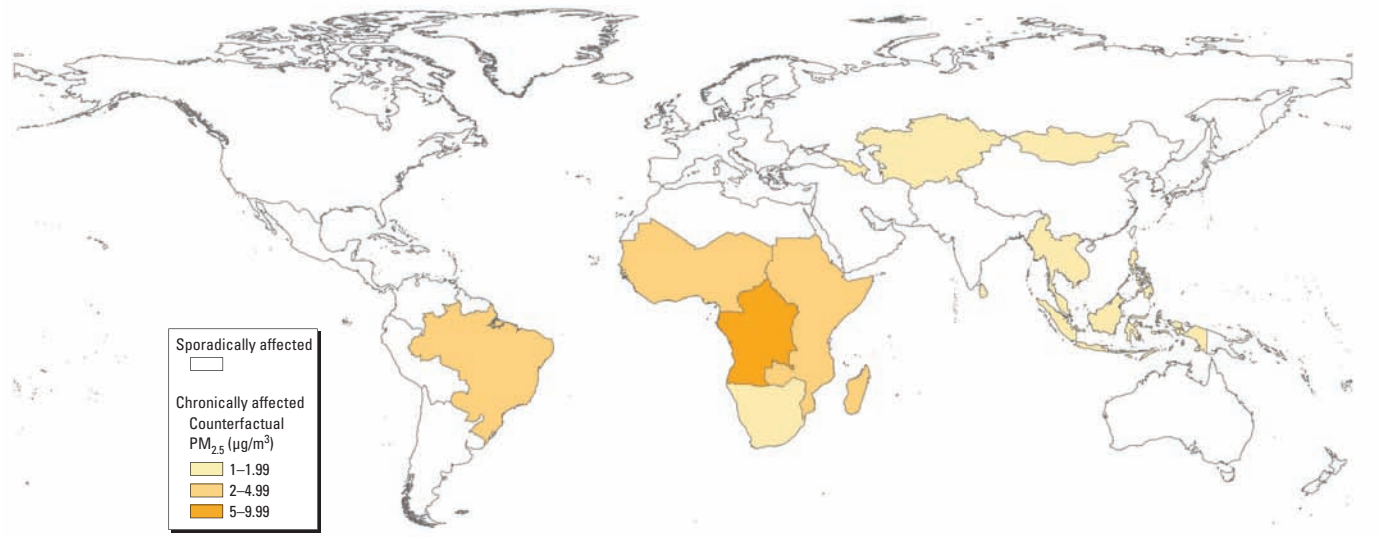

Figure 2. WHO subregions classified as sporadically and chronically affected. Subregions were classified as chronically affected if ≥ 50% of their populations and/or ≥ 50% of their land areas were covered by smoke-affected exposure cells for at least 3 months per year for ≥ 5 years. The theoretical minimum annual average (counterfactual) concentration used for chronically affected subregions was calculated by taking the mean of the minimum 12-month running average (over 120 months) of all exposure cells in the subregion. The remaining subregions were classified as sporadically affected. The theoretical minimum daily average (counterfactual) concentration used for sporadically affected subregions was zero.

0, 0.26%] per increase of 1 μg/m^3 was used with minimum and maximum concentrations of 5 and 200 μg/m^3. This means that cells with daily exposure estimates of < 5 μg/m^3 were not included, and cells with exposure estimates > 200 μg/m^3 were fixed at a value of 200 μg/m^3. The RR_{SI} was calculated using the average (weighted by the inverse of the standard errors) of values from studies reporting associations between all-cause mortality and short-term elevations of ambient PM_{10} during fire events (Morgan et al. 2010; Sastry 2002) and $PM_{2.5}$ (Hänninen et al. 2009). Associations with ambient PM_{10} were converted to associations with $PM_{2.5}$ by assuming that 75% of all particles < 10 μm were also < 2.5 μm. This is halfway between the 90% ratio measured during fire events (Ward and Hardy 1991) and the 60% ratio used by Cohen et al. (2004) in the initial GBD estimate for urban air pollution.

Burden for chronically affected subregions. No studies have yet reported on the mortality impacts of long-term exposure to LFS. As such, we estimated all-cause mortality in chronically affected exposure cells by assuming the effects of smoke-related PM to be the same as those of urban PM. Specifically, for the principal analysis we assumed a linear 0.64% (95% CI: 0.35%, 0.94%) increase in annual all-cause mortality for each 1-μg/m^3 increase in the long-term smoke-specific $PM_{2.5}$ average, as reported in the American Cancer Society study on urban air pollution (Pope et al. 1995). This is one of the most conservative concentration–response estimates that has been reported in multiple studies of urban PM (Pope and Dockery 2006). The maximum concentration of effect was assumed to be 50 μg/m^3. This means that cells with annual exposure estimates > 50 μg/m^3 were fixed at a value of 50 μg/m^3. The annual mortality attributable to LFS in each chronically affected exposure cell was calculated as

$$\text{Chronically affected attributable mortality} = M \times ([RR_{CI}(PM - CF)] - 1), \quad [3]$$

where PM is the estimated average annual smoke-specific $PM_{2.5}$ concentration in the exposure cell based on estimates for 1997 through 2006, CF is the counterfactual concentration for the WHO subregion in which the exposure cell was located, M is the annual number of deaths in the exposure cell, and RR_{CI} is the relative rate of all-cause mortality for long-term PM exposure (i.e., 0.64% for the principal analysis).

The counterfactual concentration is the theoretical minimum annual smoke-specific $PM_{2.5}$ concentration under ideal conditions. For example, if landscape fires were completely eliminated worldwide, the global counterfactual value would be zero. Given that fire is a natural part of the earth system, we used a more data-driven approach to set counterfactual values for chronically affected WHO subregions. We used a subregion-wide approach because emissions from similar landscapes in neighboring countries can vary widely because of different land management practices, so the theoretical minimum exposure estimated for a single exposure cell might not truly reflect the minimum exposure possible for that particular landscape (Bowman et al. 2011). Specifically, we determined the smallest 12-month running average smoke-specific $PM_{2.5}$ concentration for each exposure cell within a WHO subregion, and averaged the minimum annual concentrations across all exposure cells to determine the counterfactual value for that WHO subregion.

Sensitivity analyses. There are several sources of uncertainty in our inputs, and we addressed these through multiple sensitivity analyses. First, we assumed both linear and log-linear forms for the concentration–response functions (i.e., RR_{SI} in Equation 2 and RR_{CI} in Equation 3). Although there is increasing evidence of a log-linear association for cardiovascular mortality related to urban air pollution (Pope et al. 2011), we used the linear assumption for the principal analysis because studies on the cardiovascular effects of LFS have been inconclusive. We also tested a range of different exposure limits. For the sporadic assumption, the minimum concentration was varied between 1 and 10 μg/m^3 and the maximum was varied between 50 and 300 μg/m^3. For the chronic assumption, five alternative counterfactual definitions [a global value of 0 μg/m^3; cell-by-cell average for a La Niña year, September 1999–August 2000 inclusive; regional average of the values from La Niña; minimum of the 12-month running averages of each cell; and global categorization of the values above at the 90th, 97th, and 99th percentiles, applying the average of the category to all cells in the category] were tested with maximum yearly average concentrations at 30 and 50 μg/m^3. We repeated analyses using the GEOS-Chem and satellite AOD-scaled exposure estimates separately. To assess the effect of our assumptions concerning the combination of sporadic and chronic exposures, all analyses were repeated with all subregions classified as being sporadically affected and with all subregions being classified as chronically affected. There is large interannual variation in emissions from landscape fires mostly driven by changes in climatic conditions (van der Werf et al. 2008). To assess the influence of interannual climatic variability, analyses were repeated with concentration estimates for a strong El Niño year that occurred between September 1997 and August 1998 (inclusive) and a strong La Niña year that occurred between

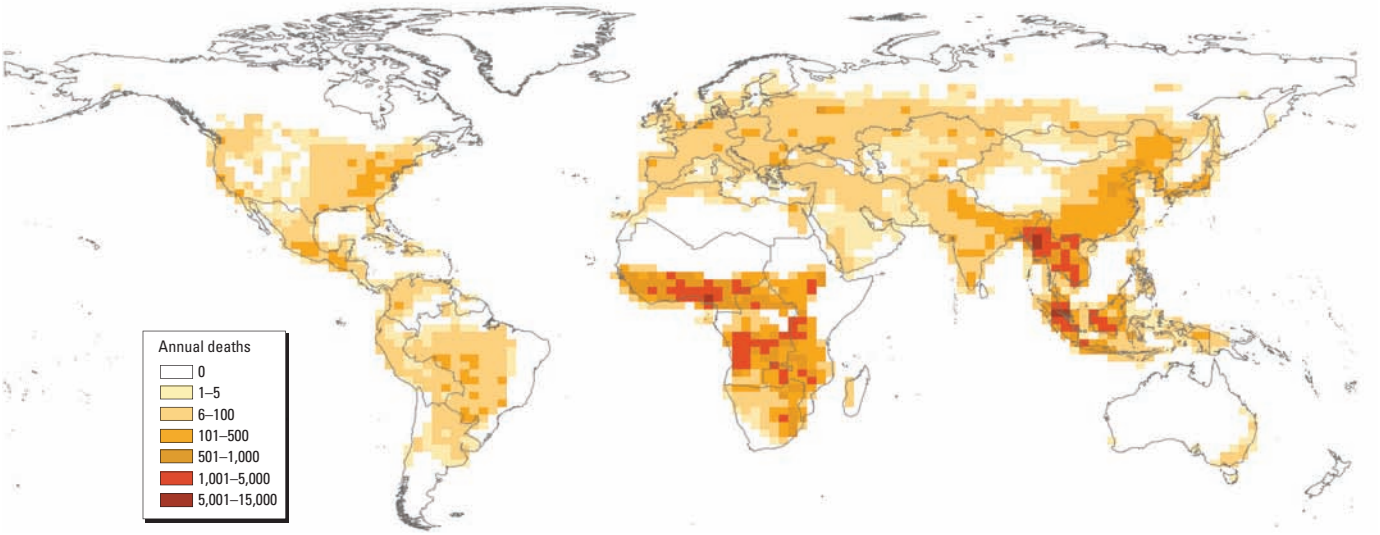

Figure 3. Map showing the principal estimates of the annual average (1997–2006) global mortality attributable to LFS.

September 1999 and August 2000 (inclusive) (van der Werf et al. 2004).

Results

Exposure. Estimated annual average concentrations ranged from 0 to 45 μg/m^3 annually (mean = 1.8 μg/m^3; Figure 1). The population-weighted annual average was 2.1 μg/m^3, ranging from 0.2 μg/m^3 in the Caribbean subregion to 12.2 μg/m^3 in sub-Saharan Africa. The population-weighted average number of annual days > 5 μg/m^3 was 28, ranging from 6 in the Caribbean subregion to 141 in sub-Saharan Africa.

Burden of mortality. Our principal estimate for the average annual mortality associated with exposure to LFS was 339,000 worldwide, including 157,000 in sub-Saharan Africa and 110,000 in Southeast Asia (Figure 3). The estimates for mortality due to LFS exposure compared with no LFS exposure at all (i.e., a zero exposure counterfactual) were 286,000 in sub-Saharan Africa and 119,000 in Southeast Asia, reflecting much higher background fire activity in sub-Saharan Africa than in Southeast Asia. During the El Niño year, the estimated mortality was higher, particularly in Southeast Asia, where El Niño is associated with dry conditions and more fires (Table 1).

Outputs from all tested models (n = 2,192) had a median of 379,000 and interquartile range of 260,000–600,000 [see Supplemental Material, Figure 7 (http://dx.doi.org/10.1289/ehp.1104422)]. Results of the sensitivity analyses are shown in Table 2. If a log-linear, rather than linear, concentration–response function was assumed, the mortality estimates more than doubled. The results were also sensitive to the exposure estimates, the assumed pattern of exposure (sporadic vs. chronic), and the choice of the counterfactual exposure estimation, all of which caused the estimated mortality to vary between 0.41 and 1.54 times the principal estimate (Table 2). Results were minimally influenced by the maximum and minimum exposures of effect, which caused the estimates to vary just 0.98 to 1.01 times the principal estimate (Table 2).

Discussion

Our estimate of 339,000 annual deaths attributable to exposure to LFS is lower than estimates for urban air pollution (800,000) and much lower than estimates for household solid fuel use (1,600,000) (Lopez et al. 2006b). Similar to other environmental risk factors such as unsafe water and indoor and urban air pollution, the mortality burden attributable to LFS falls disproportionately on low-income regions of the world (Figure 4) (Ezzati et al. 2002).

The major strengths of these analyses lie in the use of existing global data sets for terrestrial fire emissions, meteorology, population density, and mortality. Using the WHO geographic subregions and mortality estimates helped make our findings comparable with previously reported estimates for other environmental risk factors. However, there are many limitations inherent in compiling and modeling data at a global scale. A major source of uncertainty comes from the emission factors for fire-derived aerosols that were used to model the exposure estimates. We used emission factors at the lower end of the range in the literature [see Supplemental Material, Table 2 (http://dx.doi.org/10.1289/ehp.1104422)] even though larger emission factors have been shown to improve model estimates of $PM_{2.5}$ compared with satellite and surface network observations (Chin et al. 2009; Reid et al. 2009). In addition, the sum of the black carbon and organic carbon emissions factors was often lower than the observed $PM_{2.5}$ emissions factors, likely resulting in GEOS-Chem underestimates of smoke specific $PM_{2.5}$. We also chose to be conservative in applying a linear concentration–response function because other studies have suggested higher slopes at lower $PM_{2.5}$ concentrations (Pope et al. 2009).

In the absence of empirical PM data for many regions most severely affected by LFS, we evaluated our results against global data sets of visibility and ground-based AOD,

Table 1. Estimates of the global and regional annual mortality attributable to LFS and estimates from 2 years that corresponded with strong El Niño and La Niña conditions.

Scenario	Global	Sub-Saharan Africa[a]	Southeast Asia[b]	South America[c]
Annual average (1997–2006)	339,000	157,000	110,000	10,000
EL Niño year (September 1997–August 1998)	532,000	137,000	296,000	19,000
La Niña year (September 1999–August 2000)	262,000	157,000	43,000	11,000

Results are shown for the three most severely smoke-affected regions. These estimates are based on the assumptions used in the principal analysis (see Table 2).
[a]WHO subregions 18–21. [b]WHO subregion 5 only. [c]WHO subregions 11–14.

Table 2. Results of sensitivity analyses indicating the influence of varying individual assumptions on annual global mortality estimates: proportion of principal estimate of annual mortality, when all other principal analysis assumptions are held constant.

Source of uncertainty/principal analysis assumption and variations	Annual mortality proportion
Estimated $PM_{2.5}$ concentrations	
Principal analysis: LFS $PM_{2.5}$ concentrations estimated from the combination of a global chemical transport model GEOS-Chem and satellite-derived aerosol data from MODIS and MISR	1.00
MODEL: $PM_{2.5}$ concentrations estimated from the GEOS-Chem global chemical transport model	0.68
MODIS: MODEL estimate optimized using satellite-derived aerosol data from MODIS	1.47
MISR: MODEL estimates optimized using satellite-derived aerosol data from MISR	1.20
Pattern of exposure	
Principal analysis: mortality in sporadically affected subregions estimated using daily average exposure estimates and response functions; mortality in chronically affected WHO subregions estimated using yearly mean exposure estimates and response functions	1.00
Sporadic only: mortality in all subregions estimated using daily average exposure estimates and response functions	0.41
Chronic only: mortality in all subregions estimated using yearly average exposure estimates and response functions	1.54
Shape of concentration–response function	
Principal analysis: mortality response calculated as a linear function of the $PM_{2.5}$ concentration	1.00
Log-linear: mortality response calculated as a function of the logarithm of the $PM_{2.5}$ concentration	2.31
Counterfactual exposure estimates for chronically affected regions	
Principal analysis: the counterfactual estimated for each WHO subregion as the mean of the minimum 12-month running-average smoke-specific $PM_{2.5}$ concentration for each exposure cell within the subregion	1.00
Zero: a global value of 0 μg/m^3	1.44
La Niña: cell-by-cell average for a La Niña year, September 1999–August 2000 inclusive	0.45
La Niña regional average: regional average of the values from La Niña	0.81
Cell-by-cell minimum: minimum of the 12-month running averages of each cell	0.78
Cell-by-cell categorization: global categorization of the values above at the 90th, 97th, and 99th percentiles, applying the average of the category to all cells in the category	0.82
Maximum yearly average exposure used for estimating chronic mortality impacts	
Principal analysis: maximum exposure of 50 μg/m^3 was used for estimating the mortality associated with chronic exposure	1.00
Maximum exposure of 30 μg/m^3 was used for estimating the mortality associated with chronic exposure	0.99
Range of minimum and maximum daily exposures used for estimating sporadic exposure impacts	
Principal analysis: range of exposure assessed was 5–200 μg/m^3	1.00
Most restrictive range tested: 10–100 μg/m^3	0.98
Least restrictive range tested: 1–300 μg/m^3	1.01

both of which are proxies for particulate air pollution. Although there was considerable regional variation in the degree of correlation with these independent measures, the estimated $PM_{2.5}$ performed comparatively well in sub-Saharan Africa and Southeast Asia (the two global regions with highest mortality contributions). Further reductions in uncertainty of the daily exposures could be achieved with the use of higher temporal resolution fire emission inventories. For example, Mu et al. (2011) used active fire observations from *Aqua*, *Terra*, and *GOES* satellites to develop a daily and 3-hourly fire emissions product for the 2002–2010 period.

The WHO subregions with the highest mortality were those we identified as being chronically affected by LFS (Figure 2). The principal estimate of 339,000 annual deaths is composed of 81% mortality due to chronic exposure and 19% due to sporadic exposure. When the analysis was run under the sporadic-only and chronic-only assumptions (Table 2), WHO subregions identified as chronically affected contributed 53% of the total estimates (138,000 and 520,000, respectively) in both cases.

Previous estimates of the global mortality associated with urban air pollution (Cohen et al. 2005) and smoke from household solid fuel use (Lopez et al. 2006b) assumed purely chronic exposure to PM. Our distinction between chronic and sporadic impacts is a departure from this approach, reflecting the current state of epidemiological evidence and the nature of LFS exposure. On the one hand, only a few studies have reported on the mortality effects of LFS (Hänninen et al. 2009; Morgan et al. 2010; Sastry 2002), and all have estimated associations with short-term fluctuations in PM concentrations. On the other hand, urban air pollution studies have clearly demonstrated that chronic exposure to PM is associated with greater increases in mortality than are short-term fluctuations (Pope and Dockery 2006). LFS is episodic in many parts of the world, and annual average exposures are not appropriate for estimating smoke-related mortality in those regions. Similarly, fire smoke exposure is more chronic (because of high seasonal averages) in some regions, and mortality estimates based on short-term fluctuations might be overly conservative. To date, the short-term mortality impacts for PM from landscape fires have been consistent with those of urban PM. Thus, we considered it reasonable to estimate the chronic effects of PM from LFS using conservative values for the chronic effects of PM from urban sources until more specific evidence becomes available. We were also unable to account for different population responses to air pollution. Although our coefficient for acute exposure was driven by a study in Southeast Asia, no studies conducted in sub-Saharan Africa were available.

Estimates of counterfactual exposures are highly uncertain. Human influence on landscape fire activity varies considerably between ecoclimatic regions. We set the theoretical minimum for $PM_{2.5}$ from LFS as the lowest estimated for each chronically affected WHO subregion over the decade-long study period. However, variation in fire activity during the last decade will not necessarily capture the reduction in fire activity that could be achieved in each environment. For example, tropical rainforests and peat swamps, the primary source of fire emissions in Southeast Asia, rarely burn without human instigation. If such deforestation fires were to be halted, fire activity in this region (and the associated mortality) would be minimal. However, the role of human fire management in savannas, the primary source of emissions in Africa, is less well understood because fire is an integral part of these landscapes (van der Werf et al. 2008). The large estimated influence of El Niño on mortality related to LFS implies that the burden may change in the future if climate change modifies the El Niño Southern Oscillation or drier conditions occur in places with adequate fuels and ignition sources.

Landscape fire activity has been recognized as a global-scale environmental challenge because plumes transgress international boundaries and component gases and particles contribute to climate change (Bowman et al. 2009; Pope and Dockery 2006; van der Werf et al. 2008). This first attempt to quantify the global burden of mortality attributable to LFS has demonstrated important impacts at regional and global scales. We anticipate that subsequent estimates will be improved by better exposure assessment (particularly as empirical PM data become more globally available), further epidemiological studies on mortality and morbidity associated with LFS (particularly in regions with high exposure), and improved understanding of how fire regimes can be modified to reduce smoke emissions. Reducing population level exposure to air pollution from landscape fires is a worthwhile endeavor that is likely to have immediate and measurable health benefits. Such interventions could also potentially provide benefits for the mitigation of climate change and slowing the loss of biodiversity.

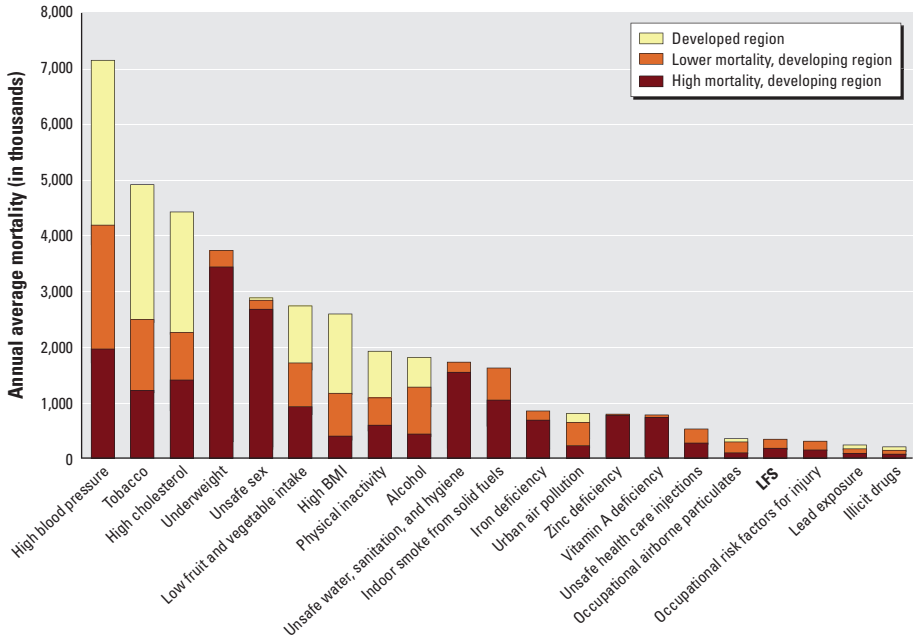

Figure 4. Annual mortality estimate for LFS in the context of estimates for other modifiable risk factors assessed as part of the WHO GBD studies (adapted from Ezzati et al. 2002).

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ORIGINAL RESEARCH

Out-of-Hospital Cardiac Arrests and Wildfire-Related Particulate Matter During 2015–2017 California Wildfires

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***Background*—**The natural cycle of large-scale wildfires is accelerating, increasingly exposing both rural and populous urban areas to wildfire emissions. While respiratory health effects associated with wildfire smoke are well established, cardiovascular effects have been less clear.

***Methods and Results*—**We examined the association between out-of-hospital cardiac arrest and wildfire smoke density (light, medium, heavy smoke) from the National Oceanic Atmospheric Association's Hazard Mapping System. Out-of-hospital cardiac arrest data were provided by the Cardiac Arrest Registry to Enhance Survival for 14 California counties, 2015–2017 (N=5336). We applied conditional logistic regression in a case-crossover design using control days from 1, 2, 3, and 4 weeks before case date, at lag days 0 to 3. We stratified by pathogenesis, sex, age (19–34, 35–64, and ≥65 years), and socioeconomic status (census tract percent below poverty). Out-of-hospital cardiac arrest risk increased in association with heavy smoke across multiple lag days, strongest on lag day 2 (odds ratio, 1.70; 95% CI, 1.18–2.13). Risk in the lower socioeconomic status strata was elevated on medium and heavy days, although not statistically significant. Higher socioeconomic status strata had elevated odds ratios with heavy smoke but null results with light and medium smoke. Both sexes and age groups 35 years and older were impacted on days with heavy smoke.

***Conclusions*—**Out-of-hospital cardiac arrests increased with wildfire smoke exposure, and lower socioeconomic status appeared to increase the risk. The future trajectory of wildfire, along with increasing vulnerability of the aging population, underscores the importance of formulating public health and clinical strategies to protect those most vulnerable. **(*J Am Heart Assoc*. 2020;9: e014125. DOI: 10.1161/JAHA.119.014125.)**

Key Words: bushfire • cardiovascular • out-of-hospital cardiac arrest • particulate matter • smoke • wildfire • wildland fire

A century of accumulated forest biomass in combination with changes in climate and forest health are accelerating the natural cycle of large-scale wildfires, exposing increasingly large populations to wildfire emissions and thereby creating the potential for smoke-related adverse health outcomes.[1–3] An estimated 57 million individuals were exposed to at least 1 episode of wildfire smoke in the United States between 2004 and 2009, and it is predicted that the number of individuals exposed yearly will grow 43% to over 82 million by midcentury.[4]

Wildfires produce massive quantities of emissions, including fine and coarse particulate matter (PM), carbon monoxide, methane, nitrous oxide, nitrogen oxides, volatile organic carbon, metals, and other toxins.[5,6] During wildfire events, air concentrations of PM can substantially exceed regulatory air quality standards[7–10] and can travel hundreds of miles to impact highly populated areas distant from the original fire.[11–13] Emissions from wildfires also contribute significantly to the burden of ambient air pollution,[1,5] accounting for ≈15% to 20% of total fine PM ($PM_{2.5}$) in the United States over the past decade.[14]

Epidemiological studies of short- and long-term air pollution exposures have consistently demonstrated associations between ambient $PM_{2.5}$ and cardiovascular-related morbidity and mortality, including ischemic heart disease and heart failure,[15–17] myocardial infarction,[18] stroke,[19–22] and

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An accompanying Data S1 is available at https://www.ahajournals.org/doi/suppl/10.1161/JAHA.119.014125

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Received July 30, 2019; accepted January 17, 2020.

Clinical Perspective

What Is New?

- Wildfire smoke exposure was associated with increased risk of out-of-hospital cardiac arrest.
- The effect appeared greater for cases of out-of-hospital cardiac arrest in lower socioeconomic communities.
- Out-of-hospital cardiac arrest has not been previously examined in the United States, and this outcome would not be included in wildfire studies based on emergency department visits or hospital admissions.

What Are the Clinical Implications?

- Smoke exposures from wildfires likely have the potential to trigger fatal and near-fatal cardiac arrest.
- Risk was the highest on dense smoke days and the effects persisted for several days following the exposure.
- Healthcare professionals and emergency medical service responders may benefit from awareness of these results to counsel patients at increased risk from the adverse health effects of poor air quality and in particular wildfire smoke on ways to limit exposure.

arrhythmias.[23] Despite these known causal relationships, evidence from epidemiological studies examining wildfire smoke exposures and cardiovascular outcomes has been mixed and inconclusive.[24,25] In a critical review, Reid et al[25] examined 66 epidemiological studies of wildfires and health effects. While they found consistent associations with respiratory outcomes, only 18 studies evaluated cardiovascular outcomes and less than a fourth of the analyses identified a statistically significant relationship. More recently, however, several studies have identified positive associations with cardiovascular end points.[11,26–30]

While respiratory conditions are prevalent and can be life-threatening, cardiovascular diseases contribute to a substantial public health burden, affecting 1 in 3 adults in the United States, an estimated 92 million individuals, and resulting in an annual economic cost of $316 billion for direct and indirect costs.[31] Out-of-hospital cardiac arrest (OHCA) represents a significant component of adverse cardiovascular events and is a leading cause of death among Americans.[32] Over 300 000 people in the United States experience OHCAs annually and 92% of these events result in sudden death.[33,34] In this context, greater clarity on inconsistencies in cardiovascular-wildfire smoke research is urgently needed to provide appropriate, evidence-based public health guidance, particularly to patients with underlying cardiovascular conditions.

Previous studies of wildfire smoke exposures and cardiovascular health have primarily relied on data from hospital admissions and emergency department visits.[24,25] However, hospitalization data do not capture all clinical events, as ≈70% of emergency medical service (EMS)–treated OHCA cases do not survive to hospital admission.[34] OHCA is an outcome that has only been examined in a few studies. A research group in Australia using a different OHCA measure examined a severe wildfire season in 2006–2007 in 2 analyses.[35,36] Researchers in Singapore also studied OHCA, but used a composite, multicontaminant measure of ambient air pollution rather than wildfire smoke or PM specifically; air quality in Singapore is regional and influenced by haze drifting from illegal agricultural burning in neighboring islands.[27,37,38] These OHCA investigations are among the limited number of epidemiological studies of biomass smoke demonstrating a relationship with a cardiovascular outcome. To the authors' knowledge, this is the first study to examine OHCA and wildfire smoke in the United States.

Our study investigated OHCA and wildfire smoke exposures in 14 climatically and demographically diverse California counties from 2015 to 2017 in order to advance our understanding of the relationship between wildfire smoke and cardiovascular health. The specific aims of our study were to investigate the impact of wildfire-related $PM_{2.5}$ on OHCA and to characterize these relationships within subpopulations by age, sex, and socioeconomic status (SES).

Methods

Data Accessibility

Because of the sensitive nature of the data collected for this study, requests to access the CARES (Cardiac Arrest Registry to Enhance Survival) data set from qualified researchers meeting the CARES data use criteria may be sent to CARES at cares@emory.edu. All exposure data have been made publicly available at the National Oceanic and Atmospheric Administration's (NOAA's) Hazard Mapping System (HMS) Archive and can be accessed at https://satepsanone.nesdis.noaa.gov/pub/volcano/FIRE/HMS_ARCHIVE/.

Health and Population Data

Reports of OHCA from January 1, 2015, to December 31, 2017, were provided by CARES, a surveillance data set from EMS and hospital providers designed to improve survival. In 2004, the Centers for Disease Control and Prevention (CDC) established CARES in collaboration with the Department of Emergency Medicine at the Emory University School of Medicine. CARES is operated on a national scale in 42 states, with a catchment of more than 130 million people.[39] CARES collects reports of OHCAs treated by EMS, excluding patients dead on EMS arrival to the scene or with do-not-resuscitate directives. Incident reports, including demographic, medical, and survival outcome

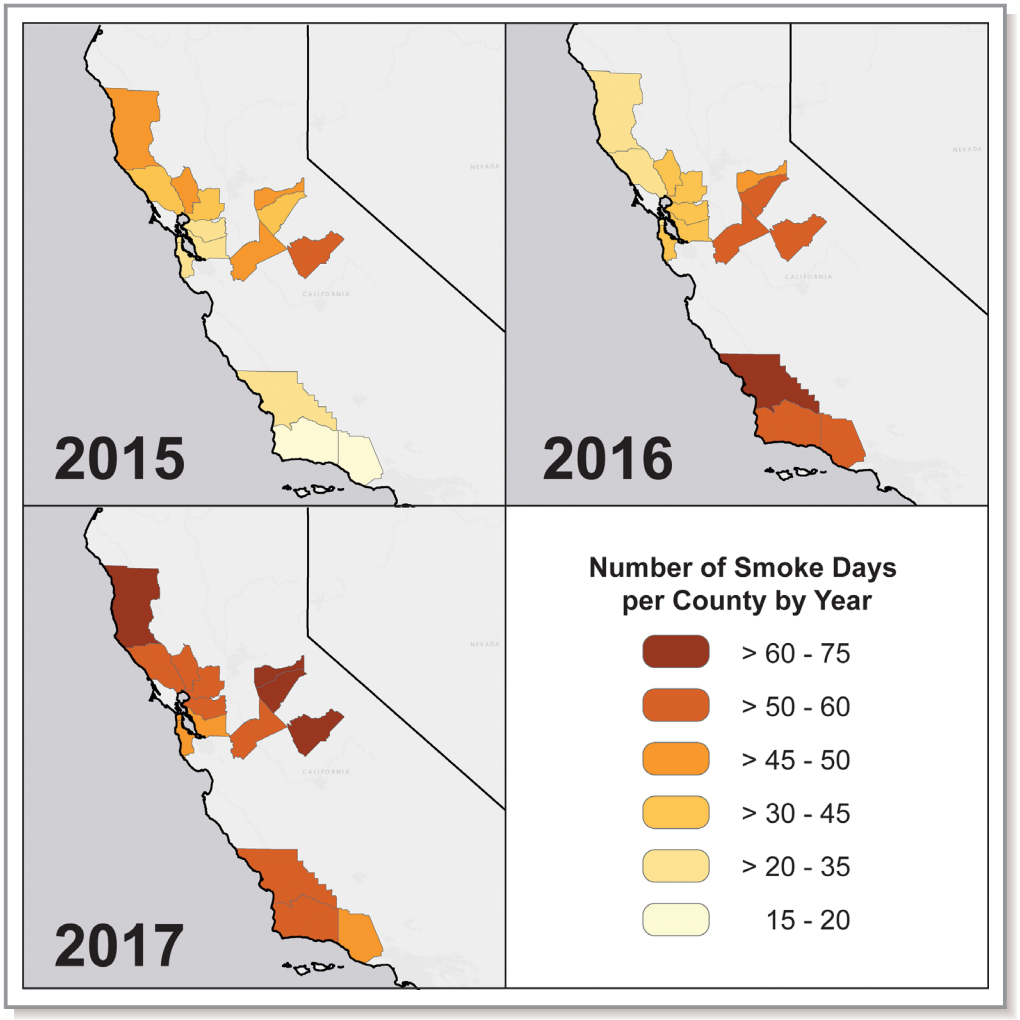

Figure 1. Map of the 14 California counties in the study region showing the number of smoke days in each county, 2015–2017, based on the National Oceanic and Atmospheric Administration's Hazard Mapping System wildfire smoke plume products.

data, are informed by EMS assessments and bystander reports and hospital outcome measures.

We obtained local permissions for receiving deidentified data from 14 California counties participating in CARES during 2015–2017 with wildfires of large size (≥50 000 acres burned) or long duration (≥50 days) within or neighboring the county during the study period: Alameda, Amador, Calaveras, Contra Costa, Mariposa, Mendocino, Napa, San Francisco, San Luis Obispo, San Mateo, Santa Barbara, Sonoma, Stanislaus, and Ventura Counties. Two counties only participated in CARES during part of the study period (Alameda County, 2016–2017; San Luis Obispo County, 2017). Counties encompass urban and rural areas across Northern, Central, and Southern California (Figure 1).

We only included cases of OHCA of presumed cardiac or respiratory/asphyxia pathogenesis and people 19 years and older. We restricted the analysis to the primary wildfire months, May to October (5336 of the 12 548 OHCA cases). Cases with origins attributed to other causes, such as drowning, trauma, overdose, or electrocution, were excluded. The address of the OHCA event defined the location of the case. We assigned census tract SES indicators using 2017 poverty data from the US Census American Community Survey 5-year estimates. We created a dichotomous variable of lower and higher SES using the federal definition of a poverty area; thus, census tracts with at least 20% of people living below the poverty level were indicated as lower SES.

Wildfire Smoke Data

Categorical estimates of smoke plume density were publicly available from the NOAA Office of Satellite and Product Operations' HMS Smoke Product. Data collected by the Geostationary Operational Environmental Satellites (GOES) and the Polar Operational Environmental Satellites (POES) are analyzed daily by algorithm and NOAA analysts, then

published as shapefiles on the NOAA website.[40] Plumes are detected using visual range of satellite images and assigned an estimated smoke-originated $PM_{2.5}$ density: light (0–10 μg/m^3), medium (10.5–21.5 μg/m^3), and heavy (>22 μg/m^3).[41]

We used the geospatial function "intersect" to spatially assign daily HMS data from NOAA's archives with US Census Block Group Population Centers to obtain a daily maximum smoke plume density by block groups in California, using the Simple Features package in R software version 3.5.0 (R Foundation for Statistical Computing). The HMS smoke product is derived primarily from instruments aboard geostationary and polar orbiting satellites and provides a spatial resolution of 1 km. Exposures at the block group level were aggregated to corresponding census tracts and the maximum smoke density was used to describe exposure of OHCA cases within respective census tracts. Three records were excluded from the analysis as a result of missing both address and latitude/longitude coordinates.

Meteorological Data

Daily mean temperature and relative humidity were obtained from the University of Idaho Gridded Surface Meteorological Dataset (gridMET) on the Google Earth Engine Data Catalog. Daily temperature and humidity were averaged for each census tract from 4-km grids using data from the National Land Data Assimilation System (NLDAS). We calculated daily heat index values for each census tract using the mean temperature and humidity values with the algorithm from Anderson et al.[42]

Statistical Analysis

We examined the association between OHCA and wildfire smoke $PM_{2.5}$ in a case-crossover study design using conditional logistic regression models. A case-crossover design, which is appropriate for investigation of acute events and has been used previously in wildfire studies, was selected to control for individual risk factors, such as age and sex, and control for day of the week.[43,44]

Wildfire smoke exposures were compared on case and noncase (control) days. Each OHCA case day was matched by day of the week with 4 control days at 1, 2, 3, and 4 weeks before the event.[44] Odds ratios (ORs) were expressed relative to days with no exposure, eg, odds of case occurrence on heavy smoke days versus odds on days without smoke.

We selected control days close to the case date to reduce selection bias and variability in individual risk factors or temporal trends that may differ beyond a month.[44,45] Heat index was incorporated as a natural cubic spline with 2 degrees of freedom. Separate models were run for exposures on lag days 0 to 3. The exposure for lag day 0 is the smoke exposure density on the day of the OHCA event or control day, for lag day 1 is the density on the day before an OHCA event or control day, for lag day 2 it is the density 2 days before an OHCA event or control day, and for lag day 3 it is the density 3 days before an OHCA event or control day.

In secondary analyses by subgroup, we explored the effects by SES, sex, and age (19–34 years, 35–64 years, and ≥65 years) in stratified analyses. We conducted a post hoc 2-sample *t* test for assessing statistically significant differences in ORs between lower and higher SES subgroups using point estimates and standard errors estimated for each group (see Data S1 for formula). We also conducted a sensitivity analysis restricted to OHCA cases of presumed cardiac pathogenesis. All analyses were conducted with SAS statistical software version 9.4 (SAS Institute Inc.). This research was approved by the Committee for the Protection of Human Subjects of the State of California Health and Human Services Agency (project number: 2018-202). Informed consent was not required, as participants were not directly involved.

Results

Characteristics of the Study Population

In 2015–2017, there were 5336 cases of OHCA occurring in wildfire months that met our inclusion criteria. Of these, 16.4% (877) were exposed to wildfire smoke. Descriptive statistics for sex, age group, SES group, and pathogenesis by exposure are shown in Table 1. Cases categorized as male, 65 years and older, reported in higher SES areas, and with presumed cardiac pathogenesis represented a greater proportion of the cases.

Wildfire Smoke Exposures

The number of days impacted by wildfire smoke increased each year in study counties (Figure 1). Heavy smoke days tended to be most frequent in July to September, although in 2017 the highest proportion occurred in October (Table 2). Heavy smoke frequency coincided roughly with the highest heat index values.

Out-of-Hospital Cardiac Arrest

Within the overall study population, ORs for OHCA were consistently elevated on days with heavy smoke and up to 3 days following exposure (Figure 2; Table 3). Associations between OHCA and heavy smoke exposure were significant at lag days 0, 2, and 3 (OR, 1.56 [95% CI, 1.05–2.33]; OR, 1.70 [95% CI, 1.18–2.45]; and OR, 1.48 [95% CI, 1.02–2.13], respectively). The association for lag day 1 was also elevated and consistent with the heavy smoke effect at other lag days

Table 1. Sociodemographic Factors of Patients With OHCA, by Number and Percent Exposed to Wildfire Smoke, in 14 California Counties for May 2015 to October 2017.

	Total Patients		Exposed Patients	
	N=5336	% of Total Patients	No.	% of Row Category
Pathogenesis				
Presumed cardiac	4967	93.1	811	16.3
Respiratory/asphyxia	369	6.9	66	17.9
SES				
Lower SES	1017	19.1	188	18.5
Higher SES	4319	80.9	689	16.0
Sex				
Women	1902	35.6	313	16.5
Men	3434	64.4	564	16.4
Age, y				
19 to 34	205	3.8	36	17.6
35 to 64	2057	38.5	358	17.4
≥65	3074	57.6	483	15.7
Total	5336	100.0	877	16.4

OHCA indicates out-of-hospital cardiac arrest; SES, socioeconomic status.

but not statistically different from null (OR, 1.20; 95% CI, 0.80–1.79). Associations with light and medium densities of smoke appeared null or at times negative in the study population as a whole. In the sensitivity analysis restricted to cases of cardiac pathogenesis only, which excluded 7% of cases thought to have an underlying respiratory mechanism as the primary cause of arrest, effects persisted for heavy smoke at lag days 0 and 2 (OR, 1.52 [95% CI, 1.00–2.31] and OR, 1.66 [95% CI, 1.14–2.43], respectively) (Table 4).

Socioeconomic Status

Risk of OHCA was elevated for both lower and higher SES groups with exposure to heavy smoke, with significant positive associations observed for higher SES cases with heavy smoke at lag days 0 and 2 (OR, 1.59 [95% CI, 1.02–2.49] and OR, 1.60 [95% CI, 1.07–2.40], respectively) (Figure 2; Table 3). In the lower SES group, ORs for heavy smoke were similar in magnitude but not statistically significant (lag day 0: OR, 1.47 [95% CI, 0.62–3.51] and lag day 2: OR, 2.25 [95% CI, 0.90–5.61) (Figure 2). At light and medium smoke densities, the lower SES group had elevated but not significant ORs for nearly all lags, while the higher SES group had a consistent negative effect, which was statistically significant for medium smoke at lag day 2 (OR, 0.78; 95% CI, 0.61–0.98). Overall, although both SES groups had elevated risk with heavy smoke exposure, lower SES cases tended to have elevated effects at medium and possibly light smoke, while higher SES cases showed null results or deficits. However, a significant difference in effects between lower and higher SES was observed only for medium smoke at lag day 2. As these secondary analyses had overlapping CIs and small sample sizes, differences between these subgroups studied, ie, SES, sex, and age, are uncertain based on these data.

Sex

Analysis by sex showed that both men and women experienced increases in OHCA under heavy smoke conditions (Figure 3; Table 5). Risk in women was highest with heavy smoke at lag day 0 (OR, 2.02; 95% CI, 1.10–3.70); and the highest OR in men occurred on lag day 2 with heavy smoke exposure (OR, 1.67; 95% CI, 1.08–2.59). Small cell sizes prevented further analysis by multiple strata.

Age Group

Heavy smoke increased risk of OHCA across age groups at multiple lags, although the age group 19 to 34 years had an insufficient number of cases and is not displayed in the figures. The 35- to 64-year age group experienced their highest risk at lag day 0 (OR, 1.91; 95% CI, 1.07–3.42) (Figure 4; Table 6). A delayed effect was suggested in the age group 65 years and older for heavy smoke, as the association with OHCA appeared stronger on later lag days (lag day 2: OR, 2.12 [95% CI, 1.31–3.46]; lag day 3: OR, 1.67 [95% CI, 1.02–2.72). However, similar limitations to our subgroup comparisons apply to any interpretation based on comparing differences in timing (lags) and whether they reflect a biologically or behaviorally meaningful pattern.

Discussion

Investigation of wildfire smoke exposure and OHCA in the diverse California population across varying geographies has provided additional evidence for an association between wildfire smoke and clinically important cardiovascular outcomes. In our analysis of 14 wildfire-impacted California counties in 2015–2017, we observed a significant increase in OHCA with exposure to heavy density of wildfire smoke. This increased risk persisted for several days after exposure, and consistent associations were present across study population subgroups. Because the majority of patients with OHCA do not survive to hospital admission, this surveillance data enabled us to capture effects for cases that would not have been included in previous wildfire studies examining cardiovascular outcomes based on hospital admissions or emergency department visits.[7,30,46,47]

Table 2. Percentage of Study Days by Month and Year in Census Tracts From 14 California Counties by Wildfire Smoke Density and Mean and Maximum Heat Index for May 2015 to October 2017

Smoke Density	May	June	July	August	September	October	November
2015							
None, %	92.0	94.0	95.3	74.7	80.4	95.6	99.9
Light, %	7.8	5.9	3.5	17.7	15.5	4.1	0.0
Medium, %	0.2	0.1	1.0	6.0	4.0	0.4	0.0
Heavy, %	0.0	0.0	0.2	1.5	0.1	0.0	0.0
Heat index, mean, °F	58.0	66.7	68.4	70.2	69.7	66.8	51.6
Heat index, maximum, °F	77.4	87.9	87.4	88.5	89.0	85.5	75.5
2016							
None, %	99.9	96.1	84.0	53.1	58.9	99.9	100.0
Light, %	0.1	3.3	10.0	40.5	38.1	0.1	0.0
Medium, %	0.0	0.5	3.9	5.9	2.9	0.0	0.0
Heavy, %	0.0	0.1	2.1	0.6	0.1	0.0	0.0
Heat index, mean, °F	61.1	66.2	66.7	66.1	65.6	61.2	55.9
Heat index, maximum, °F	83.9	86.1	87.3	85.2	84.5	79.7	77.2
2017							
None, %	99.9	99.9	87.6	46.3	61.7	73.8	100.0
Light, %	0.1	0.2	10.4	51.5	21.5	16.0	0.0
Medium, %	0.0	0.0	1.4	1.9	16.3	4.5	0.0
Heavy, %	0.0	0.0	0.6	0.3	0.5	5.7	0.0
Heat index, mean, °F	61.4	66.4	68.8	69.2	68.9	62.4	55.6
Heat index, maximum, °F	83.6	99.1	91.5	95.8	93.9	87.0	79.7

Although the pathophysiological mechanisms linking wildfire smoke exposure specifically to OHCA have not been studied, much is known about the mechanisms relating particle air pollution to cardiovascular outcomes, such as acute ischemia, myocardial infarction, heart failure, thromboembolism, and arrhythmia, conditions known to place an individual at risk for sudden death.[48] This body of knowledge recognizes multiple pathways by which wildfire smoke can disrupt the cardiovascular system.

PM deposits in pulmonary airways and alveoli, prompting imbalances in the autonomic nervous system, inflammation, and oxidative stress. The primary initiating pathways stem from oxidative stress and direct translocation of PM from the lung to the circulation. Secondary pathways have been described that include effects on vascular function, activation of central nervous system pathways, activation of prothrombotic pathways, activation of the hypothalamic and pituitary-adrenal axis, systemic inflammatory pathways, and epigenetic changes.[49] Figure 5 shows a conceptual model of proposed adverse outcome pathways that transduce wildfire smoke exposure to clinically relevant cardiovascular outcomes including OHCA based on what is known about airborne PM.[48] Such a conceptual model is important for developing research to investigate mechanisms triggering cardiovascular events including out-of-hospital cardiac death.

In addition to underlying cardiovascular disease, conditions such as aging, obesity, and diabetes mellitus are important biologic modifiers of cardiac electrophysiology that might influence the mechanistic relationship between PM and cardiac arrest. A variety of chronic respiratory conditions, such as asthma, chronic obstructive pulmonary disease, pulmonary fibrosis, and pulmonary hypertension, if severe, might also place individuals at risk for sudden cardiac death.

Our results are consistent with the few other studies that examined wildfire smoke and OHCA. Although the Singapore OHCA study used a multipollutant exposure metric not directly comparable with our study,[37,38] they observed increased risk at moderate and unhealthy exposure levels.[27] The OHCA registry used in the 2 Australian studies had broader inclusion criteria than CARES, allowing cases in which resuscitation was not attempted by EMS.[35,36,51] One of these analyses examined hourly exposure levels and cumulative lag

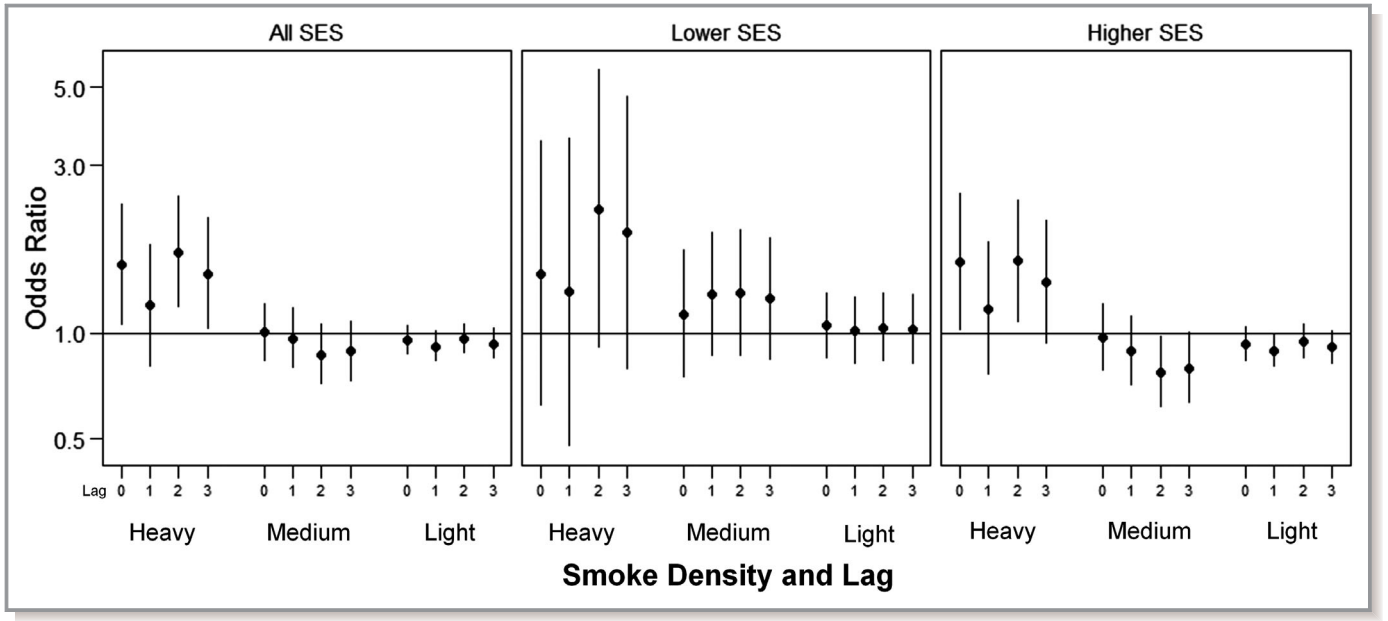

Figure 2. Odds ratios and 95% CIs for out-of-hospital cardiac arrest in 14 California counties, May 2015 to October 2017, by wildfire smoke exposure on lag days 0 to 3 for the whole study population and stratified by socioeconomic status (SES).

effects, showing the strongest effect across 24 to 48 hours, similar to our finding of the strongest effect for heavy smoke with a lag effect of daily averaged exposure.[35]

Precipitating conditions to OHCA such as ischemic heart disease and cardiac arrhythmia have also been examined for associations with wildfire smoke, but while some recent studies have found positive associations,[29,30,36,52] other studies have null or even negative findings.[47,52–56] However, in a previous analysis, we found significant increases in emergency department visits for arrhythmia, among others, with the greatest effect among adults 65 years and older exposed to dense smoke.[30] Possibly because of the high fatality rate with OHCA, wildfire studies reporting OHCA or mortality have tended to result in more consistent associations; significant positive associations were seen in 8 of 13 all-cause or cardiovascular mortality analyses in Reid's critical review.[25] This may reflect in a situation in which acute cardiovascular events are not always captured in hospital or emergency department studies.

Our study and other studies have shown deficits in cardiovascular events, and while we do not hypothesize a true physiologically protective effect, further exploration is

Table 3. OHCA and Wildfire Smoke in 14 California Counties for May 2015 to October 2017, Within the Whole Study Population and Stratified by SES for Patients With OHCA Who Had Presumed Cardiac or Respiratory/Asphyxia Pathogenesis

		Lag Day 0		Lag Day 1		Lag Day 2		Lag Day 3	
SES	Smoke Density	No.	OR (95% CI)	No.	OR (95% CI)	No.	OR (95% CI)	No.	OR (95% CI)
All SES	Heavy	34	1.56 (1.05–2.33)*	31	1.20 (0.80–1.79)	41	1.70 (1.18–2.45)*	39	1.48 (1.02–2.13)*
	Medium	153	1.01 (0.83–1.22)	142	0.97 (0.79–1.18)	134	0.87 (0.71–1.06)	139	0.88 (0.72–1.08)
	Light	691	0.95 (0.86–1.05)	679	0.92 (0.83–1.01)	687	0.96 (0.87–1.06)	681	0.93 (0.84–1.03)
Lower SES	Heavy	7	1.47 (0.62–3.51)	5	1.31 (0.48–3.58)	7	2.25 (0.90–5.61)	7	1.93 (0.79–4.73)
	Medium	34	1.14 (0.75–1.73)	36	1.30 (0.87–1.95)	35	1.30 (0.86–1.96)	39	1.22 (0.81–1.83)
	Light	148	1.04 (0.84–1.30)	140	1.02 (0.81–1.27)	137	1.03 (0.82–1.30)	136	1.03 (0.82–1.29)
Higher SES	Heavy	27	1.59 (1.02–2.49)*	26	1.17 (0.76–1.82)	34	1.60 (1.07–2.40)*	32	1.40 (0.93–2.10)
	Medium	119	0.97 (0.78–1.21)	106	0.89 (0.71–1.12)	99	0.78 (0.61–0.98)*	100	0.80 (0.63–1.00)
	Light	543	0.93 (0.83–1.04)	539	0.89 (0.80–1.00)	550	0.95 (0.85–1.06)	545	0.91 (0.82–1.02)

OHCA indicates out-of-hospital cardiac arrest; OR, odds ratio; SES, socioeconomic status.
*Significant findings (α=0.05).

Table 4. OHCA and Wildfire Smoke in 14 California Counties for May 2015 to October 2017, Stratified by SES for Patients With OHCA Who Had Presumed Cardiac Pathogenesis Only

SES	Smoke Density	Lag Day 0		Lag Day 1		Lag Day 2		Lag Day 3	
		No.	OR (95% CI)	No.	OR (95% CI)	No.	OR (95% CI)	No.	OR (95% CI)
All SES	Heavy	31	1.52 (1.00–2.31)*	29	1.18 (0.78–1.79)	38	1.66 (1.14–2.43)*	35	1.39 (0.95–2.05)
	Medium	136	0.95 (0.78–1.17)	134	0.97 (0.79–1.20)	125	0.87 (0.70–1.07)	123	0.83 (0.67–1.02)
	Light	644	0.94 (0.85–1.05)	626	0.91 (0.82–1.00)	628	0.94 (0.85–1.04)	629	0.91 (0.82–1.01)
Lower SES	Heavy	6	1.33 (0.53–3.36)	5	1.31 (0.48–3.59)	7	2.47 (0.98–6.24)	7	1.94 (0.79–4.76)
	Medium	32	1.12 (0.73–1.72)	36	1.30 (0.87–1.95)	33	1.32 (0.86–2.03)	35	1.18 (0.78–1.80)
	Light	143	1.07 (0.86–1.34)	130	0.98 (0.78–1.24)	127	1.03 (0.82–1.30)	129	1.03 (0.81–1.30)
Higher SES	Heavy	25	1.58 (0.99–2.52)	24	1.15 (0.73–1.82)	31	1.53 (1.01–2.32)*	28	1.29 (0.84–1.98)
	Medium	104	0.91 (0.72–1.15)	98	0.89 (0.70–1.13)	92	0.77 (0.60–0.98)*	88	0.74 (0.58–0.95)*
	Light	501	0.91 (0.82–1.03)	496	0.89 (0.79–1.00)	501	0.92 (0.82–1.03)	500	0.89 (0.79–1.00)

OHCA indicates out-of-hospital cardiac arrest; OR, odds ratio; SES, socioeconomic status.
*Significant findings (α=0.05).

warranted to investigate potential reasons.[47,52,57] Johnston and colleagues[52] noted deficits for cardiac arrhythmia, positing that this effect may be caused by an offsetting increase in cardiac arrests occurring outside a hospital setting, thereby decreasing the hospital presentations.

Competing risks may also play an important role in a lack of association or deficit for cardiovascular outcomes. Given the substantial prevalence of comorbidity in people with both cardiovascular and respiratory conditions in the population,[58] it may be possible that people with both types of conditions may first develop a respiratory problem and seek emergency care or be hospitalized for this condition, and thus be prevented from developing an adverse cardiovascular event during a wildfire exposure period. DeFlorio-Barker et al[26] similarly suggested that acute respiratory effects of PM may reduce the risk pool for cardiovascular events in their analysis of cardiopulmonary hospitalizations for older adults.

Our study findings are consistent with previous research on the importance of SES vulnerability factors in wildfire impacts.[59] Besides external factors such as exposure or adaptive capacity, lower SES populations may have a greater

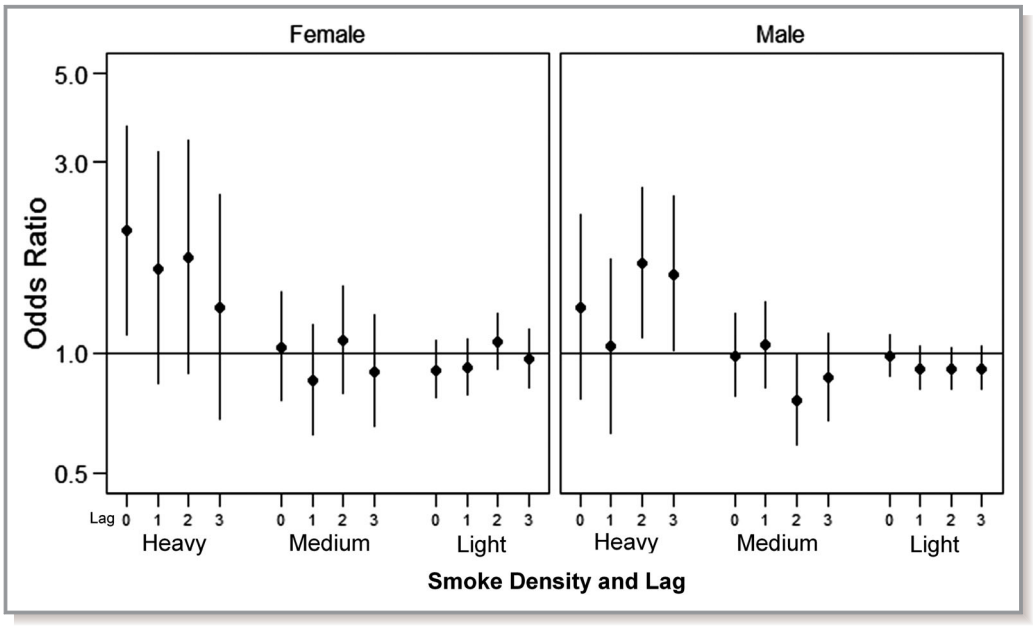

Figure 3. Odds ratios and 95% CIs for out-of-hospital cardiac arrest in 14 California counties, May 2015 to October 2017, by wildfire smoke exposure on lag days 0 to 3, stratified by sex.

Table 5. OHCA and Wildfire Smoke in 14 California Counties for May 2015 to October 2017, Stratified by Sex

Sex	Smoke Density	Lag Day 0		Lag Day 1		Lag Day 2		Lag Day 3	
		No.	OR (95% CI)	No.	OR (95% CI)	No.	OR (95% CI)	No.	OR (95% CI)
Women	Heavy	16	2.02 (1.10–3.70)*	12	1.62 (0.83–3.17)	12	1.73 (0.88–3.41)	12	1.29 (0.68–2.48)
	Medium	59	1.03 (0.76–1.42)	52	0.85 (0.62–1.18)	58	1.07 (0.78–1.47)	53	0.90 (0.65–1.25)
	Light	238	0.91 (0.77–1.07)	252	0.92 (0.78–1.08)	254	1.06 (0.90–1.26)	244	0.96 (0.81–1.14)
Men	Heavy	18	1.30 (0.76–2.21)	19	1.04 (0.63–1.72)	29	1.67 (1.08–2.59)*	27	1.57 (1.01–2.46)*
	Medium	94	0.98 (0.77–1.26)	90	1.05 (0.81–1.35)	76	0.76 (0.58–0.99)*	85	0.87 (0.67–1.12)
	Light	452	0.98 (0.87–1.11)	427	0.91 (0.81–1.04)	432	0.91 (0.80–1.03)	437	0.91 (0.81–1.04)

OHCA indicates out-of-hospital cardiac arrest; OR, odds ratio.
*Significant findings (α=0.05).

prevalence of underlying health conditions that would increase their risk of adverse health outcomes during wildfires. Rappold et al[60] found that SES factors strongly influenced the wildfire smoke effect on congestive heart failure. A study of respiratory effects from a northern California wildfire also found the highest impact in low-income zip codes.[47] In Australia, Johnston and colleagues[57] observed a deficit in cardiovascular hospital admissions for the nonindigenous population yet a consistent increase among the more vulnerable indigenous population.

In addition to the increased vulnerability in lower SES communities, several possible protective behaviors could be considered that may be more relevant to higher SES individuals, as a result of their greater capacity to make adaptive changes through behavioral modification: (1) individuals in the high-risk group, eg, with preexisting cardiopulmonary conditions, may leave the area during the fires; (2) at the advent of fires, at-risk individuals may act to decrease exposure by some combination of staying indoors, using portable air filters, and/or using N95 respirators; and (3) the at-risk group may modify activities to avoid exertion, thereby averting the biological processes that could trigger cardiac arrest. In addition to behavioral modifications that may reduce risk, higher SES individuals may also live in homes with air-conditioning and better air filtration.

Few studies have investigated differences in wildfire smoke effects for cardiovascular health outcomes by sex, with mixed results.[29,61] Two Australian OHCA studies found that men

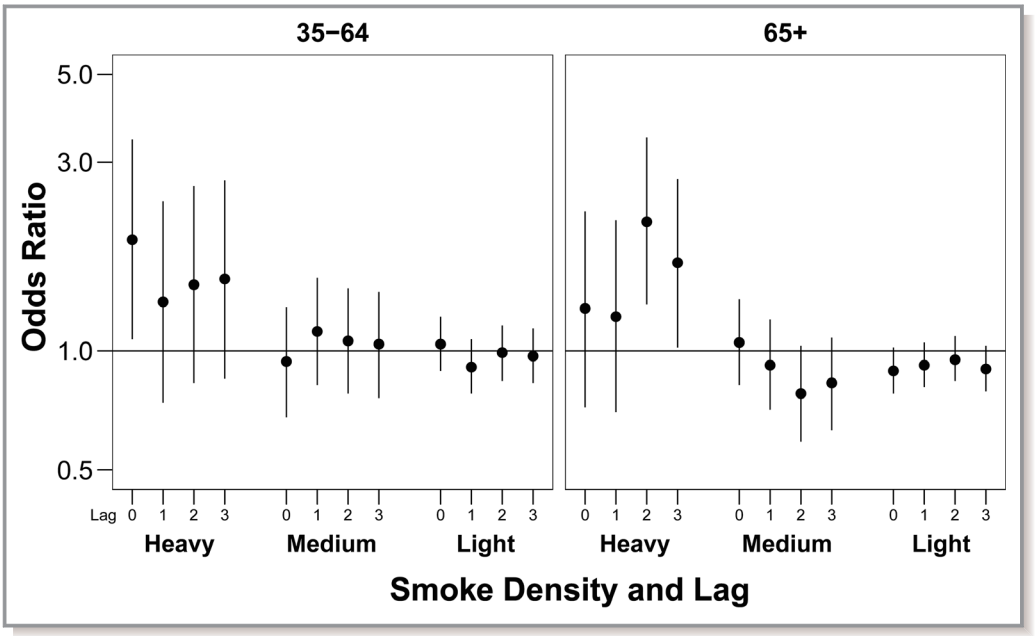

Figure 4. Odds ratios and 95% CIs for out-of-hospital cardiac arrest in 14 California counties, May 2015 to October 2017, by wildfire smoke exposure on lag days 0 to 3, stratified by age group. Younger adults aged 19 to 34 years are not shown because of low numbers.

Table 6. OHCA and Wildfire Smoke in 14 California Counties for May 2015 to October 2017, Stratified by Age Group

Age, y	Smoke Density	Lag Day 0		Lag Day 1		Lag Day 2		Lag Day 3	
		No.	OR (95% CI)	No.	OR (95% CI)	No.	OR (95% CI)	No.	OR (95% CI)
19 to 34	Heavy	<5	3.19 (0.20–52.07)	0	. . .	0	. . .	0	. . .
	Medium	7	0.97 (0.40–2.38)	5	0.46 (0.16–1.30)	<5	0.38 (0.11–1.34)	<5	0.28 (0.06–1.24)
	Light	28	0.93 (0.57–1.51)	26	0.87 (0.53–1.43)	28	0.81 (0.50–1.32)	29	0.99 (0.61–1.61)
35 to 64	Heavy	17	1.91 (1.07–3.42)*	15	1.33 (0.74–2.39)	16	1.47 (0.83–2.61)	16	1.52 (0.85–2.70)
	Medium	52	0.94 (0.68–1.29)	59	1.12 (0.82–1.53)	62	1.06 (0.78–1.44)	60	1.04 (0.76–1.41)
	Light	289	1.04 (0.89–1.22)	262	0.91 (0.78–1.07)	269	0.99 (0.84–1.16)	270	0.97 (0.83–1.14)
≥65	Heavy	16	1.28 (0.72–2.25)	16	1.22 (0.70–2.14)	25	2.12 (1.31–3.46)*	23	1.67 (1.02–2.72)*
	Medium	94	1.05 (0.82–1.35)	78	0.92 (0.71–1.20)	69	0.78 (0.59–1.03)	76	0.83 (0.63–1.08)
	Light	373	0.89 (0.78–1.02)	391	0.92 (0.81–1.05)	389	0.95 (0.84–1.09)	382	0.90 (0.79–1.03)

OHCA indicates out-of-hospital cardiac arrest; OR, odds ratio.
*Significant findings (α=0.05).

were more affected than women and suggested this may be attributable to sex differences in biologic susceptibility to cardiac arrest.[35,36] Women's cardiovascular risk may also be shaped by their levels of risk awareness for cardiovascular conditions, which remains low despite advances in research,[31] and they may therefore be less likely to take preventive measures.[62]

Aging can also modify the risk of underlying cardiovascular conditions, and the population of patients older than 65 years would be expected to have higher baseline risk. Not surprisingly, a number of studies have found stronger associations between wildfire smoke exposure and cardiovascular health effects in adults 65 or older.[11,30,36] However, we also observed elevated effects for adults aged 35 to 64 years. Similar to women, our finding that younger as well as older adults experienced elevated risk may relate to lower awareness of their potential risk, causing them to continue activities involving exertion and exposure during wildfire smoke episodes. This middle-aged adult population is of particular concern for public health officials, characterized by CDC and Centers for Medicare and Medicaid Services (CMS) as a priority population because of increasing risk for adverse cardiovascular outcomes.[63] OHCA among the younger population is likely related to uncommon congenital or familial conditions and comprises a small proportion of OHCA, limiting our ability to detect effects.[34]

Study Limitations

There are limitations within this study. Because the CARES registry includes only patients with OHCA for which EMS provided treatment, and not EMS-assessed patients who were dead on EMS arrival or presented with a do-not-resuscitate directive, our results may not be generalizable to all cases of OHCA. If case status (dead/alive) at the time of arrival is dependent on wildfire smoke exposure, our results would likely underestimate the overall risk. Our interpretation of findings assumed that probability of EMS treatment on arrival does not depend on smoke exposure, but rather is influenced by factors such as comorbidities, arrest witness status, and local EMS protocols. Considering that smoke, unlike fire, does not impact timing and delivery of EMS service, this may be a reasonable assumption to make, although difficult to verify. Also, classification of cardiac versus respiratory pathogenesis may be inaccurate, as it is largely provided by field EMS assessments and occasional bystander reports. Finally, although we considered the entire population of OHCA cases in this analysis, our sample size is limited, therefore we advise caution in interpreting results and recommend that our findings be viewed in the context of other evidence reported in the literature.

Our secondary analyses of subgroups should similarly be viewed with caution in light of the relatively small sample size in our study to detect statistically robust differences. Further investigation with a large, diverse sample would be desirable to understand potential differences and reasons for these differences by SES, age, and sex.

Exposure misclassification may also impact this study because we cannot presume that the place of exposure is consistent across lag days. However, smoke exposure is typically spatially widespread on any given day, and the majority of OHCA cases in the United States, ≈66%, are reported to occur at private residences.[33]

Our exposure is classified based on satellite images of smoke plumes. While satellite imagery is good at depicting spatial and temporal domain of exposure and contrasts between high and low exposure days, it does not capture smoke concentrations at ground level, which monitoring data

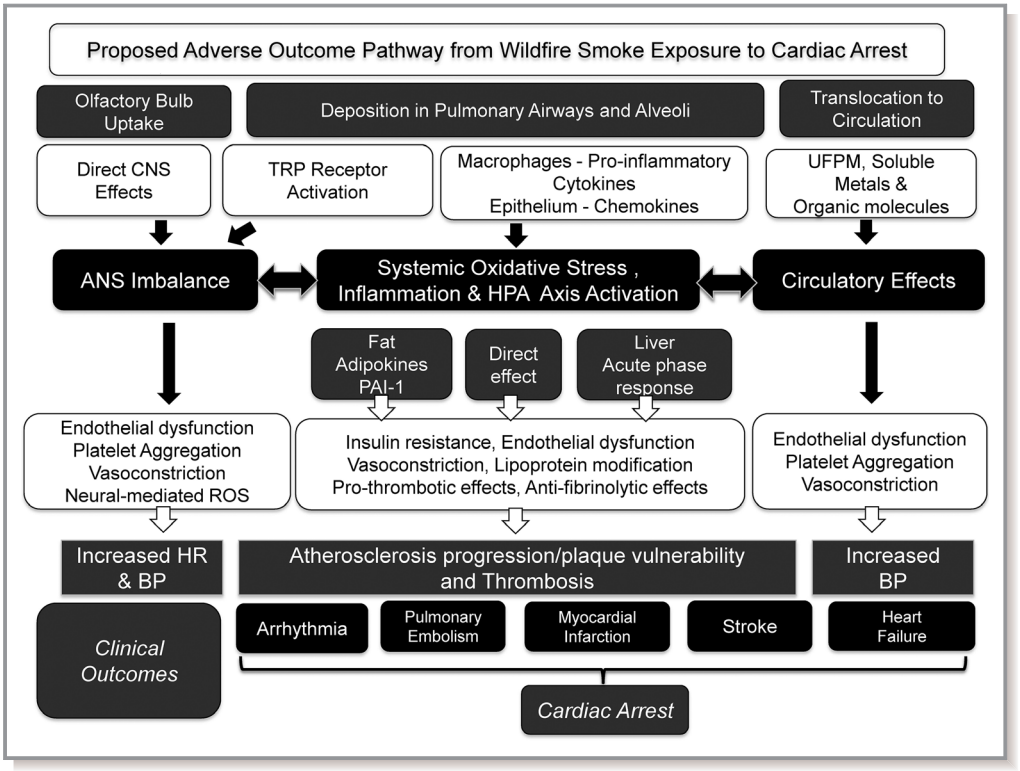

Figure 5. Biochemical and physiological responses to inhaled particulate matter: linking inhalation of wildfire smoke to out-of-hospital cardiac arrest. Inhaled ambient air particles deposit in airways and alveoli activating the transient receptor potential (TRP) channels receptors and modulating autonomic control of the heart rhythm and affecting vascular function. Pulmonary macrophages and epithelium produce proinflammatory cytokines and chemokines and contribute to systemic oxidative stress and inflammation mediating responses in adipose tissue and the liver (C-reactive protein, fibrinogen). These responses are associated with vascular effects, insulin resistance, lipoprotein modification, increased coagulation, and decreased fibrinolysis. Ultrafine particulate matter (PM) and soluble components of PM translocate to systemic circulation. Direct translocation to the central nervous system (CNS) through the nose and olfactory bulb has also been postulated. The biological and physiological responses appear to accelerate atherosclerosis and contribute to plaque vulnerability over the long-term and thrombosis in the short-term. Short-term clinical responses to ambient air particle pollution include myocardial infarction, stroke, pulmonary embolism, heart failure, and arrhythmia (these apical end points are also documented in response to wildfire smoke in[30]) ANS indicates autonomic nervous system; BP, blood pressure; DVT, deep venous thrombosis; HPA, hypothalamic and pituitary adrenal; HR, heart rate; PAI-1, plasminogen activator inhibitor-1; PE, pulmonary embolism; ROS, reactive oxygen species; UFPM, ultrafine particulate matter. Adapted from.[50]

would provide. However, the lack of comprehensive placement of stationary monitors presents other shortcomings, requiring further calculations to estimate concentrations for much of California. The validity of satellite-derived HMS plumes is supported by recent studies correlating elevated $PM_{2.5}$ concentrations measured by ground monitors with the presence and density of HMS plumes.[64,65] To investigate this issue further, we compared HMS smoke plumes during 2016–2017 with daily averages of hourly $PM_{2.5}$ measurements from monitors in California, which are part of the US Environmental Protection Agency's (EPA's) air quality system.[66] We found that average $PM_{2.5}$ concentrations at monitor locations increased in correspondence with HMS smoke plume density category: no smoke: 9.6 $\mu g/m^3$; light smoke: 12.6 $\mu g/m^3$; medium smoke: 18.2 $\mu g/m^3$; heavy smoke: 26.1 $\mu g/m^3$; and all differences between categories were significant at a 95% level. Our categorical exposure metric does not allow for examination of a potential dose response function at higher concentrations. Wildfire-derived $PM_{2.5}$ has been monitored in ranges exceeding the 100s, even reaching the 1000s $\mu g/m^3$.[9,10] Effects we noted for heavy smoke may have been dependent on higher concentrations.

Mega-wildfires, which can produce high concentrations of PM, are increasingly arising from extended drought and extreme weather events combined with accumulated biomass. Climate models predict weather conditions that signal a

future in which severe wildfires and their emissions will continue to impact both rural and heavily populated urban areas for generations to come. In addition to climate-driven increases, health effects from wildfire emissions will also be intensified by demographics of the aging American population, the increasingly large proportion of the population living in the wildland-urban interface, and the rising prevalence of comorbid conditions, not only cardiopulmonary but conditions such as obesity and diabetes mellitus.

Historically in the United States, the ambient particle air pollution has been curtailed through implementation of the Clean Air Act and associated policies, which have primarily addressed emissions from power plants, vehicles, and industry. Despite renewed attention on best practices for forest management, the options to curb wildfire emissions are limited. Because of this, personal interventions to decrease exposure to smoke assume greater importance in the efforts to protect those at greatest risk, patients with cardiopulmonary disease, namely ischemic heart disease, heart failure, cerebrovascular disease, arrhythmia, chronic obstructive pulmonary disease, and asthma. Engineering interventions might include creating cleaner air spaces in homes, work places, and public buildings through improved heating, ventilating, and air-conditioning air ventilation systems. Models of the health benefit of portable high-efficiency particulate air filtration in homes of older adults at higher risk for adverse health effects from wildfire smoke exposure suggest that such an intervention would be cost-effective.[67] The need for empirically proven interventions to provide better guidance to the public at risk has been raised by multiple agencies including the National Heart, Lung, and Blood Institute; National Institutes of Environmental Health Sciences; CDC; CMS; and the US EPA.[68]

The principal finding of our study showing the temporal association between wildfire smoke exposure and OHCA provides direct evidence of cardiovascular clinical events. An expert panel convened by the American Heart Association and others recommends advising patients with cardiovascular disease about the risks from air pollution.[50] The US EPA offers continuing education for healthcare providers on particle air pollution at https://www.epa.gov/pmcourse/continuing-education-particle-pollution-course.

Conclusions

Further research should investigate ways to enhance the public's adaptive capacity to increasingly frequent and widespread wildfire smoke conditions. Addressing disparities in vulnerabilities and protective capacity is critical to mounting an effective wildfire smoke response, as low SES appears to be a factor that intensifies the health burden. Studies to assess the thresholds for health effects, as well as the mechanisms of action and physiological processes that culminate in a cardiovascular outcome, can be used to develop more targeted wildfire smoke advisories. The future trajectory of wildfire along with the increasing vulnerability of our population highlights the far-reaching nature of the threat to Californians and others worldwide and underscores the importance of formulating strategies to protect those most vulnerable.

Acknowledgments

The CARES Surveillance Group comprises all members of contributing EMS agencies and hospitals. We would like to acknowledge their contributions, although we are unable to individually list all of the members, as CARES includes individuals from the 1800+ EMS agencies and 2000+ hospitals that participate in the registry. We wish to thank the members in the contributing EMS agencies and hospitals that participate in the CARES Surveillance Group for granting inclusion of their jurisdiction's surveillance data. We greatly appreciate CARES's Director of Data Management and Software Development, Kimberly Vellano, MPH, for her expert guidance in facilitating our obtaining and interpreting their data. Caroline Collins, MPH, California Department of Public Health, assisted us in obtaining and analyzing additional exposure data. The research described in this article has been reviewed by the National Health and Environmental Effects Research Laboratory, US EPA, and approved for publication. Approval does not signify that the contents necessarily reflect the views and the policies of the Agency, nor does mention of trade names of commercial products constitute endorsement or recommendation for use. The findings and conclusions of this study are those of the authors and do not necessarily represent the official position of the California Department of Public Health.

Sources of Funding

All authors were supported for this work through their listed affiliations.

Disclosures

None.

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SUPPLEMENTAL MATERIAL

Data S1.

Supplemental Methods

The following formula for the t-test follows, where d' indicates approximated degrees of freedom used.

$$t = \frac{\widehat{OR}_{high} - \widehat{OR}_{low}}{\sqrt{SE\left(OR_{high}\right)^2 + SE(OR_{low})^2}}$$

$$d' = \left\lfloor \frac{\left[SE\left(OR_{high}\right)^2 + SE(OR_{low})^2\right]^2}{\frac{\left[SE\left(OR_{high}\right)^2\right]^2}{n_1 - 1} + \frac{[SE(OR_{low})^2]^2}{n_2 - 1}} \right\rfloor$$

A Section 508–conformant HTML version of this article is available at https://doi.org/10.1289/EHP2200.

Mutagenicity and Lung Toxicity of Smoldering vs. Flaming Emissions from Various Biomass Fuels: Implications for Health Effects from Wildland Fires

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Background: The increasing size and frequency of wildland fires are leading to greater potential for cardiopulmonary disease and cancer in exposed populations; however, little is known about how the types of fuel and combustion phases affect these adverse outcomes.

Objectives: We evaluated the mutagenicity and lung toxicity of particulate matter (PM) from flaming vs. smoldering phases of five biomass fuels, and compared results by equal mass or emission factors (EFs) derived from amount of fuel consumed.

Methods: A quartz-tube furnace coupled to a multistage cryotrap was employed to collect smoke condensate from flaming and smoldering combustion of red oak, peat, pine needles, pine, and eucalyptus. Samples were analyzed chemically and assessed for acute lung toxicity in mice and mutagenicity in *Salmonella*.

Results: The average combustion efficiency was 73 and 98% for the smoldering and flaming phases, respectively. On an equal mass basis, PM from eucalyptus and peat burned under flaming conditions induced significant lung toxicity potencies (neutrophil/mass of PM) compared to smoldering PM, whereas high levels of mutagenicity potencies were observed for flaming pine and peat PM compared to smoldering PM. When effects were adjusted for EF, the smoldering eucalyptus PM had the highest lung toxicity EF (neutrophil/mass of fuel burned), whereas smoldering pine and pine needles had the highest mutagenicity EF. These latter values were approximately 5, 10, and 30 times greater than those reported for open burning of agricultural plastic, woodburning cookstoves, and some municipal waste combustors, respectively.

Conclusions: PM from different fuels and combustion phases have appreciable differences in lung toxic and mutagenic potency, and on a mass basis, flaming samples are more active, whereas smoldering samples have greater effect when EFs are taken into account. Knowledge of the differential toxicity of biomass emissions will contribute to more accurate hazard assessment of biomass smoke exposures. https://doi.org/10.1289/EHP2200

Introduction

Each year, tens of millions of people globally experience destructive wildland fires and subsequent health impacts from smoke exposure (Levine et al. 1999). Trends for warmer and drier conditions are expected to result in greater frequency, size, and intensity of wildfires in many parts of the world (Abatzoglou and Williams 2016; Landis et al. 2017; Westerling et al. 2006). Besides the damage caused by fire itself, smoke emitted from fires is a serious public health concern. Biomass smoke is associated with increased incidence and severity of cardiopulmonary disease, and is recognized by the World Health Organization as a probable human lung carcinogen (IARC 2010; Straif et al. 2006). Consequently, the health risks due to short- and long-term exposure to wildland fire (or biomass burning) smoke are important for firefighters as well as for people living in communities near or downwind of wildland fires (Adetona et al. 2016).

Recent reviews cite numerous studies that have reported associations between wildland fires and health outcomes, including respiratory infections, asthma, cardiovascular diseases, and mortality (Liu et al. 2015; Reid et al. 2016). More specifically, it was estimated in one report that worldwide exposures to fine-fraction (<2.5 μm) particulate matter ($PM_{2.5}$) from wildland fires during 1997–2006 were associated with approximately 340,000 deaths per year, with larger numbers of deaths during years with dryer conditions and more fires (Johnston et al. 2012). In the United States, increases in forest fires during recent decades have been attributed in part to changing weather patterns that may continue to increase the likelihood, scale, and severity of fires in the future (Abatzoglou and Williams 2016; Westerling et al. 2006).

Despite the public health threat from an increased exposure to wildland fire smoke, studies examining the specific role of smoke components on disease incidence or severity following exposure are lacking. Specifically, it is important to determine whether the chemical composition of the emissions vary with the types of fuel burned and combustion conditions (flaming vs. smoldering), and how these variables affect the potential health effects of the resulting emissions. Of the myriad components in wildland fire smoke, primary and secondarily formed PM are major factors of concern because they can remain in the air for days or weeks and can be transported over long distances (Reisen et al. 2015). The spatiotemporal variability of PM, including smoldering vs. flaming emissions, can

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Supplemental Material is available online (https://doi.org/10.1289/EHP2200).

The authors declare they have no actual or potential competing financial interests.

Received 12 May 2017; Revised 15 December 2017; Accepted 15 December 2017; Published 26 January 2018.

complicate the characterization of health risks of wildland fire smoke exposure to firefighters and the general public (Adetona et al. 2016).

Several studies have compared the chemical composition of PM from wildland fires or laboratory combustions of different fuel types under different burning conditions (Burling et al. 2010; Gilman et al. 2015; McMeeking et al. 2009; Reid et al. 2005); however, less work has integrated these findings with toxicological effects of the emissions. Moreover, due to considerable variability in study design and combustion conditions within and among laboratories, it is difficult to compare the toxicological findings across reported studies.

To address these issues, we generated biomass smoke during flaming or smoldering phases of combustion from five different fuel types using a quartz-tube furnace coupled to a multistage cryotrap system. We burned red oak, peat, pine needles, pine, and eucalyptus under flaming and smoldering phases to represent contrasting fuel types. These fuel types were selected as surrogates for major forest types across the United States. We assessed the resulting PM for lung toxicity in mice by measuring a panel of biomarkers after oropharyngeal aspiration and for mutagenicity in the *Salmonella* mutagenicity assay.

The data are presented in two ways: *a*) as a potency expressed as toxicity per mass of PM, which can be used to facilitate understanding and qualitative prediction of potential health effects, and *b*) as an emission factor (EF), which reflects exposure based on mass of fuel consumed, and can be further expressed by thermal energy of fuel combustion. These latter analyses were performed in order to provide information on how wildfire emissions and potential health effects can be quantified based on fuel consumption and to provide comparison with emissions from other fuels and combustion processes.

Methods

Fuel Types

We burned five different biomass fuels in this study: northern red oak (*Quercus rubra*), pocosin peat, ponderosa pine (*Pinus ponderosa*) needles, lodgepole pine (*Pinus contorta*), and eucalyptus (*Eucalyptus globulus*). Red oak was used to represent eastern and central wildland fires in the United States and was obtained from the Air and Energy Management Division at the U.S. Environmental Protection Agency (EPA). Peat was used to represent peatland/coastal wildfires, which are found mostly in the midwestern and southeastern United States, and was collected from the coastal oligotrophic plain of eastern North Carolina (Alligator River National Wildlife Refuge) using a Russian peat borer tool (De Vleeschouwer et al. 2010). Ponderosa pine needles and lodgepole pine were used to represent western wildland fires in the United States and were provided by the U.S. Forest Service Missoula Fire Sciences Laboratory. Eucalyptus (purchased commercially from Woodworkers Source) was used to represent chaparral (i.e., fire-prone) biome-type wildland fires, which are found in most of the southern part of coastal California in the United States as well as other continents (e.g., the west coast of South America and southwestern Australia) (Kellison et al. 2013). The red oak, pine, and eucalyptus samples were cut into approximately 2-cm-long wood chips to facilitate uniform combustion conditions. The peat sample was crumbled into a loose agglomerate, whereas the pine needles were burned without further processing. All biomass fuels were stored in a temperature- and humidity-controlled room (23°C and 39% relative humidity) until used.

Combustion and Smoke Collection

Biomass combustion was conducted in a quartz-tube furnace (Klimisch et al. 1980; Werley et al. 2009) under both smoldering and flaming phases (Figure 1). This system consisted of a quartz tube (1 m long and 3.8 cm diameter) and a ring furnace (11.4 cm long). The furnace surrounding the quartz tube was mounted on a linear actuator driven with a combination travel speed controller that was set to maintain a speed of 1 cm/min as it traversed along the length of the quartz tube. The biomass fuel (15 g) was placed uniformly inside the length of the quartz tube, and the temperature was adjusted to achieve steady-state smoldering (approximately 500°C) and flaming (approximately 640°C) combustion conditions (Figure S1). The furnace system was able to sustain stable flaming or smoldering phases consistently for 60 min. The primary air flow (air through the quartz tube) was approximately 2 L/min.

We collected the smoke using a multistage cryotrap system (Figure 1). This system was employed for two principal reasons: *a*) to collect volatile and semivolatile components, which typically pass through filters, and *b*) to collect particles, which are difficult to extract from filter matrixes. Half of the outlet biomass smoke flow (approximately 1 L/min) from the tube furnace was drawn into the cryotrap system consisting of three sequential impingers maintained at −10°C, −50°C, and −70°C. PM and condensable gas-phase semivolatiles in the biomass smoke (termed smoke condensate henceforth) were captured by cryogenic trapping in the impingers. Each impinger was packed with mixed-size glass beads (1 and 0.4 cm diameter) to provide a large surface area for collection of the smoke. The other half of the biomass smoke flow (approximately 1 L/min) was diluted with secondary air flow (15 L/min) and then analyzed continuously for carbon dioxide (CO_2) and carbon monoxide (CO) using a nondispersive infrared analyzer (602 CO/CO_2; CAI, Inc.).

We also collected PM on glass–fiber filters installed in both the exhaust line of the tube furnace and the cryotrap system exhaust during the combustion (60 min) and determined mean PM concentrations gravimetrically by weighing the filter before and after PM collection. Particle-size distributions (in the range of 32 nm to 10.57 μm) were monitored in real time by an electrical low-pressure impactor (ELPI; model 97-2E; Dekati Ltd.). Number-based size distribution data were converted into the surface area–weighted distributions using the ELPIvi software (version 3.0; Dekati Ltd.) (Schmid and Stoeger 2016). Flow rates of the biomass smoke were precisely controlled by a vacuum controller (XC-40; Apex Instruments, Inc.) located at the end of each exhaust line. A pressure gauge (Magnehelic®, Dwyer Instruments Inc.) was placed in the outlet of the tube furnace to ensure a constant pressure drop throughout each burn.

Characterization of Biomass Smoke

Concentrations of CO_2, CO, and PM were used to routinely characterize the biomass smoke emissions. Flaming and smoldering combustion phases are typically characterized by modified combustion efficiency (MCE), which is defined as $\mathrm{MCE}\,(\%) = [\Delta CO_2/(\Delta CO_2 + \Delta CO)] \times 100$, where ΔCO_2 and ΔCO are the excess concentrations of CO_2 and CO (Ward and Radke 1993). We considered combustion to be flaming when the MCE was >95% and smoldering when MCE was 65–85%, as suggested by Urbanski (2014).

Smoke properties are also described using EFs, which are defined as the mass of species t emitted per mass of dry fuel consumed, which can be calculated as $\mathrm{EF}\,t(\mathrm{g/kg}) = (\mathrm{F}c \times \mathrm{C}t \times \mathrm{M}t \times 1{,}000)/(\mathrm{M}c \times \mathrm{C}_T)$, where F$c$ is the mass fraction of carbon in the dry biomass fuel (assumed to be 0.5), Mt is the molar mass of species t, Mc is the molar mass of carbon, C_T is the total mass of carbon associated to all species in the biomass smoke, and Ct is the mass of carbon emitted as species t, and given by $Ct(\mathrm{mg/m^3}) = (\mathrm{M}c \times N \times \mathrm{V}t)/24.45$, where N is the number of carbon atoms in species t, and Vt is the concentration of species t in ppm (Soares Neto et al. 2009). In order to validate EFs

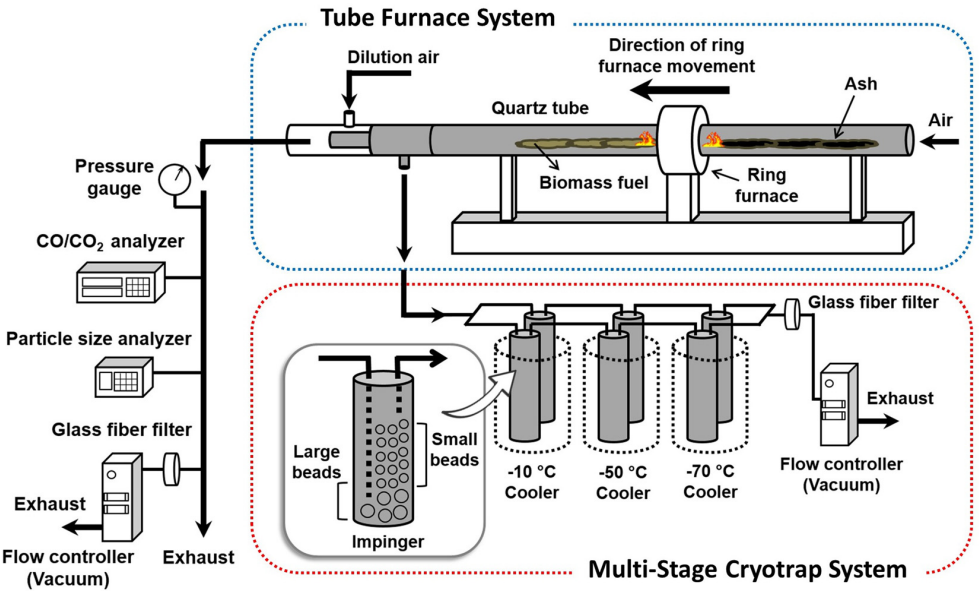

Figure 1. Diagram of the biomass combustion and smoke collection system. The tube furnace system consisted of a quartz tube and a ring furnace that traversed along the length of the quartz tube and was able to sustain stable flaming or smoldering phases consistently for 60 min. The multistage cryotrap system had three sequential impingers that were cooled cryogenically at −10, −50, and −70°C, permitting the capture of PM and semivolatile organic compounds from the biomass smoke emissions.

estimated from the tube furnace in the present study, EFs for CO, CO_2, and PM were compared with the published EFs from various fuel combustion conditions (in-ground vs. aboveground biomass fuels). We also expressed EFs per megajoule$_{thermal}$ (MJ_{th}) by using the heat energy (MJ_{th}/kg) of each fuel burned, which was 21.70 for the red oak (Ince 1979), 23.00 for the peat (Morvay and Gvozdenac 2008), 11.96 for the pine needles (de Muñiz et al. 2014), 20.00 for the pine (Nielson et al. 1985), and 19.25 for the eucalyptus (de Muñiz et al. 2014).

Biomass Smoke Condensate Analysis

Following the combustion tests, we extracted smoke condensate from the cryogenically cooled impingers and loose beads by washing them with acetone. We then pooled the smoke condensate suspension and concentrated it with a rotary evaporator (Rotavapor® R-200; Buchi). The smoke condensate was then dried under nitrogen gas to obtain predominantly solid PM (termed dried smoke condensate or PM henceforth), which underwent subsequent analyses.

For carbon species analysis, the aliquot of the smoke condensate suspension was pipetted onto prebaked 1.5-cm^2 quartz filter punches, dried, and analyzed for organic carbon (OC) and elemental carbon with a carbon analyzer (107A; Sunset Laboratory, Inc.). The OC fraction was further analyzed for polar (methoxyphenols and levoglucosan) and nonpolar [polycyclic aromatic hydrocarbons (PAHs) and *n*-alkanes] organic compounds with a thermal desorption unit (TD; TDSA2/TDS, Gerstel, Inc.) coupled to a gas chromatograph-mass spectrometer (GC-MS; 6890/5973, Agilent Technologies, Inc.). A full list of the organic compounds is shown in Table S1. The TD-GC-MS sample load (0.5–1 μg OC/μL) was optimized or determined by the OC content measured by the thermo-optical method (Hays et al. 2002). A prebaked quartz filter punch was placed inside a glass TD tube and spiked with a deuterated internal standard solution (1 μL) and aliquot of the PM suspension (1 − 12 μL). Nitrogen (50 mL/min for 40 s) was used to evaporate solvent prior to TD-GC-MS analysis. An autosampler (TDSA2, Gerstel Inc.) was utilized to insert the glass tube into the TD unit, which heated the sample (325°C) under He (50 mL/min). Sample flow was directed to a cryogenically cooled inlet (−100°C), which was rapidly heated to 300°C following the desorption step. Semivolatile organic compounds (SVOC) were chromatographed using a capillary column (30 m long and 0.25 mm inside diameter; DB-5) ramped from 65 to 300°C. The MS was operated in single ion monitoring mode. A separate TD-GC-MS analysis for levoglucosan was performed by spiking 10 μL of the smoke condensate suspension with ^{13}C levoglucosan internal standard (20 ng/μL) and reacting with 50 μL of N,O-bis(trimethylsilyl)trifluoroacetamide reagent for 30 min at 70°C. This mixture (1 μL) was spiked onto a Carbotrap F/Carbotrap C tube (Sigma-Aldrich) and dry purged under nitrogen and analyzed as described above. Samples were quantified using the internal standard method and expressed in μg/g units. SVOC concentrations were blank subtracted using an acetone solvent check (4 μL). A four-level calibration was established prior to sample analysis. The calibration ranged from 0.1 to 1 ng/μL for most PAH targets (total 28 PAHs including 16 EPA-regulated priority PAHs) and 0.63 to 6.25 ng/μL for most alkanes (total 36 alkanes). A midlevel check standard was run with each daily target set and used to assess the daily target recovery. If the midlevel check standard failed to pass the minimum agreement criterion for the number of acceptable targets, it was used as a daily continuing calibration, in which case an average response factor curve fit was used for quantification. Detection limits were established for each target listing. Raw values that fell below the detection limit threshold were listed as not detected.

For inorganic elemental analysis, the dried smoke condensate was digested in 3:1 aqua regia mixture (1 mL concentrated hydrochloric acid: 0.33 mL concentrated nitric acid, both Optima grade; Fisher Scientific) to leach trace elements. After dilution to a final concentration of 2% total acid, supernatants were separated by centrifugation (405 × g for 15 min at 22°C), then assayed for 44 target elements (listed in Table S2) by high-resolution-magnetic

sector field inductively coupled plasma mass spectrometry (HR-ICP-MS; ELEMENT™ 2; Thermo Scientific). In preparation for major ion analysis, the dried smoke condensate was diluted in 10 mL of American Society for Testing and Materials Type I ultrapure water (18.2 MΩ · cm), sonicated, and analyzed for nitrate (NO_3^-), sulfate (SO_4^{2-}), chloride (Cl^-), sodium (Na^+), ammonium (NH_4^+), potassium (K^+), magnesium (Mg^{2+}), phosphate (PO_4^{3-}), and calcium (Ca^{2+}) using a dual ion chromatography system (ICS-2000, Dionex). The smoke condensate suspension (in acetone) was solvent-exchanged into saline at a final concentration of 2 mg PM/mL and then further analyzed for pH and endotoxin levels. The pH value was measured with a calibrated pH meter (440; Corning®). For the endotoxin measurement, the dried smoke condensate suspension (in saline) was vortexed and sonicated to ensure homogeneity, and then diluted in endotoxin-free water at a concentration of 1 mg/mL. Endotoxin measurements were performed using the Limulus amebocyte lysate assay (QCL-1000™; Lonza) as per the manufacturer's protocol. Aliquots of the dried smoke condensate suspensions (in saline) were stored at −80°C until toxicity testing.

Experimental Animals

Adult pathogen-free female CD-1 mice (approximately 20-g body weight) were purchased from Charles River Breeding Laboratories and were housed in groups of five in polycarbonate cages with hardwood chip bedding at the U.S. EPA Animal Care Facility, which is accredited by the Association for Assessment and Accreditation of Laboratory Animal Care, and were maintained on a 12-h light-to-dark cycle at 22.3 ± 1.1°C temperature and 50 ± 10% humidity. Mice were given access to rodent chow and water *ad libitum* and were acclimated for at least 10 d before the study began. Mice were treated humanely and with regard for alleviation of suffering. The studies were conducted after approval by the U.S. EPA Institutional Animal Care and Use Committee. Mice were weighed and weight-randomized into 24 groups of six mice each for each exposure condition.

Mouse Exposure to the PM

We solvent-exchanged the smoke condensate suspension in acetone into saline to a final PM concentration of 2 mg/mL, and then administered it into the lungs of CD-1 mice at 100 μg in 50 μL by oropharyngeal aspiration. We performed oropharyngeal aspiration on mice anesthetized in a small plexiglass box using vaporized anesthetic isoflurane, following a previously described technique (Kim et al. 2014b). Briefly, the tongue of the mouse was extended with forceps, and 100 μg of PM in 50 μL saline was pipetted into the oropharynx. Immediately, the nose of the mouse was then covered, causing the liquid to be aspirated into the lungs. The selection of PM dose (100 μg) was based on the following information. Although PM exposure near wildland fires averages several hundred $\mu g/m^3$ (Naeher et al. 2007), some studies have identified peak values ranging from 1.9 mg/m^3 [measured PM_{10} levels at a site in India heavily affected by haze from nearby wildland fires (Naeher et al. 2007)] to 2.8 mg/m^3 [respirable $PM_{3.5}$ levels experienced by firefighters while fighting a fire (Swiston et al. 2008)]. Note that the PM dose in this study was determined based on these extreme exposure values (Naeher et al. 2007; Swiston et al. 2008). Therefore, wildfire PM deposited in the human lungs for 24 h in this particular case [assuming a human respiratory minute volume and surface area of 20 L/min (NRC 1992) and 70 m^2 (Fröhlich et al. 2016), respectively] would be 78.2–115.2 ng/cm^2. Assuming a mouse respiratory minute volume and surface area of 0.0269 L/min (Bide et al. 2000) and 642 cm^2 (Weibel 1973), respectively, mice could inhale between 74 and 108 μg (equivalent to 114.5–168.8 ng/cm^2) of wildfire PM over a 24-h period. We chose a single PM dose of 100 μg because *a*) this dose represents a peak 24-h exposure for a wildfire event, and *b*) this dose (equivalent to 154 ng/cm^2 in mouse lungs) appeared to be relevant to the inhaled wildfire PM concentrations in the human lungs. Moreover, because the same PM dose was used in other lung toxicity studies (Gilmour et al. 2007; Kim et al. 2014a, 2014b, 2015), the chosen PM dose enabled us to examine the comparative lung toxicity of various inhaled particles. We instilled additional mice with 2 μg of lipopolysaccharide in 50 μL saline (LPS; *Escherichia coli* endotoxin; 011:B4 containing 10^6 unit/mg material; Sigma-Aldrich) as a positive control to demonstrate maximal responsiveness to this well-characterized inflammatory agent. We also instilled additional mice with 50 μL saline alone as a negative control.

Lung Toxicity Assay

At 4 and 24 h postexposure, six mice from each treatment group were euthanized with 0.1 mL intraperitoneal injection of Euthasol (diluted 1:10 in saline; 390 mg pentobarbital sodium and 50 mg phenytoin/mL; Virbac AH Inc.), and blood was collected by cardiac puncture using a 1-mL syringe containing 17 μL sodium citrate to prevent coagulation. The trachea was then exposed, cannulated, and secured with suture thread. The thorax was opened, and the left mainstem bronchus was isolated and clamped with a microhemostat. The right lung lobes were lavaged three times with a single volume of warmed Hanks balanced salt solution (HBSS; 35 mL/kg mouse). The recovered bronchoalveolar lavage fluid (BALF) was centrifuged at 300 × g for 10 min at 4°C, and the supernatant was stored at both 4°C (for biochemical analysis) and −80°C (for cytokine analysis). The pelleted cells were resuspended in 1 mL HBSS (Sigma-Aldrich). Total BALF cell count of each mouse was obtained by a Coulter counter (Beckman Coulter Inc.). Additionally, 200 μL resuspended cells were centrifuged in duplicate onto slides using a Cytospin™ (Shandon™) and subsequently stained with Diff-Quik solution (American Scientific Products) for enumeration of macrophages and neutrophils with at least 200 cells counted from each slide. Hematology values including total white blood cells, total red blood cells, hemoglobin, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin concentration, and platelets were measured using a Coulter® AcT 10 Hematology Analyzer (Beckman Coulter, Inc.).

Albumin and total protein concentrations in BALF were measured by the SPQ™ test system (DiaSorin) and the Coomassie Plus Protein Assay (Pierce Chemical) with a standard curve prepared with bovine serum albumin (Sigma-Aldrich), respectively. Concentrations of lactate dehydrogenase (LDH) and γ-glutamyl transferase (GGT) in BALF were determined using commercially available kits (LDH-L Reagent and Gamma GT Reagent, Thermo Scientific). Activity of *N*-acetyl-β-D-glucoaminidase (NAG) in BALF was determined using a NAG assay kit (Roche Applied Science). All biochemical assays were modified for use on the KONELAB 30 clinical chemistry spectrophotometer analyzer (Thermo Clinical Lab Systems), as described previously (Kim et al. 2014a). Concentrations of tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6) and macrophage inhibitory protein-2 (MIP-2) in BALF were determined using commercial multiplexed fluorescent bead-based immunoassays (MILLIPLEX® Map Kit, Milliore Co.) measured by a Luminex® 100™ (Luminex Co.) following the manufacturer's protocol. The limits of detection (LOD) of each cytokine were 6.27, 3.28, and 29.14 pg/mL for TNF-α, IL-6, and MIP-2, respectively, and all values below these lowest values were replaced with a fixed value of one-half of the LOD value.

We calculated the lung toxicity potency by determining the neutrophil counts in BALF (i.e., an equal PM mass basis). We

then multiplied these values (neutrophils/μg PM) by the calculated EF for PM (g PM/kg fuel) for each fuel and burning condition to give the lung toxicity EF (neutrophils/kg fuel).

Mutagenicity Assay

For mutagenicity analysis, we dried the smoke condensate suspension under nitrogen gas (TurboVap II; Zymark), resuspended the dried smoke condensate in dichloromethane (DCM), sonicated it for 45 min, and filtered the extractable organic material (EOM) sequentially through 0.2- and 0.02-μm Anotop filters (Whatman, Midland Scientific Inc.). We determined the percentage EOM by gravimetric measurement performed by adding 100 μL of DCM extract to each of three preweighed aluminum weighing boats. The DCM was evaporated by heating the boats at 100°C until dry; then the cooled boats were weighed again. The three different weights were averaged and represented to micrograms of EOM/μL of DCM extract. We solvent-exchanged the EOM into dimethyl sulfoxide (DMSO) at 10 mg EOM/mL DMSO.

We performed the *Salmonella* plate-incorporation mutagenicity assay (Maron and Ames 1983) using the base-substitution strain TA100 [*hisG46 chl-1005 (bio uvrB gal) rfa-1001* pKM101$^+$ Fels-1$^+$ Fels-2$^+$ Gifsy-1$^+$ Gifsy-2$^+$] and the frameshift strain TA98 [*hisD3052 chl-1008 (bio uvrB gal) rfa-1001* pKM101$^+$ Fels-1$^+$ Fels-2$^+$ Gifsy-1$^+$ Gifsy-2$^+$] (Porwollik et al. 2001). We evaluated the EOM in the presence and absence of metabolic activation using S9 mix/plate composed of 1 mg S9 protein/500 μL of S9 mix (Maron and Ames 1983); S9 was an aroclor-induced Sprague-Dawley rat liver homogenate (Moltox). TA100 and TA98 have been used extensively to evaluate the mutagenicity of biomass emissions (Bell and Kamens 1990; IARC 2010). Strain TA100 + S9 detects base-substitution mutagens, such as PAHs, TA98 + S9 detects frameshift mutagens such as PAHs and aromatic amines, and TA98 − S9 detects nitroarenes. As positive controls, 2-aminoanthracene (for TA98 + S9 and TA100 + S9), 2-nitrofluorene (for TA98 − S9), and sodium azide (for TA100 − S9) were used, and DMSO was used as a negative control.

With some exceptions due to limited sample quantity, the samples were evaluated among nine doses (5, 10, 20, 25, 40, 100, 200, 250, and 500 μg EOM/plate) at one plate/dose in four independent experiments. We defined a positive mutagenic response as a reproducible, dose-related response with an increase in revertants (rev) per plate relative to the DMSO control from the four independent experiments. We calculated the mutagenic potency by determining the linear regressions over the linear portion of the dose–response curves created by the average of the primary data (rev/plate) from the four independent experiments (Figures S2 and S3). The linear portion was defined by the line with the highest coefficient of determination (r^2) value. Dose–response data outside of the linear portion were not used in the linear regressions because these resulted in a downturn in the curve and a reduction of the r^2 values.

We multiplied the mutagenic potencies of the EOM (rev/μg EOM) by the percentage EOM to give the mutagenic potencies of the PM (rev/μg PM) for each fuel/combustion condition. We then multiplied these values (rev/μg PM) by the calculated EF for PM (g PM/kg fuel) for each fuel and burning condition to give the mutagenicity EF (rev/kg fuel). We then converted the rev/kg fuel to rev/MJ_{th} using the values for the heat energy of the fuels (MJ_{th}/kg) described in the "Characterization of Biomass Smoke" section. In order to evaluate the mutagenicity EFs of the biomass smoke in the present study, the rev/MJ_{th} values were compared with the published mutagenicity EFs for red oak burned in cookstoves as well as for a variety of other emissions available from the literature.

Statistical Analysis

For the analysis of lung toxicity data (pro-inflammatory cytokine, protein, albumin, NAG, LDH, and GGT values in BALF and hematology values), we used one-way analysis of variance (ANOVA) followed by the Dunnett's multiple comparison adjustment to compare the biological responses between PM-exposed groups and a negative control group. This analysis was performed using GraphPad Prism software (version 6.07; GraphPad Software, Inc.). We modeled neutrophil and *Salmonella* responses as dependent variables to characterize their association with different fuel types and combustion phases. This analysis was performed using SAS software for Windows (version 9.4; SAS Institute Inc.). For analysis of the neutrophil count data (lung toxicity), we used negative binomial regression in the SAS GENMOD procedure; for analysis of the *Salmonella* (mutagenicity) responses, we used two-way factorial ANOVA for fixed effects in the SAS MIXED procedure. Negative binomial regression is commonly used for overdispersed count data, that is, where the variance exceeds the mean, as observed for the neutrophil count data in this study (Diggle et al. 2002; Lawless 1987). The linear or log scale for statistical tests of the *Salmonella* responses was determined by evaluating normality of model residuals (Shapiro-Wilk test in SAS UNIVARIATE). We also modeled the lung toxicity EFs and mutagenicity EFs with linear regression analysis to characterize their association with the smoke emission characteristics (i.e., EFs for PAH, OC, and PM). This analysis was performed using GraphPad Prism software (version 6.07; GraphPad Software, Inc.). We expressed the data as mean ± standard error of the mean (SEM) and assigned the statistical significance level at a probability value of $p < 0.05$.

Results

Properties of Smoldering and Flaming Combustion Emissions

Specific properties, including MCE, PM size distribution, PM concentration, and pollutant EFs, of the smoke from five biomass fuels (red oak, peat, pine needles, pine, and eucalyptus) and two combustion phases (smoldering and flaming) are listed in Table 1. The MCE values were 63–83% during the smoldering and 97–99% during the flaming phase. For all fuel types, the median diameters for the PM based on surface area–weighted particle size distributions from the smoldering phase were >1 μm (mean = 2.04 μm), whereas those from the flaming phase were <1 μm (mean = 0.59 μm).

The mean ± SEM of the EFs for CO, CO_2, and PM of the smoldering phase smoke was 233 ± 26, 1,026 ± 74, and 121 ± 16 g/kg fuel, respectively, whereas the average EFs for CO and PM of the flaming phase smoke were decreased to 22 ± 3 and 1 ± 0 g/kg fuel, respectively. In contrast, the average EF for CO_2 increased with flaming combustion to 1,795 ± 5 g/kg fuel. These data confirm that the flaming combustion conditions were more efficient, converting much of the carbon to CO_2, whereas more carbonaceous PM and CO were emitted during smoldering.

We plotted the pollutant EFs for CO, CO_2, and PM as a function of the MCE, and compared their relationships with published field and laboratory measurement data (Figure 2). Except for the EFs developed for smoldering peat, EFs were linearly dependent on the MCE of each fuel, and the linear trends were fitted to the published data obtained from aboveground fuel combustions ($r^2 = 0.97$, $r^2 = 0.82$, and $r^2 = 0.86$ of EFs for CO, CO_2, and PM, respectively) (McMeeking et al. 2009). Although the EFs of the peat smoke fell outside the linear trend lines and this deviation increased in the plot of the EF for PM vs. the MCE, they were in good

Table 1. Characteristics and emission factors (EFs) of the biomass smoke emitted from the tube furnace system.

	Red oak		Peat		Pine needles		Pine		Eucalyptus	
Variable	Smoldering	Flaming	Smoldering	Flaming	Smoldering	Flaming	Smoldering	Flaming	Smoldering	Flaming
Characteristic (unit)										
MCE (%)[a]	73 ± 1	99 ± 0	71 ± 1	97 ± 0	83 ± 0	98 ± 0	76 ± 1	98 ± 0	63 ± 1	98 ± 0
PM size (μm)[b]	1.38 (1.22)	0.65 (2.09)	2.73 (1.41)	0.89 (2.96)	2.70 (1.40)	0.54 (1.27)	2.37 (2.76)	0.40 (1.46)	1.02 (2.90)	0.48 (1.41)
CO (ppm)	793 ± 30	80 ± 6	1,385 ± 135	159 ± 10	602 ± 34	121 ± 8	766 ± 25	105 ± 14	1,201 ± 53	109 ± 10
CO_2 (ppm)	2,167 ± 111	5,597 ± 173	3,425 ± 373	5,042 ± 161	3,067 ± 192	6,576 ± 161	2,458 ± 120	6,844 ± 222	2,058 ± 67	6,407 ± 160
PM (mg/m^3)	973	8	488	15	624	18	1,050	14	1,418	10
Emission factor (g/kg fuel)[c]										
CO	231	16	288	33	154	20	198	21	292	20
CO_2	990	1,804	1,120	1,777	1,233	1,797	999	1,797	787	1,798
PM	131	1	71	1	98	1	143	1	160	1

Note: Error ranges represent standard error of the mean (SEM). PM, particulate matter.
[a]Modified combustion efficiency (MCE) = $\Delta CO_2/(\Delta CO_2 + \Delta CO)$.
[b]Surface median aerodynamic diameters calculated from surface area–weighted particles size distributions; values in brackets represent the geometric standard deviation (GSD) of the particle size distributions.
[c]Emission factor (EF)t(g/kg) = (fuel carbon fraction × mass of carbon emitted as t × molecular weight t × 1,000)/(molecular weight carbon × total mass of carbon).

agreement with the published EFs of smoldering phase smoke from ground fuel combustions (e.g., duff and organic soils) (Urbanski 2014) and peatland wildfires (Geron and Hays 2013) ($r^2 = 0.83$, $r^2 = 0.93$, and $r^2 = 0.61$ of EFs for CO, CO_2, and PM, respectively) (Figure 2).

The major chemical compounds measured in the biomass smoke condensate samples are shown in Figure 3 and Table 2; more details on ionic, inorganic, and semivolatile organic species are presented in Tables S3–S5. Depending on the sample, the smoldering combustion emitted 4–49 times more PM (or dried smoke condensate) mass than flaming combustion, but endotoxin (average of 329 and 241 endotoxin units (EU)/g for smoldering and flaming, respectively) and pH levels (average pH of 3.57 and 3.67 for smoldering and flaming, respectively) of the PM were similar on a mass basis between the two combustion conditions (Table 2). The wood smoke condensate samples (i.e., red oak, pine, and eucalyptus) averaged 56 and 60% (of PM mass) total carbon for smoldering and flaming, respectively, whereas the nonwood smoke condensate samples (i.e., peat and pine needles) had a slightly higher percentage of total carbon for smoldering (average of 76% of PM mass) but lower for flaming (average of 43% of PM mass) combustion.

Ionic species (mostly Cl^-, SO_4^{2-}, and PO_4^{3-}) accounted for <1% and 15.6% of PM in the smoke condensate samples from smoldering and flaming combustion, respectively (Figure 3 and Table S3). Similarly, inorganic species (mostly Ca, Na, S, and metals) of the smoke condensate collected from smoldering contributed to an average of 1% of PM mass, and inorganic species from flaming samples contributed to an average of 6% of PM mass (Figure 3 and Table S4). The peat flaming smoke condensate contained the highest level of heavy metals (e.g., Cr, Cu, Fe, Ni, Pb, Sb, and Zn), accounting for up to 1.2% of PM mass. For both the flaming and smoldering conditions, the wood smoke condensate was enriched in levoglucosan (up to 12.6% of PM mass) compared with the nonwood smoke condensate (up to 4.1% of PM mass), whereas total methoxyphenols made up a higher percentage of the PM mass in smoldering smoke condensate (up to 6.5% of PM mass) than in flaming smoke condensate (up to 1.6% of PM mass) for all fuel types (Figure 3 and Table S5). Levels of *n*-alkanes and PAHs in the smoke condensate samples also varied on the basis of combustion conditions and fuel type (Figure 3 and Table S5). *N*-alkanes contributed the most to PM mass (0.9%) in the smoke condensate sample from smoldering peat, whereas the highest contributions of PAHs (0.5%) were found in the smoke condensate samples following flaming combustion of the pine and eucalyptus, respectively. Overall, the toxic heavy metals and PAHs were relatively enriched in the flaming smoke condensate samples; more specifically, nonwood smoke condensate comprised up to 12,247 μg/g of heavy metals (Table S4) and wood smoke condensate contained up to 5,138 μg/g of PAHs (Table S5).

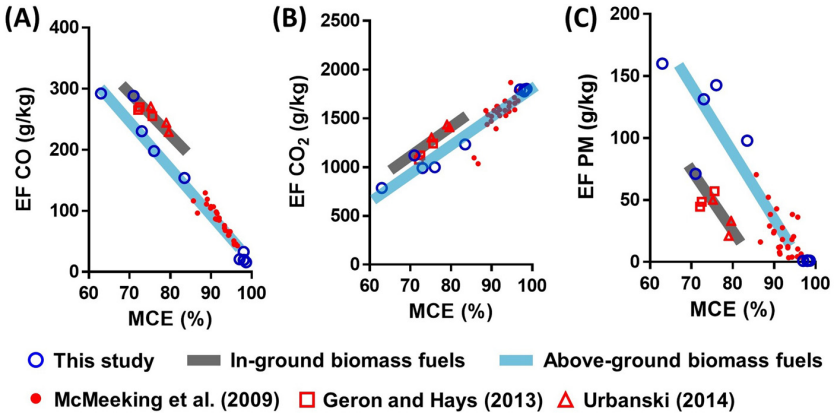

Figure 2. Comparison of emission factors (EFs) estimated from the tube furnace system in this study with published EFs from various fuel combustion. (*A*), (*B*), and (*C*) pollutant EFs for CO, CO_2, and PM vs. modified combustion efficiency (MCE). Open circles are pollutant EFs estimated in this study. Solid dots represent pollutant EFs from the open combustion of various plant fuels (McMeeking et al. 2009). Open squares are pollutant EFs from peatland wildfires (Geron and Hays 2013). Open triangles are pollutant EFs from the smoldering combustion of ground biomass fuels, such as duff and organic soils (Urbanski 2014). McMeeking et al. (2009) Geron and Hays (2013) Urbanski (2014).

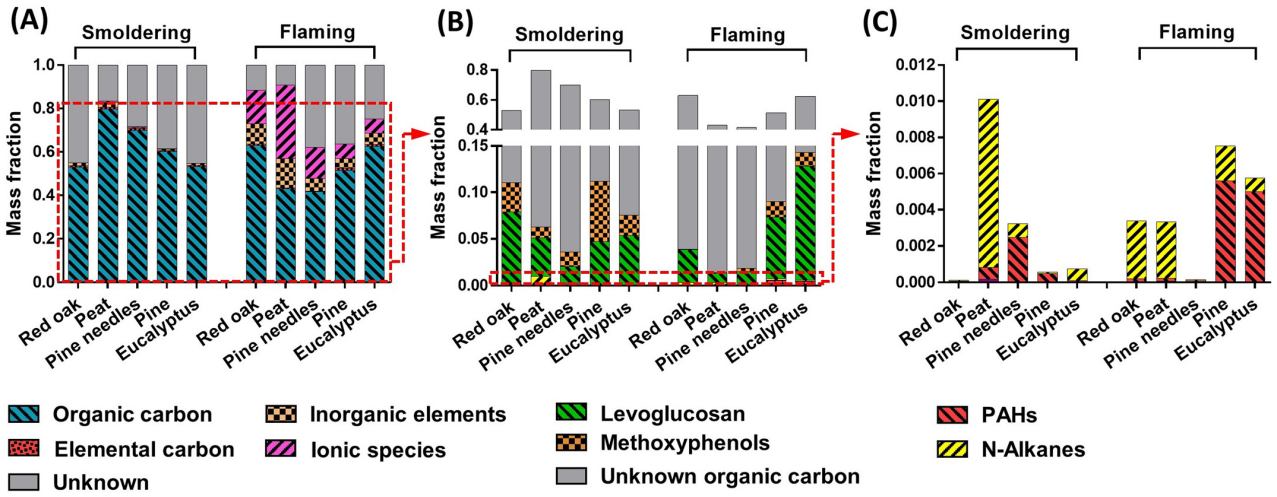

Figure 3. Chemical components in the biomass smoke condensate collected from different fuel types and combustion phases. (*A*) mass fraction of major chemical compounds, (*B*) organic carbon species [the dashed line, superimposed on (*A*)], and (*C*) semivolatile compounds [the dashed line, superimposed on (*B*)] in the biomass smoke condensate from smoldering and flaming combustion.

Lung Toxicity Potencies of the Biomass Smoke Particulate Matter

After exposing mice to an equal mass (100 μg) of the PM samples, we analyzed the BALF for markers of lung toxicity, including markers of lung inflammation (neutrophils and macrophages), pro-inflammatory cytokines (IL-6, TNF-α, and MIP-2), and markers of cellular injury (protein, albumin, NAG, LDH, and GGT) (Figures 4 and 5 and Tables S6–S8). Neutrophil counts (per mass of PM) were highest in mice exposed to the flaming peat and eucalyptus PM at 4 h (Figure 4 and Table S6). The average proportion of neutrophils relative to the total number of BALF cells was 22% following both exposures, compared with only 2% in controls at 4 h. At 24 h, BALF neutrophil counts in the mice exposed to the flaming peat and eucalyptus PM were higher than (or similar to) counts in exposed mice evaluated at 4 h, and neutrophils accounted for 44 and 21% of total lavageable cells on average, respectively, compared with 2% in controls (Figure 4). The flaming peat and eucalyptus PM were associated with significantly higher neutrophil recruitment than other fuel PM samples at 24 h postexposure. The total numbers of macrophages were similar for each PM sample in mice evaluated 4 and 24 h postexposure (Figure 5A and Table S6).

Further analysis of pro-inflammatory cytokines in BALF revealed that the concentrations of IL-6, TNF-α, and MIP-2 were significantly elevated in mice exposed only to the flaming peat PM at 4 h, compared with control mice evaluated at the same time point (Figures 5B–5D and Table S7). Although the number of neutrophils was higher in mice evaluated at 24 h than in mice evaluated at 4 h postexposure to the flaming peat, the concentrations of TNF-α and MIP-2 were lower at 24 h, and not significantly different from saline controls. The concentration of IL-6 was also lower in mice evaluated at 24 h than in mice evaluated at 4 h postexposure, but it remained significantly higher than in saline controls. For mice exposed to the flaming peat PM, the concentrations of protein, albumin, NAG, and LDH, but not GGT, in BALF were significantly higher than saline controls evaluated at 24 h, but were not significantly different from saline controls evaluated at 4 h postexposure (Figures 5E–5I and Table S8). Thus, for most exposures, the lung toxicity potencies of the flaming PM were higher than those of the smoldering PM for neutrophils, IL-6, TNF-α, MIP-2, protein, albumin, NAG, and LDH. Mice exposed to the peat PM showed the greatest differences from controls. The statistical analysis also showed that the lung toxicity potencies were significantly associated with different fuel types and combustion phases at 24 h ($p < 0.01$) but not 4 h ($p = 0.17$) postexposure (Table S9).

Hematology analysis showed that, compared with controls, mice exposed to the smoldering eucalyptus PM had significantly lower white blood cell counts, and mice exposed to the smoldering pine PM or eucalyptus PM had significantly lower lymphocyte counts at 4 h postexposure. Mice exposed to the flaming

Table 2. Chemical compositions of the biomass smoke condensate collected from the multistage cryotrap system.

	Red oak		Peat		Pine needles		Pine		Eucalyptus	
Component (unit)	Smoldering	Flaming	Smoldering	Flaming	Smoldering	Flaming	Smoldering	Flaming	Smoldering	Flaming
PM mass (mg)	488	10	117	28	449	27	789	25	955	21
EOM[a] (% of PM mass)	50	47	73	38	62	35	60	43	52	40
pH	3.37	3.78	4.26	3.17	3.85	3.51	3.08	3.92	3.30	3.98
Endotoxin (EU/g)	449	249	270	161	343	232	321	256	262	306
Ion (μg/g)	1,285	155,982	7,148	339,077	3,379	143,418	949	66,625	330	65,259
Ion (% of PM mass)	0	16	1	34	0	14	0	7	0	7
Organic carbon (μg/g)	529,508	629,242	797,863	430,830	699,443	416,413	601,394	513,893	532,723	624,508
Elemental carbon (μg/g)	7,787	8,160	8,120	3,968	7,795	2,774	5,070	12,509	5,026	10,081
Total carbon (% of PM mass)	54	64	81	43	71	42	61	53	54	63
Inorganic element (μg/g)	11,081	91,367	20,559	131,583	5,505	56,962	5,920	42,788	8,045	51,879
Inorganic element (% of PM mass)	1	9	2	13	1	6	1	4	1	5

[a]Extractable organic matter (EOM) represents nonvolatile organic material present in the biomass smoke particulate matter (PM) that was extracted by dichloromethane.

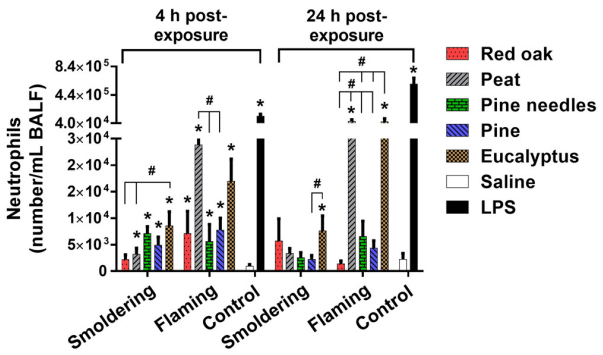

Figure 4. Comparative lung toxicity potencies of the biomass smoke particulate matter (PM) emitted from different fuel types and combustion phases. Lung toxicity potencies assessed from the number of neutrophils in bronchoalveolar lavage fluid (BALF) based on the equal PM mass. Mice were exposed to the PM (100 μg) by oropharyngeal aspiration, and BALF was obtained at 4 and 24 h postexposure. Data are mean ± standard error of the mean (SEM) and obtained from six mice for each group. $^{*}p < 0.05$ compared with the saline-exposed (a negative control) group from the same time point. $^{\#}p < 0.05$ compared with the different fuel group from the same combustion phase. Mice exposed to 2 μg of lipopolysaccharide (LPS) served as a positive control. The statistical tests were performed using negative binomial regression in the SAS GENMOD (version 9.4; SAS Institute Inc.) procedure.

peat, pine needles, pine, and eucalyptus PM had significantly lower white blood cell and lymphocyte counts at 4 h postexposure. At 24 h postexposure, white blood cell and lymphocyte counts were not significantly different from controls (Figure S4 and Table S10). Other hematology values (e.g., red blood cell counts, hemoglobin, and hematocrit) were not significantly different between exposed mice and controls at 4 h or 24 h postexposure.

Lung Toxicity Emission Factors

In order to estimate the lung toxicity EFs, which is toxicity/mass of fuel burned, we selected only the neutrophil numbers that showed a noticeable effect in all the biomass smoke PM exposures in this study. We adjusted the neutrophil number per PM mass (referred to lung toxicity potency) for the EFs for PM (g PM/kg fuel, Table 1) and then expressed it as neutrophils/kg fuel (Figure 6 and Table S11). In contrast to the lung toxicity potencies (neutrophils/mass of PM) in which flaming conditions produced the highest values, the lung toxicity EFs (neutrophils/mass of fuel burned) of the smoldering PM were greater than those of the flaming PM at both 4 and 24 h postexposure (Figure 6 and Table S11). Under smoldering conditions, the eucalyptus PM, which had the highest EF in this study, also had the highest lung toxicity EF (i.e., the largest number of neutrophils/kg fuel) of all of the PM tested at 4 h (significantly higher than for red oak and peat) and 24 h (significantly higher for peat, pine needles, and pine), indicating that EF and the related PM exposure potencies (neutrophil counts) strongly influence the degree of lung toxicity from biomass smoke emissions. The statistical analysis showed that the lung toxicity EFs were significantly associated with different fuel types and combustion phases at 4 h ($p < 0.03$) and 24 h ($p < 0.01$) postexposure (Table S9). The lung toxicity EFs were also highly associated with emission characteristics of OC ($r^2 = 0.70$; $p < 0.01$) and PM ($r^2 = 0.74$; $p < 0.01$) in the biomass smoke (Figure S5).

Mutagenic Potencies of the Biomass Smoke Extractable Organic Material and Particulate Matter

The mutagenic potencies of the EOM (rev/μg EOM) and the PM (rev/μg PM) are shown in Figures 7A and 7B and summarized in Table S12. Note that only two of the extracts (smoldering peat and pine needles in TA100 −S9) gave dose–response curves with p-values >0.05, which we would consider to be nonmutagenic (Figures S3G and S3H and Table S12). All the rest were mutagenic. Overall, the highest mutagenic potencies of the PM were those from flaming peat and pine, and their potencies were also significantly higher than those of the majority of other fuel PM in both strains +/−S9. Similar to the lung toxicity potencies (neutrophils/mass of PM), the mutagenic potencies of the EOM and PM (i.e., on a mass basis) were far higher under flaming phases than from smoldering phases.

The mutagenic potencies of the EOM and PM for each biomass fuel in each strain was similar with and without metabolic activation (+S9 and −S9, respectively), consistent with a mix of direct- and indirect-acting mutagenic activity. However, the EOM and PM from each biomass fuel was typically more mutagenic in TA100 than in TA98, consistent with mutagenicity due to base-substitution (vs. frameshift) mutations (Figures 7A and 7B and Table S12). All the mutagenic potencies of the PM in this study were significantly associated with different fuel types and combustion phases ($p < 0.01$) (Table S9).

Mutagenicity Emission Factors

In contrast to the mutagenic potencies of the EOM and PM, for which flaming conditions were associated with the highest values (rev/mass of EOM or PM), and similar to the lung toxicity EFs, smoldering conditions were associated with the highest mutagenicity EFs (rev/mass of fuel burned) in nearly all strain/S9 combinations expressed as either rev/kg fuel or rev/MJ_{th}; the only exception was peat in TA100 −S9 (Figures 7C and 7D and Table S12). Pine smoke PM was associated with the highest and second-highest mutagenicity EFs (rev/kg fuel) in TA100 and TA98, respectively, under flaming conditions (statistically significant only in both strains with S9), whereas there was no statistically significant pattern of response with the smoldering PM samples. Overall, the mutagenicity EFs in TA98 + S9 were only significantly associated with different fuel types and combustion phases (Table S9). All smoldered fuels had the highest mutagenicity EFs in TA100 + S9, consistent with a dominant role of PAHs in these samples (taking into account EFs). In contrast, under flaming conditions, all PM samples had the highest mutagenicity EFs in TA100 −S9, indicating that base-substitution mutagens that were not PAHs accounted for much of these effects. The mutagenicity EFs for PM produced under flaming conditions were similar with and without S9, whereas the mutagenicity EFs of the smoldering samples were generally higher in strains supplemented with S9 than those without S9. The mutagenicity EFs were also significantly associated with emission characteristics of OC, PAHs, and PM in the biomass smoke: mutagenicity EFs in TA100 +S9 vs. EFs for OC ($r^2 = 0.90$; $p < 0.01$) and PM ($r^2 = 0.80$; $p < 0.01$); mutagenicity EFs in TA98 + S9 vs. EFs for OC ($r^2 = 0.61$; $p < 0.01$), PAHs ($r^2 = 0.53$; $p < 0.02$), and PM ($r^2 = 0.44$; $p < 0.04$); mutagenicity EFs in TA98 −S9 vs. EFs for OC ($r^2 = 0.59$; $p < 0.01$) and PM ($r^2 = 0.59$; $p < 0.01$) (Figure S6). Furthermore, the mutagenic responses in TA100 + S9 and TA98 −S9 were only associated with emission characteristics of OC and PM, and these factors were also significantly correlated with the lung toxicity EFs ($r^2 = 0.69$; $p < 0.01$ in TA100 +S9 and $r^2 = 0.42$; $p < 0.05$ in TA98 −S9) (Figure S7).

We determined mutagenicity EFs based on fuel energy used (rev/MJ_{th}) and compared these with the published mutagenicity EFs for various combustion emissions obtained from TA98 + S9 (Figure 8). The mutagenicity of the flaming emissions (1.1×10^5 rev/MJ_{th}; average of the five fuel-burning emissions) was relatively similar

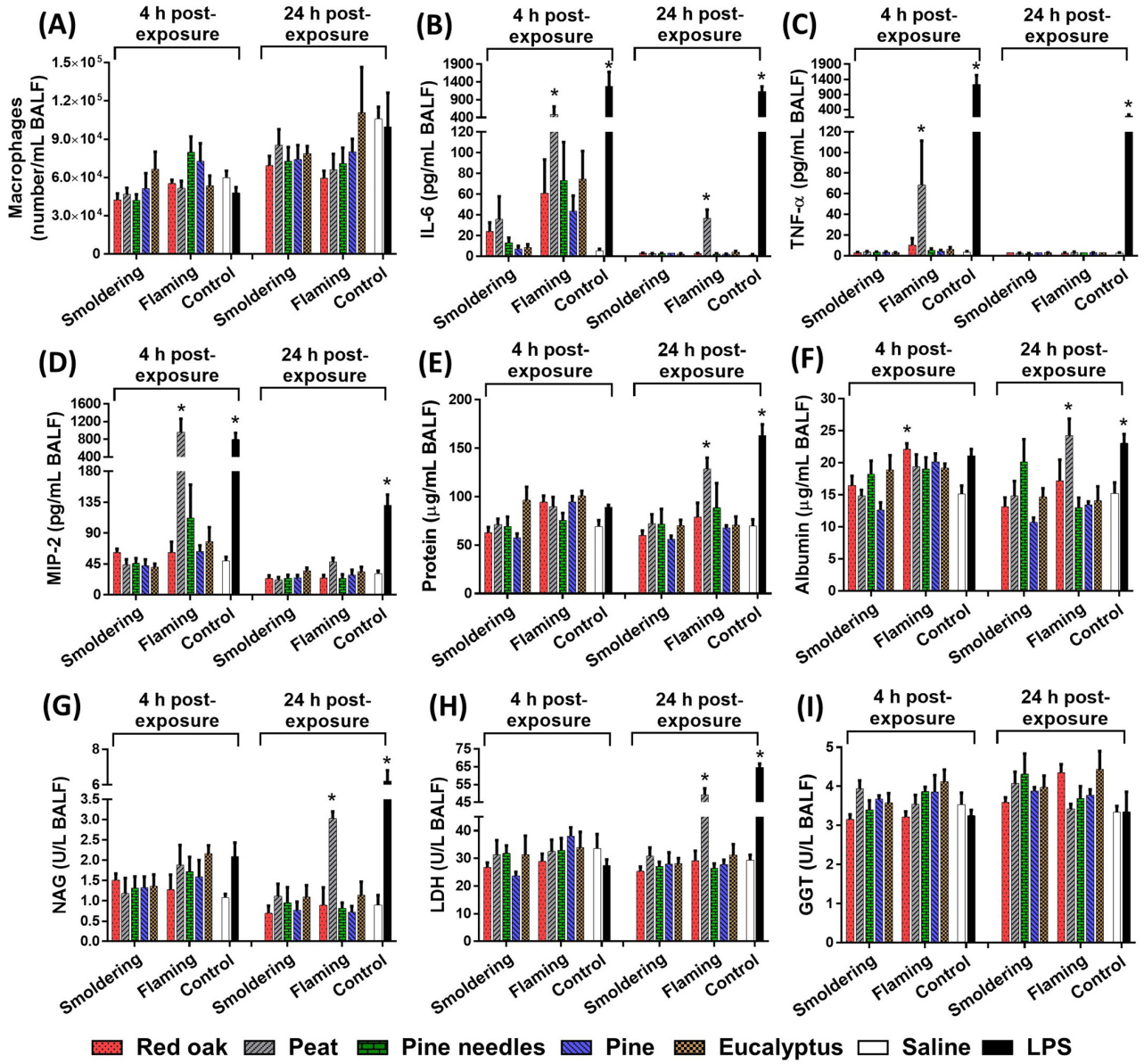

Figure 5. Comparative lung responses in mice exposed to the biomass smoke particulate matter (PM) emitted from different fuel types and combustion phases. (*A*) number of macrophages, concentrations of (*B*) interleukin (IL)-6, (C) tumor necrosis factor-α (TNF-α), (*D*) macrophage inhibitory protein-2 (MIP-2), (*E*) protein, (*F*) albumin, (*G*) *N*-acetyl-β-D-glucoaminidase (NAG), (*H*) lactate dehydrogenase (LDH), and (*I*) γ-glutamyl transferase (GGT) in bronchoalveolar lavage fluid (BALF) based on the equal PM mass. Mice were exposed to the PM (100 μg) by oropharyngeal aspiration, and BALF was obtained at 4 and 24 h postexposure. Data are mean ± standard error of the mean (SEM) and obtained from six mice for each group. $^*p < 0.05$ compared with the saline-exposed (a negative control) group from the same time point. Mice exposed to 2 μg of lipopolysaccharide (LPS) served as a positive control. The statistical test was performed using one-way analysis of variance (ANOVA) followed by the Dunnett's multiple comparisons.

to that of woodburning cookstove emissions (1.3×10^5 rev/MJ_{th}; average of force draft stove, natural draft stove, and three-stone fire emissions; (Mutlu et al. 2016)). Although the smoldering emissions (6.6×10^5 rev/MJ_{th}; average of the five fuel burning emissions) were less mutagenic than the emission from the open burning of tire [22.7×10^5 rev/MJ_{th} (DeMarini et al. 1994)], they were more mutagenic than those of diesel exhaust [0.4×10^5 rev/MJ_{th} (Mutlu et al. 2015)], municipal waste combustion [0.4×10^5 rev/MJ_{th}; (Watts et al. 1992)], and the open burning of agricultural plastic [2.5×10^5 rev/MJ_{th} (Linak et al. 1989)].

Discussion

Pollutant Emission Factors by Biomass Fuel Types and Combustion Phases

Our system produced PM from well-controlled smoldering and flaming combustion that was within the respirable size range (<2.5 μm in diameter), consistent with other laboratory and field studies (McMeeking et al. 2009; Reisen et al. 2015; Ward and Hardy 1991). Moreover, the pollutant EFs for major emission constituents (CO, CO_2, and PM) agreed well with those from both field and laboratory measurements (Geron and Hays 2013;

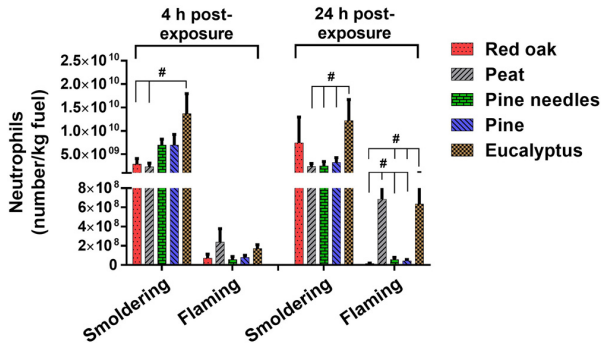

Figure 6. Comparative lung toxicity emission factors (EFs) of the biomass smoke particulate matter (PM) emitted from different fuel types and combustion phases. Lung toxicity emission factors (EFs) were calculated based on the emitted PM mass per mass of fuel burned. The lung toxicity potency values (neutrophils/μg PM) directly obtained from the bronchoalveolar lavage fluid (BALF) analysis (Figure 4) were converted to lung toxicity EFs (neutrophils/kg fuel) by multiplying them by the EFs for PM (g PM/kg fuel, Table 1). Data are mean ± standard error of the mean (SEM) and obtained from six mice for each group. $^{\#}p < 0.05$ compared with the different fuel group from the same combustion phase. The statistical tests were performed using negative binomial regression in the SAS GENMOD (version 9.4; SAS Institute Inc.).

McMeeking et al. 2009; Urbanski 2014). When we combined our pollutant EF data with those of others, we found high correlations between the EFs for CO, CO_2, and PM vs. the percent MCE. We also found that the correlations were distinguished by specific fuel types (e.g., above- and in-ground fuels). Observing this difference from uncontrolled combustions (e.g., open burning) is challenging because pollutant EFs are obtained from limited combustion phases. However, since our system produced pollutant EFs of various fuel types from a wide range of well-controlled combustion phases (60% < MCE <99%), we were able to identify strong correlations between the EFs for aboveground fuels (woods and needles) and those at in-ground level (peat or partly decayed organic matter on the forest floor called duff); note that the y-intercepts of the regression lines for the pollutant EFs as a function of MCE were quite different between the two fuel types (Figure 2). This suggests that there are distinct differences in the emission characteristics from biomass fuels from in- vs. aboveground.

In the natural environment (uncontrolled combustion), flaming and smoldering phases often occur simultaneously and are difficult to resolve (Urbanski 2014), while our system can readily distinguish between these conditions and explain the relative contributions of the different combustion phases in field measurements. For example, pollutant EFs for aboveground emissions during uncontrolled combustion were associated primarily with a flaming phase mixed with intermittent smoldering, resulting in EFs that were weighed more toward the "pure" flaming EF [see linear regression results in Figure 2; the published EFs for aboveground emissions were mostly obtained during flaming (MCE >90%)]. However, pollutant EFs from in-ground biomass combustions (peat and organic soils) were associated primarily with the pure smoldering EF [Figure 2; the published EFs for in-ground emissions were obtained mostly during smoldering (MCE >80%)]. This is consistent with a previous report (Kasischke and Bruhwiler 2002) that assumed that 80% of the emissions from aboveground biomass were produced by flaming and 20% by smoldering, whereas 80% of emissions from in-ground biomass (or 100% of peat) derived from smoldering and 20% by flaming. Overall, comparing our data to literature values suggests that pollutant EFs from controlled or uncontrolled combustion of biomass are highly dependent on the distribution of the biomass fuels vertically (aboveground or in-ground) rather than horizontally (i.e., the genus or family of wood or biomass).

Chemical Composition of the Biomass Smoke Condensate Relative to Fuel Types and Combustion Phases

The cryotrap sampling system used collects and composites chemical compounds across a wide volatility range. Thus, the cryotrap samples are expected to be quite different from those collected using traditional filter-based PM and gas-phase sampling methods, which typically attempt to separate compounds by chemical and physical state. The use of the cryotrap allowed us to collect volatile and semivolatile organic compound emissions in a single sample, eliminating the well-known artifacts and interferences associated with classical sample collection (McDow and Huntzicker 1990). It also allowed us to more accurately predict specific chemical components associated with exposures to biomass smoke. OC accounted for approximately 58% of the PM mass on average. This value is similar to observations made by Kim et al. (2014b), who found that PM samples from the peat bog wildfire were comprised of 53.4% organic matter. Similarly, Reid et al. (2005) reviewed the properties of biomass-burning particles and found that the percentage of fresh smoke particles to which OC contributed varied from 13.6–67%, depending on the biomass type and combustion phase. The OC range, however, was 42–80% from nonwood (peat and pine needles) fuels, including smoldering and flaming conditions, which was wider than the 53–63% OC seen for the wood species (red oak, pine, and eucalyptus) burns (Figure 3). This variability in carbon composition is possibly explained by the fact that nonwood fuels vary more than wood fuels in their concentration of wax, cellulose, lignin, and elemental components (Hays et al. 2002).

The concentration of levoglucosan, which is a pyrolysis product of cellulose, in the smoke condensate was generally higher for the wood fuels (red oak, pine, eucalyptus) than the nonwood plant species (peat and pine needles) (Table S5). Specifically, the flaming pine and eucalyptus produced the highest levoglucosan concentrations, whereas the red oak and the two nonwood fuels showed higher concentrations during smoldering (Table S5). These findings are consistent with a larger general trend showing high levoglucosan concentrations in PM from woodburning (George et al. 2016; Hays et al. 2002; Schauer et al. 2001). Likewise, the fraction of methoxyphenols, which are lignin pyrolysis products, in the woody biomass smoke condensate was generally higher (Table S5). However, unlike levoglucosan, methoxyphenol concentrations were higher in the smoldering smoke condensate. This is consistent with the finding that methoxyphenols (wood smoke tracer compounds) are formed mainly during incomplete combustion at lower temperatures (Kjällstrand and Olsson 2004).

Previous studies show that PAH concentrations in wood smoke PM increase with combustion temperature (Bølling et al. 2012; McDonald et al. 2000; McMahon and Tsoukalas 1978; Reid et al. 2005). Presently, the PAH concentrations in the wood smoke condensate (red oak, pine, eucalyptus) were higher for flaming conditions; however, for the nonwood fuels (peat and pine needles), PAHs were higher for smoldering conditions (Table S5). Furthermore, higher combustion temperatures during flaming also increased the amount of ionic and inorganic species in the smoke condensate from flaming compared to smoldering conditions (Figure 3 and Tables S3 and S4) in agreement with a report showing that trace element concentrations for hot burning woods were two orders of magnitude higher than those for cool burning woods (Rau 1989). Similarly, Frey et al. (2009) reported that wood burning at high temperatures was associated with high emissions of ions and trace elements (20 and 1% of EF for PM,

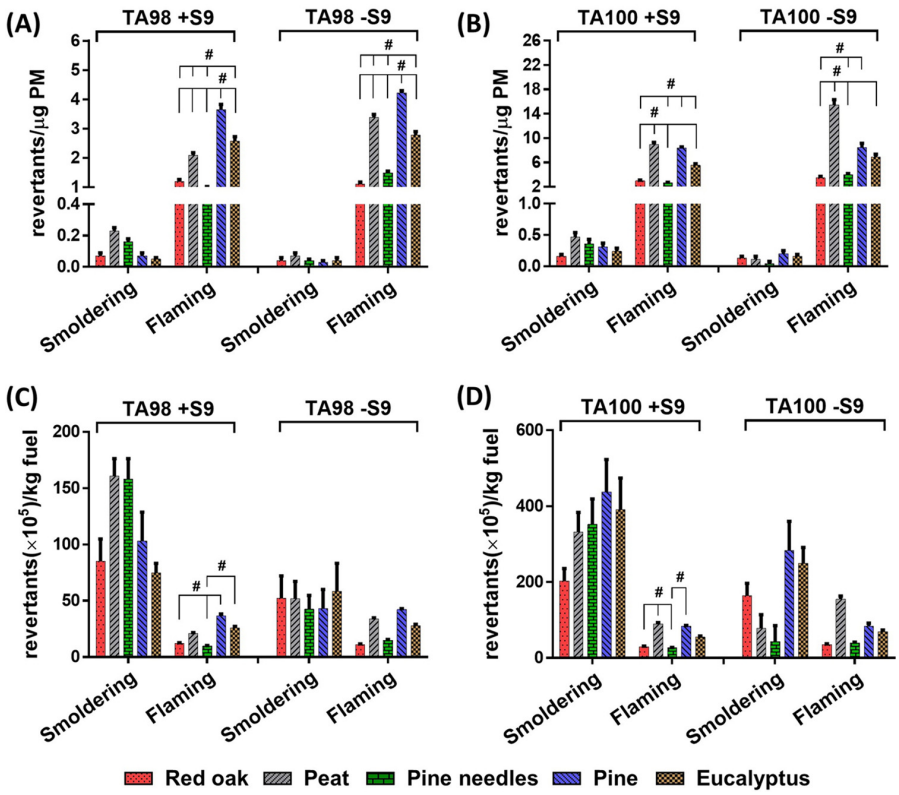

Figure 7. Comparative mutagenicity of the biomass smoke particulate matter (PM) emitted from different fuel types and combustion phases. (*A*) and (*B*) mutagenic potencies in strains TA98 + / − S9 and TA100 + / − S9 calculated based on the equal PM mass, and (*C*) and (*D*) mutagenicity emission factors (EFs) in strains TA98 + / − S9 and TA100 + / − S9 calculated based on the emitted PM mass per mass of fuel burned. Mutagenic potencies of the extractable organic material (EOM) were calculated from the slope of the linear portion of the dose–response curve created by the average of the primary data (rev/plate) from four independent mutagenicity experiments (Figures S2 and S3). The mutagenic potencies of the EOM were then multiplied by the percent EOM to give mutagenic potencies of the PM (rev/μg PM). These values were then converted to mutagenicity EFs (rev/kg fuel) by multiplying them by the EFs for PM (Table 1). Data are mean ± standard error of the mean (SEM) and obtained from four independent mutagenicity experiments. $^{\#}p < 0.05$ compared with the different fuel group from the same combustion phase. The statistical tests were performed using two-way factorial analysis of variance (ANOVA) in the SAS MIXED (version 9.4; SAS Institute Inc.) procedure.

respectively) compared to low temperature combustion (2 and 0.3% of EF for PM, respectively). Collectively, our findings show that the chemical composition of biomass smoke varies substantially depending on combustion conditions and fuel types, especially between wood or nonwood biomass fuels.

Lung Toxicity of the Biomass Smoke Particulate Matter and Role of Fuel Types and Combustion Phases

On an equal mass basis, the flaming PM samples had higher lung toxicity (neutrophil counts) than the smoldering samples, with peat and eucalyptus being the most potent at both the 4- and 24-h time points (Figure 4). Lung injury and inflammation can be triggered by a number of different signals from both inorganic and organic moieties that cause oxidative stress in one form or another (Bølling et al. 2009; Bølling et al. 2012). The flaming peat sample had the highest levels of heavy metals (Cr, Cu, Fe, Mn, Ni, Pb, Sb, and Zn) and sulfate, many of which have been implicated in lung injury and inflammation through increased redox cycling (Fang et al. 2017; Gavett et al. 1997; Happo et al. 2013; Reiss et al. 2007; Veranth et al. 2006). On the other hand, the flaming eucalyptus had the highest levels of certain PAHs, such as phenanthrene, anthracene, and fluoranthene; the capacity for PAHs to induce oxidative stress through quinone formation is well documented (Bølling et al. 2009). The acute toxicity of eucalyptus smoke has also been linked specifically to phenolic compounds such as phenol and o-Cresol (Pimenta et al. 2000).

Although our data clearly showed stronger associations of flaming samples with toxicity markers, other studies have reported that PM from low-temperature combustion was more potent at inducing cellular damage and inflammatory cytokine release than that from high-temperature combustion (Bølling et al. 2012; Jalava et al. 2010). In some cases, the combustion conditions were less precisely controlled, and smoldering or flaming samples were taken at various periods of a complex burn that possibly reflected both combustion phases (Bølling et al. 2012). In one report, however, the results actually reflected effects based on EF and, like our study, found that flaming samples were more toxic on a mass basis; however, when adjusted for EF, the smoldering sample was more potent (Jalava et al. 2010). Thus, the smoldering PM samples from all the fuels had much higher lung toxicity when expressed as EFs, which consider both the potency of the sample as well as the amount of PM produced from a specific mass of fuel burned (Figure 6).

In addition to the combustion effect on the lung toxicity (potency and EF), the statistical analysis further demonstrated that the lung toxicity (neutrophil counts; potency and EF) from different combustion phases was also significantly associated with different fuel types (Table S9). For example, the eucalyptus or peat PM from smoldering or flaming condition had the highest lung

toxicity potency and EF at 24 h postexposure, and they were significantly higher as compared to different combustion samples (Figures 4 and 6 and Tables S6 and S11). Although the lung toxicity of the flaming eucalyptus PM was associated with high levels of PAHs, the correlation analysis showed that the lung toxicity EFs correlated better with EFs for PM and OC than with PAHs, to which it correlated poorly (Figure S5). These results indicate that the lung toxicity of the smoldering eucalyptus PM was more likely to be associated with total PM emissions than just PAHs alone. Similarly, Bølling et al. (2012) reported that the toxicity of wood smoke particles was highly associated with the organic matter, but negatively associated with the total PAH content.

Mutagenicity of the Biomass Smoke Particulate Matter and Role of Fuel Types and Combustion Phases

Like the lung toxicity data, the mutagenic potencies of the PM expressed on an equal mass basis were highest for flaming samples, with pine, peat, and eucalyptus having the highest values. Of the flaming samples, the increase in the mutagenic potency of peat without S9 was higher than other fuel types, suggesting that unlike wood smoke, the organic components from peat smoke were primarily direct-acting mutagens in the *Salmonella* assay. The higher mutagenic potencies of the PM samples in TA100 vs. TA98 were consistent with findings from other studies of wood smoke (Asita et al. 1991; Mutlu et al. 2016), suggesting that the base-substitution mutagenic activity was generally more prominent than frameshift activity for these PM samples. Mutagenicity from different combustion phases was also significantly associated with different fuel types, at least for the responses expressed per unit of PM (Table S9), suggesting that the mutagenic potency of various biomass fuels in any one strain depends on the combustion phase. However, unlike the lung toxicity EF data, the mutagenicity EFs were not different between different fuel types or combustion phases except for the strain TA98 + S9 condition, suggesting that fuel types (or combustion phases) may not play a critical role in the degree of biomass smoke exposure and subsequent mutagenicity. This also supports the concept that despite having lower mutagenicity per mass, the smoldering PM produced up to 10 times more mutagenicity, resulting in an overall higher exposure and greater potential for health effects.

To understand relationships between specific chemical classes and mutagenicity EFs, we performed correlation analyses for key results. Significant correlations between mutagenicity EFs and pollutant EFs in this study were for TA100 + S9 vs. OC and TA98 + S9 vs. OC or PAHs, indicating that PAHs played an important role in the mutagenicity EFs of the fuels (Figure S6). Nitroarenes also showed positive associations with effects, as indicated by the correlation between the mutagenicity EF in TA98–S9 vs. OC or PM (Figure S6). Such results are consistent with those from other studies of biomass smoke (Asita et al. 1991; Mutlu et al. 2016). It should be pointed out, however, that like other studies (McDonald et al. 2006; Reed et al. 2006), the sum of the mass of the PAHs analyzed accounted for $<1\%$ of the mass of the PM extract (Table S5), and many other chemical classes besides PAHs and nitroarenes likely play a role in the toxicity and mutagenicity of the biomass PMs evaluated here. Interestingly, mutagenicity EFs in TA100 + S9, which detects base-substitution-inducing PAHs, correlated well with lung toxicity EFs (Figure S7), suggesting that some of the same chemical components (or components that track with these) are inducing both mutagenicity and lung toxicity. Specifically, the chemical components from the smoldering, but not the flaming, smoke emissions appeared to be responsible for both biological effects.

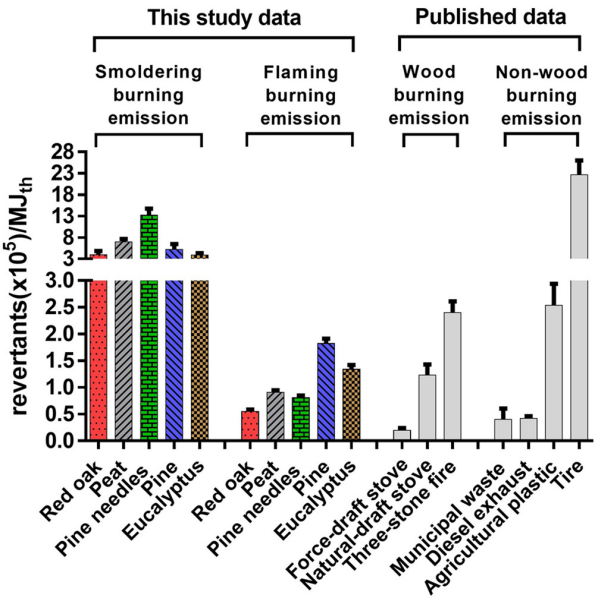

Figure 8. Comparison of mutagenicity emission factors (EFs) of various combustion emissions in strain TA98 + S9. The mutagenicity EFs (rev/kg fuel; Figure 7C and Table S12) were converted to rev/MJ_{th} using the values for the heat energy of each fuel (MJ_{th}/kg fuel). The mutagenicity EFs for emissions from cookstoves burning red oak were 0.2, 1.2, and 2.4×10^5 rev/MJ_{th} for the force-draft stove, natural-draft stove, and three-stone fire, respectively; data from Mutlu et al. (2016). The mutagenicity EFs for nonwood burning emissions were 0.4, 0.4, 2.5, and 22.7×10^5 rev/MJ_{th} for the municipal waste, diesel exhaust, agricultural plastic, and tire, respectively; data from DeMarini et al. (1994); Linak et al. (1989); Mutlu et al. (2015); Watts et al. (1992). All data are presented as mean ± standard error of the mean (SEM).

Comparison of Mutagenicity Emission Factors from a Variety of Combustion Emissions

Finally, after converting the results to megajoule$_{thermal}$(MJ_{th}), we compared the mutagenicity EFs in TA98 + S9 to those of a variety of other combustion emissions (Figure 8), and found that the smoldering values were substantially higher than those of nearly all other combustion emissions. For example, the smoldering mutagenicity EF was found to be approximately 5, and 16 times higher than those of oak combusted in cookstoves (Mutlu et al. 2016), and of municipal waste combustion (Watts et al. 1992) or diesel exhaust (Mutlu et al. 2015), respectively. Thus, in this context, the smoldering emissions from wildland fires are highly mutagenic and support the notion that smoldering wood smoke is genotoxic and ultimately carcinogenic in humans (IARC 2010; Kato et al. 2004; Long et al. 2014).

Conclusions

We have developed a novel combustion and smoke collection system that can be used for chemical/toxicological analyses of biomass smoke under precise combustion conditions and whose data can be used to understand the potential health effects from exposures to various biomass combustions. The lung toxicity and mutagenic potencies of biomass smoke emissions on a mass basis were greater from flaming than smoldering phases for a variety of biomass fuels; however, the EFs for these toxicological endpoints were greater for smoldering than flaming conditions. Although regulatory decisions are more relevant to the potency values (i.e., PM mass), the EFs reflect real-world exposures and should be considered in assessing the health effects of wildland fires.

Both the chemical and toxicological data illustrate the distinctive contribution of vertical vs. horizontal or wood vs. nonwood components of wildlands to the adverse biological effects of wildland fires. The greatest lung toxicity (neutrophils/kg fuel) was for eucalyptus, which is representative of chaparral-type wood, whereas the greatest mutagenicity (rev/kg fuel) was for pine, which is broadly distributed across the United States. Overall, the results suggest that emissions from fires in regions rich in those type of fuels may induce greater health effects than those from fires of similar magnitude with other types of biomass.

It should be noted that further work on *a*) more complete chemical speciation of the biomass smoke (gas and PM phase), *b*) characterization of physiologic consequences of the smoke inhalation, and *c*) disparities in health outcomes from different exposure situations (e.g., occupational, incidental, and accidental exposure) is needed to extrapolate our findings to real-world wildland fires. However, the results provide insight into the composition of forests (wood and nonwood) and the combustion conditions (smoldering and flaming) that result in emissions with decidedly distinct levels of two different types of adverse biological effects (lung toxicity and mutagenicity). Such data should provide guidance on the protection from inhalation to wildland fire smoke for firefighter and public health responses to wildland fires, whose scale and severity are increasing worldwide.

Acknowledgments

The authors would like to thank C. Copeland, L. Copeland, W. Williams, D. Andrews, and J. Richards for technical assistance in toxicological analyses; J. McGee and K. Kovalcik for technical assistance in PM chemical analyses; S. Urbanski (Missoula Fire Sciences Laboratory) for kindly providing biomass samples; and S. Gavett and W. P. Linak for their careful review of this manuscript.

This study was supported by the Joint Fire Science Program Project (14-1-04-16) and was performed while Y.H.K. held a National Research Council Senior Research Associateship Award at the U.S. EPA. Additional support was provided by the intramural research program of the Office of Research and Development, U.S. EPA, Research Triangle Park, North Carolina.

The research described in this manuscript has been reviewed by the National Health and Environmental Effects Research Laboratory, U.S. EPA, and approved for publication. Approval does not signify that contents necessarily reflect the views and policies of the agency, nor does the mention of trade names or commercial products constitute endorsement or recommendation for use.

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Pyroaerobiology: the aerosolization and transport of viable microbial life by wildland fire

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Citation: Kobziar, L. N., M. R. A. Pingree, H. Larson, T. J. Dreaden, S. Green, and J. A. Smith. 2018. Pyroaerobiology: the aerosolization and transport of viable microbial life by wildland fire. Ecosphere 9(11):e02507. 10.1002/ecs2.2507

Abstract. The field of aerobiology is expanding due to a recognition of the diversity of roles microbes play in both terrestrial and atmospheric ecology. Smoke from global biomass burning has had significant and widespread ecological and human health consequences, but the living component of smoke has received little attention. Microbes aerosolized and transported by wildland fire may have profound effects on atmospheric and environmental factors, acting as nuclei for ice condensation, transporting pathogens or symbionts, and otherwise influencing ecosystems and human populations downwind. The potential for smoke to aerosolize and transport viable microbes is a virtually blank piece of the microbial biogeography puzzle with far-reaching implications. This study characterized the aerosolization of viable microbes via wildland fire smoke from burns in contrasting coniferous forests. Seventy aerosolized microbial morphotypes were recovered, and of these, a subset was identified using DNA analysis which revealed both pathogenic and non-pathogenic fungal species. Overall microbial colony-forming units decreased with increasing distance from smoke source, driven by bacterial abundance. Organisms were more abundant in smoke derived from mechanically treated fuels than intact forest floors and were most abundant in smoke from a dry, biennially burned *Pinus palustris* sandhill forest in Florida. Our findings of smoke-transported viable microbes have implications for ecosystem restoration/conservation, global biodiversity, meteorology, and human health.

Key words: aerobiology; atmospheric biology; bioaerosols; emissions; fire ecology; forest pathogen; fungal dispersal; microbial ecology; microbiology; prescribed fire; smoke; wildfire.

Received 3 October 2018; accepted 15 October 2018. Corresponding Editor: Michael F. Allen.

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INTRODUCTION

Long-distance transport of microbes has been documented across continents and oceans (Brown and Hovmoller 2002, Hara and Zhang 2012, Smith et al. 2012, 2013), as well as before and after storm winds and dust storms (Murata and Zhang 2014). These large-scale changes in microbial distribution demonstrate the likelihood for similar transport mechanisms of viable microbial communities in a different type of atmospheric vector—wildland fire smoke. Global biomass burning is responsible for aerosolizing approximately 42.2 Tg of particulate matter (PM) annually (Andreae and Merlet 2001), yet the contribution of combustion-aerosolized and viable microbial organisms has received little scientific attention. Aerosolized microbes can be pathogenic or beneficial to plant and human

health (Roux and O'Brien 2001, Griffin 2007), or may act to change microbial communities and their roles in both atmospheric and terrestrial environments (Morris et al. 2013, Golan and Pringle 2017). These diverse, abundant, and adaptable organisms may be integral drivers of ecosphere resilience and recovery, especially as natural and human-induced changes to climate and disturbance regimes continue.

Although wildland fire smoke plume temperatures can reach maximums well over the thresholds for most life-forms (e.g., >290°C at 4.5 m above a typical grassland fire; Clements 2010), the mixing of burned and unburned fuels, fluctuations in oxygen availability, meteorological factors, and entrainment of ambient air result in a mosaic of fire intensities and temperatures across spatial and temporal scales (Hiers et al. 2009). High-energy convection columns carry a wide range of particle sizes due to intense vertical air mixing (Clark et al. 1998, Lynch et al. 2004) and can result in the aerosolization and transport of organic matter and even mineral soils (Pisaric 2002, Bormann et al. 2008). Bioaerosols, or airborne particles with biological origins, have the potential for long-range transport and are likely to be associated with particulates, as previous studies in continental dust transport have shown (Hara and Zhang 2012). Wildland fire produces uniquely suitable substrates for organisms that may not otherwise survive in smoke. For example, pyrogenic C particles have been shown to provide habitat for soil microbes (Pietikäinen et al. 2000), a role that may extend to PM within a smoke column. A recent review of the long-distance transport of fungi mentions the potential, yet unknown, role for fire as a biogeographical dispersal vector (Golan and Pringle 2017), because even prescribed fire can evoke smoke plume rise to >1 km above ground level (Liu 2014). Yet wildland fire behavior has received little attention for its singular potential to aerosolize living microbes and transport them via smoke plumes.

The viability of microorganisms in smoke plumes is likely to be controlled by a combination of atmospheric and fire conditions, including relative humidity; temperature; convective forcing; degree of mixing; ultraviolet (UV) radiation; and oxygen content. Of parallel importance are the traits of the source microbial community and the types of material aerosolized (e.g., fuel source, pigmentation, high G + C nucleic acid content, high DNA repair ability, and UV protection; Mohr 2007). However, these complex associations have not been characterized in relation to wildfire or prescribed fire smoke presence. The composition of viable microbes transported by smoke may have significant implications for forest ecosystems and management. Understanding the fate of specific pathogenic and beneficial microbes can help direct broader restoration efforts for the conservation of affected ecosystems (Klopfenstein et al. 2010).

Of the numerous research publications pertaining to wildland fire smoke or aerobiology, we have only uncovered a single study that connects the two disciplines, and no exploration of this phenomenon in terms of microbiology, smoke science, fire behavior, and fire ecology from an interdisciplinary viewpoint (Fig. 1). Mims and Mims (2004) found a strong correlation ($r^2 = 0.78$) between fungal spores and aerosolized PM (assessed microscopically through visual counts of particles) deposited in Texas, USA, by smoke originating from wildfires on the Yucatán Peninsula, México. No assessment was conducted to verify that smoke particles were physically or biologically associated with the fungal spores. This case study also incorporated biological samples from a backyard experimental fire and measured higher numbers of colony-forming units (CFUs) on nutrient films exposed to smoke compared to those in ambient air. However, statistical tests were not conducted, and further study was not pursued by the authors.

The transport of viable aerosolized microorganisms via wildland fire smoke, hereafter referred to as "pyroaerobiology" (PAB; Fig. 1), is an integration of micro- and aerobiology, smoke and atmospheric sciences, fire behavior, and fire ecology in a coherent effort to understand the ecological and societal impacts of smoke-vectored microbes. The objective of this study was to gain a foundational understanding of the capacity of wildland fire to aerosolize viable fungi and bacteria in smoke, and how different combustion processes and sources may affect the aerosolized communities. Although various microbiological methods could be used to assess microbial composition and abundance in air masses (Haig et al. 2016), a commonly employed method for assessing the likelihood that organisms would survive after being aerosolized (i.e., capacity of microbes to

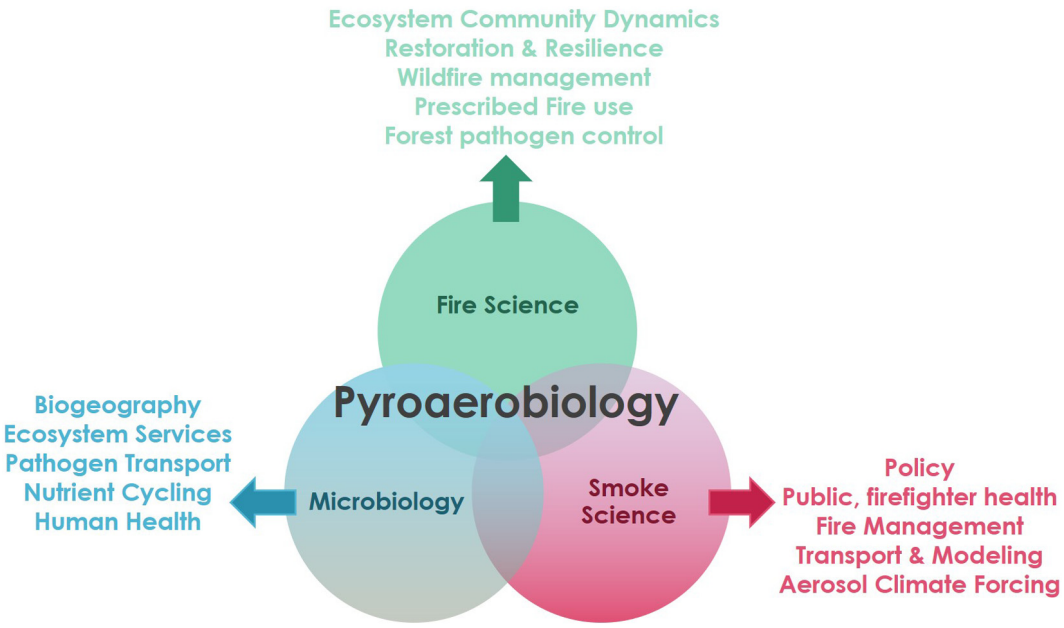

Fig. 1. Pyroaerobiology integrates theory and methods from microbiology, smoke and atmospheric sciences, and fire ecology, with a range of broader impacts and value added to each discipline.

influence the environment where they land) is the culturing of organisms (e.g., Yao and Mainelis 2007). This method is preferable because not only does it assess microbial presence, but it also allows for determination of post-fire viability. Because prescribed fires consume more biomass and typically burn more acreage than wildfires across the United States on an annual basis (NIFC 2018), and because prescribed fire scenarios allow for safe, direct access to the flaming front to control for differences in combustion type and fireline intensity, we performed this initial experiment using prescribed fires. To assess temperatures and determine whether mass loss corresponded to culturable microbial abundance, we conducted an additional study using burns in a controlled combustion laboratory using different fuel types. We tested three hypotheses about smoke-transported microbes during prescribed burns and laboratory combustion experiments by culturing impacted microbes, microscopic identification of morphotypes, and genetic analyses: (1) Viable microbe abundance as measured by CFUs will vary with increasing distance from the smoke source and will differ from ambient air; (2) viable species abundance will differ with the type of combustion (smoldering vs. flaming); and (3) viable species abundance will differ by site, where historical fire frequency, management, or fuel types differ.

Materials and Methods

Study sites

Two distinct studies with different methodology were conducted, based on limited available resources. In the Florida-based study, we utilized three 5- to 10-ha prescribed burns, while in Idaho, we transported forest floor samples and combusted them in a laboratory. The three burns were conducted in humid sub-tropical forests at the University of Florida's Austin Cary Forest approximately 18 km northeast of Gainesville, Florida, USA. The first burn was conducted on 3 April 2015 in a mature (70–90 yr old) *Pinus palustris* sandhill ecosystem (Myers and Ewel 1990) maintained by a two-year prescribed fire return interval since 2003 (Sandhill Biennial), while the second burn was on 6 August 2015 in a mature longleaf pine flatwoods (distinguished from sandhills by a higher water table and sub-surface spodic horizon) ecosystem that was burned annually since ca. 1990 (Flatwoods Annual). The third and final prescribed burn took place on 25 September 2015 in a hydric *P. palustris* and

Pinus elliottii flatwoods ecosystem that was previously (and incompletely) burned only once since 1940 (in 2012), characterized by a heavy buildup of surface and ground fuels (i.e., understory vegetation and organic soil horizons).

North Idaho forest floor sampling sites were located in semi-arid steppe forests at the University of Idaho Experimental Forest (UIEF) on the Palouse Range, about 20 km northeast of Moscow, Idaho, USA. These mixed-conifer forest stands consist of a diverse coniferous overstory dominated by *Pseudotsuga menziesii*, *Abies grandis*, *Thuja plicata*, and *Pinus ponderosa* var. *ponderosa*, and understory species dominated by shrubs, with fuel reduction treatments as described by Sparks et al. (2017).

Soil sample collection—mixed-conifer forest in Idaho

Soil samples of the entire organic horizon (O horizon) were collected from three forest stands planted in 1982. Each stand had two treatments including understory fuel reduction (all surface fuels and small trees shredded and left on site—masticated; see Sparks et al. 2017), and one left untreated—control. Three soil samples were collected to the entire depth of the organic soil horizon (including O_i, O_e, and O_a layers) using a 17 cm diameter ring at three randomly located plots within both treatments in each stand (n = 18). Samples were kept cold for seven days (2°C), air-dried for 48 h in the laboratory under a sterilized closed laminar flow hood, and then composited by treatment by stand prior to combustion. Compositing was used to account for high spatial variability within sub-sections, and to achieve sufficient masses for continuous flaming and residual smoldering combustion subsequently in the combustion laboratory (n = 6).

Bioaerosol sampling—combustion laboratory in Idaho

Because we sought to culture the microbes, sampling durations were limited to 2 min in order to reduce the potential for desiccation and damage to the organisms (Mainelis and Tabayoyong 2010). To assess background levels of aerosolized microbes, ambient air samples were taken in the field at each sampling location by exposing one Petri dish with sterilized potato dextrose agar medium at one meter above the ground surface, quickly sealing it with Parafilm, and storing it in a cooler for immediate transport to the laboratory (n = 18). A combustion laboratory (IFIRE Lab, University of Idaho, Idaho, USA) was used for the burn experiments. The laboratory is comprised of a preparation and control room where data are monitored, and a separate combustion chamber within a climatically controlled room containing an over-sized dedicated fume hood. O horizon samples were transported to the combustion laboratory, and using sterile techniques, ~100–200 g of soil was transferred into sterilized metal pans prior to ignition. Pan contents were burned on a table scale to measure real-time mass loss rates (per second, and as percent of initial mass). Three type-K, 20-AWG fiberglass-sheathed thermocouple wires (Omega Engineering, Stamford, Connecticut, USA) were positioned within the fuel bed at 0, 15, and 60 cm above the fuel bed to monitor temperatures during burning using a datalogger in the preparation room. During flaming and then smoldering combustion, three Petri dishes were suspended approximately 50 cm to 1 m above the fuel source for a total of nine replicate smoke samples for each stand × fuel treatment combination (total number of Petri dishes subjected to smoke in laboratory = 18, with nine smoldering and nine flaming combustion samples). Two additional dishes were exposed for the same time period prior to any ignitions to serve as laboratory blanks. After exposing the dishes to smoke and ambient laboratory air, dishes were sealed, stored in a cooler, and transported to a microbiology laboratory for incubation using sterile handling techniques.

Bioaerosol sampling—prescribed burns in Florida

The three prescribed burns were conducted within the late-spring-to-late-summer growing season. All prescribed burns were ignited using a combination of flanking and strip head fire ignition patterns. Flame lengths (indicative of fire intensity and ranging from 0.5 to 3 m) were estimated by ocular comparison to a 1-m pole (Kreye and Kobziar 2015) and ranged from an average value of 0.9 m in the Flatwoods Annual site, 2.3 m in the Sandhill Biennial site, to 3.8 m in the Long-Unburned site.

All samples were collected via passive impaction onto malt extract agar (MEA) medium in Petri dishes manually oriented into the wind.

Sampling locations at each burn were established when consistent fire rate of spread was observed, and outside of direct ignition zones. Samples were collected at the origin (approximately 0.5 m) and increasing distances from the flaming front relative to flame length. Sterilized Petri dishes with MEA were suspended approximately 1–2 m above ground level on platforms attached to extension poles facing upwind into smoke plumes for two minutes, closed, sealed with Parafilm, and stored in a cooler for 1–2 h before transportation to the laboratory. During the burns, three to four ambient air samples per burn were taken at a minimum of 9 m upwind and away from any perceivable influence of smoke, but within the same hour and site conditions. Samples were also collected during smoldering combustion at 0.5–1 m from the source of combustion. No smoldering samples were collected at the Flatwoods Annual site due to the insufficient source of smoldering combustion. The number of samples taken was dictated by opportunities for safe entry into the burn zone, and the total duration of each burn.

Microbial culture processing—both locations

Samples were transported to the laboratory and kept at room temperature (~23°C) in the dark until colonies had developed, 72 h and 7–14 d for Florida and Idaho samples, respectively. Plates were visually examined under a 40× microscope, classified into morphotypes, and CFUs for fungi and yeasts were counted. After approximately one week, a colony from each morphotype from the Florida sites was subcultured on acidified potato dextrose agar (APDA) to enable targeted analysis of morphological characteristics (e.g., spore production, colony features). Of seventeen different fungal morphotypes, eight randomly selected morphotypes were subcultured on sterilized cellophane on APDA for seven days, to facilitate growth and harvesting of material for DNA extractions. Standard sterile technique and analysis within a laminar air flow hood were used throughout with all cultures maintained at ~23°C in the dark.

DNA extraction, amplification, and sequencing—Florida samples

The randomly selected unique morphotypes from the Florida smoke samples had DNA extracted by harvesting a portion of the colony from the cellophane, using the DNeasy Plant Mini Kit (QIAGEN, Hilden, Germany), following the manufacturer's instructions. The nuclear ribosomal internal transcribed spacer (ITS) region from fungal morphotypes was amplified by polymerase chain reaction (PCR) with primers ITS1F (5′-CTTGGTCATTTAGAGGAAGTAA-3′) and ITS4 (5′-TCCTCCGCTTATTGATATGC-3′; White et al. 1990) using PCR conditions from Sena et al. (2018), Sanger-sequenced at the University of Florida Genetics Institute in Gainesville, Florida, USA, and compared to sequences in GenBank. The Idaho samples were not sequenced due to resource limitations.

Statistical analysis

Data were explored and analyzed in the R environment (R Core Team 2016). Total CFUs from the Florida study were analyzed pooled and separate for each site in order to evaluate the relationships between colonized microbes and fire or site factors, respectively. The total CFUs were then analyzed by growth forms. To meet statistical assumptions, total CFU data were transformed by $\log(x + 1)$ and then compared with an ANOVA against combustion type and distance from flaming front within each ecosystem. When split into organism types, we used Kruskal–Wallis tests as the data did not fit all assumptions of ANOVA. Where applicable, transformed CFU data were tested in a regression model against distance from the flaming front. Total CFUs from the Idaho study were transformed by $\log(x + 1)$ to meet ANOVA assumptions and tested against fuel type, organism types, and regressed against temperatures and mass loss (%) during combustion.

Results

Florida prescribed burn sites

Across the three Florida prescribed burn sites, distance was negatively related to the average number of CFUs for all organism types during flaming combustion ($n = 36$, $r^2 = 0.77$, $P < 0.001$; Fig. 2). Although there were higher CFU counts for filamentous fungi compared to bacteria and yeast ($n = 123$, 47, and 25, respectively), the relationships between CFUs and distance (Fig. 2) were driven by bacteria. Overall, filamentous

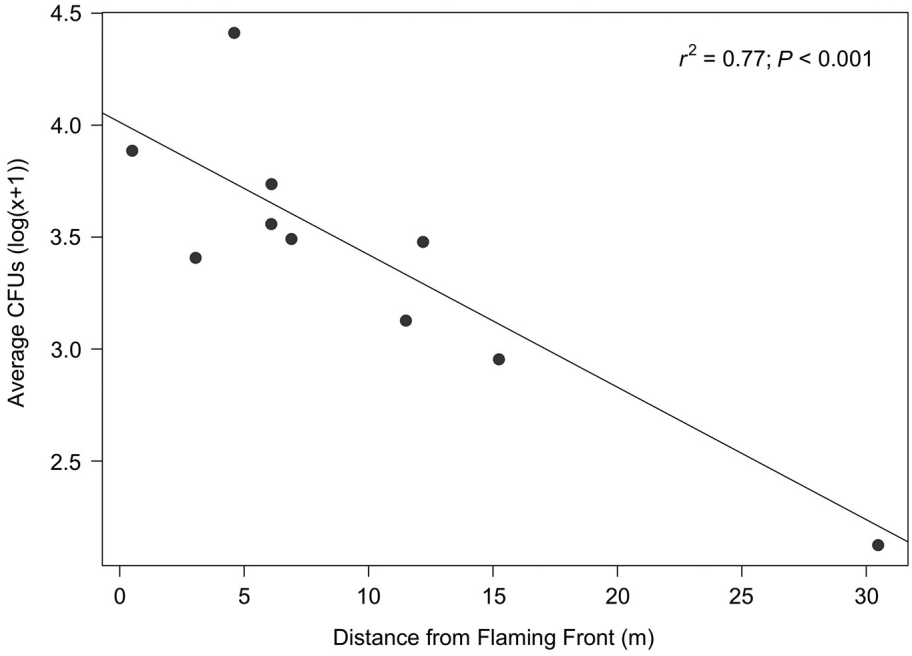

Fig. 2. Number of colony-forming units averaged for each distance from the flaming front of experimental prescribed fires in all north Florida sites (n = 7, 8, 7, 2, 4, 2, 2, 2, 2, 2, from 0.5 to 30 m, respectively). Total number of samples taken = 38.

fungal and yeast CFUs were not significantly related to distance or distance divided by flame length when analyzed independently ($P > 0.05$). Smoke samples from the Sandhill Biennial site showed a significant ($P < 0.001$) and negative correlation ($r^2 = 0.40$) between bacterial CFUs and distance from the flaming front. The Flatwoods Long-Unburned site showed significant differences ($P < 0.01$) in CFUs among distances from the flaming front, which were lowest at the origin and outside of the smoke (at 30 m) but highest at 3- and 6-m (2–3 times the flame lengths) collection points, suggesting convective wind vortices may have aerosolized organisms from the abundant and tall (>1 m) understory vegetation unique to this site.

Samples taken during flaming combustion yielded higher CFUs compared to ambient samples ($P < 0.05$) but were not significantly different from smoldering samples (ambient n = 7, flaming n = 28, smoldering n = 22; Fig. 3). Total CFUs, regardless of combustion type, were highest in the driest (based on soil type) burned site (Sandhill Biennial: 171, n = 30) and the mesic Flatwoods Annual (104, n = 14) sites, compared to the Flatwoods Long-Unburned site (69, n = 27). Colony-forming units were significantly higher in the Sandhill Biennial site compared to Flatwoods Long-Unburned site ($P < 0.001$). In the Sandhill Biennial site, CFUs were highest and most variable when aerosolized by flaming combustion; they were significantly lower in ambient samples when compared to both types of combustion ($P < 0.05$; Fig. 3). Eight unique fungal morphotypes isolated from smoke samples and identified using ITS sequences show a diverse group of fungi, representing several orders and ranging from pathogens to non-pathogens with diverse ecological roles (Table 1).

Smoke samples collected during laboratory experiments on soils from Idaho bore unique morphotypes in all treatments: These were highest in masticated fuel sites (ambient samples contained 15 total morphotypes with five unique; burned samples had 11 morphotypes with five unique). The unique morphotype assemblages in burned, unburned, and masticated fuel beds suggest that combustion aerosolized microbes that would not be found in ambient air in the conditions and season in which we sampled.

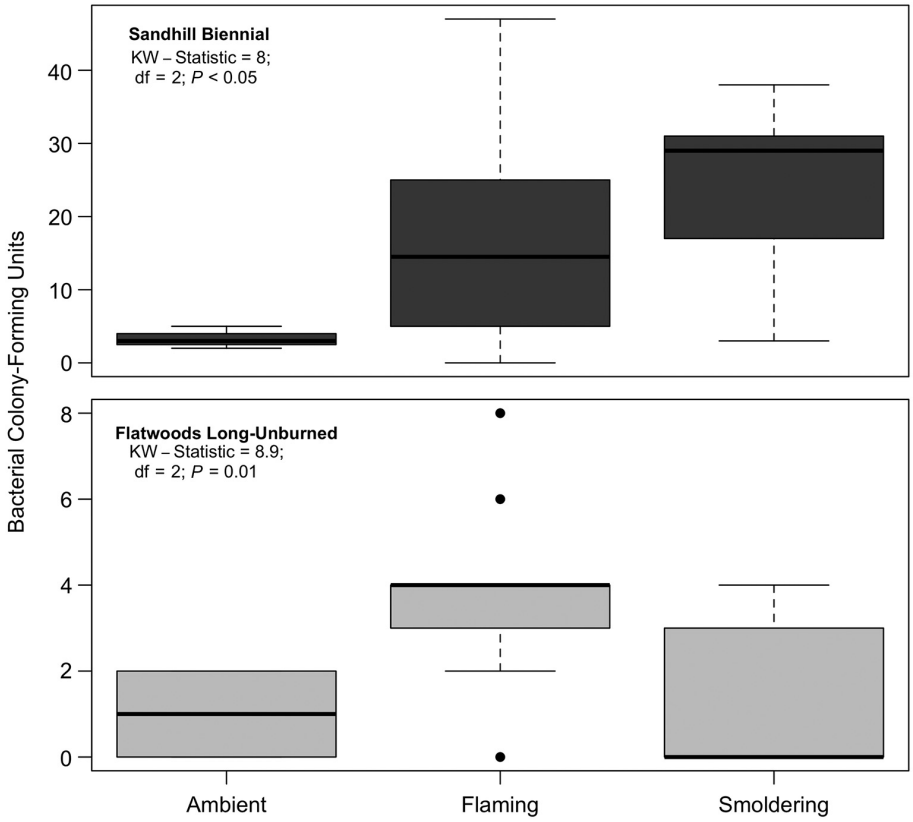

Fig. 3. Bacterial colony-forming units collected during ambient, flaming, and smoldering combustion in Florida prescribed burns in Sandhill Biennial sites (n = 3, 18, 9 for ambient, flaming, and smoldering combustion types, respectively) and Long-Unburned Flatwoods (n = 4, 10, 13 for ambient, flaming, and smoldering combustion types, respectively). The average flame length at the Sandhill Biennial site was 2.3 m with a relative humidity (RH) of 39%, and 3.8 m at the Long-Unburned Flatwoods sites with an RH of 57%. The Flatwoods Annual site was not included because it lacked sufficient smoldering combustion.

Table 1. Fungal identifications for eight unique morphotypes from prescribed fire smoke samples in a biennially burned Sandhill pine ecosystem and Long-Unburned Flatwoods pine ecosystems in north Florida.

Best BLAST	GenBank no.	Identities	Ecological function
Trichoderma strigosum	EU718074	601/604	Functions in nutrient and mineral uptake, genus important in agricultural remediation
Dothideomycete sp. 1	EU680480	546/546	Often found as pathogens, endophytes, or epiphytes of living plants. Saprobes degrade cellulose and other complex carbohydrates in dead or partially digested plant matter
Dothideomycete sp. 2	HQ631008	584/595	As above
Pestalotiopsis sp.	KX757719	546/546	Parasitic fungus that targets ants, also plant pathogens
Epicoccum nigrum	MF687186	541/541	Endophyte and plant pathogen, produces anti-fungal and anti-bacterial compounds
Neopestalotiopsis australis	KY398730	547/547	Endophytic fungus capable of breaking down and digesting polyurethane, can metabolize under anaerobic conditions
Hypocreales sp.	KP306921	437/451	Diverse functions by species
Penicillium lagena	NR_153223	549/549	Non-pathogenic fungi present around mycorrhizal roots

Notes: Identities are based on best BLAST matches to the NCBI database using the internal transcribed spacer region. Identities are nucleotide matches between the morphotype and GenBank accession. Ecological function is based on brief literature review of the best BLAST match.

Unlike trends in the Florida samples, Idaho forest microbial CFUs in smoke derived from flaming combustion did not differ significantly between combustion types nor when compared with the ambient samples. The composition of morphotypes, however, differed between the ambient and the combusted samples, with eight unique morphotypes occurring only in smoke samples. In addition, the masticated fuel type aerosolized more CFUs compared to untreated fuel, but neither had significantly different CFU numbers from the ambient samples ($P < 0.01$). While the quality of the fuel source (i.e., masticated or not) of burned organic matter had a significant influence on the number of CFUs in smoke, the quantity burned (% mass loss or total mass loss [g]) did not correlate with total CFUs ($n = 18$, $r^2 = 0.30$, $P = 0.26$). Idaho smoke samples showed higher mean CFUs per sample of fungi than bacteria (13 and 4, respectively). Temperatures were not measurably correlated with morphotype composition or number of CFUs; however, the maximum temperatures of flaming vs. smoldering combustion from the fuel bed thermocouples were higher (528°C vs. 395°C, respectively), and temperatures at 60 cm height, where samples were taken, at times exceeded 60°C during both flaming and smoldering combustion.

Discussion

Pyroaerobiology, a term we introduce in this study, represents an interdisciplinary and little-researched line of inquiry, integrating terrestrial ecology, aerobiology, smoke science, microbiology, fire behavior, and fire ecology in a coherent effort to understand the impacts of aerosolized live pyrogenic material. Because this line of inquiry is a new application of aerobiology, our exploratory study was designed to provide evidence for the potential for smoke to aerosolize and transport viable microorganisms and to test some basic hypotheses. Our study was inherently limited by the specific source/fuels sampled, fire behavior characteristics, sampling duration and methods, and processing methods, so that specific results should not be extrapolated to other fires. We used culture-based methods to capture and grow viable microbes from smoke using a single medium in each study. It is well established that the media used will affect the microbes recovered and that most microbes are unculturable. We used a general growth medium able to grow many fungi and bacteria but presumably only cultured a small portion of the potentially viable microbes in the smoke. In addition, different sampling durations would likely lead to different results (Mainelis and Tabayoyong 2010).

These initial studies using prescribed burns and laboratory experiments show that fire aerosolizes and smoke transports a variety of viable, culturable microbes, and these assemblages are dissimilar in composition and abundance from the communities aerosolized by background aerosolization drivers (e.g., wind, gravity, spore propulsion) in paired samples. While our study addressed forest stand-level transport of organisms, longer-distance transport and its implications would depend on fire behavior and sources of the microbes (e.g., the microbial community), season (which affects sporulation, activity, and probably survival), environmental conditions (recent rain events, winds affect background levels of aerosolized organisms), and the physiological hardiness and growth potential of the organisms or propagules aerosolized (e.g., fungal hyphae, spores, and their dispersal mechanisms; Golan and Pringle 2017).

Two of our hypotheses were supported by our data, including that abundance (CFUs) varied with distance from the smoke sources and that species abundance and composition would differ by site/site conditions (e.g., mastication). Our results suggest that the more frequently burned sites have higher numbers of viable aerosolized organisms in smoke overall, which may reflect fire history and associated microbial fire adaptations (Glassman et al. 2016), or differences in the types of fuels combusted. Grasses and pine litter drove fire behavior in the frequently burned sites in contrast to shrubs, grasses, and even small trees in the Long-Unburned site. Comparisons of the source microbial populations among sites would be needed to draw conclusions about whether the differences in aerosolized communities are a function of source, fire behavior, or even sampling protocols (including culture medium used). These results suggest that future PAB research should include an assessment of smoke source communities in order to derive predictions for the potential impact on atmospheric and downwind terrestrial

communities. Other improvements would include larger sampling sizes, methods parameterized for expected aerosol densities, and employing metagenomic analyses to reduce bias against non-culturable species.

The Idaho mixed-conifer forest samples demonstrated that masticated fuels produced more CFUs and unique microbial communities than non-treated stands, as indicated by distinct morphotypes, and that both differed from ambient communities. Mastication changes individual fuel (soil O horizon) surface areas, fuel packing ratios, moisture content, and depth (Kreye et al. 2013), hence changing the microhabitat. As such, fuels treatments may have an impact on microbial communities that then extends to aerosolized and dispersed microbes when and if fire occurs. Mixing of the soil organic horizons due to heavy equipment used for fuel reduction treatments may expose organisms whose habitats would otherwise be unavailable for aerosolization via combustion.

Our second hypothesis that species abundance differed with combustion phase was only supported in the Florida sites. Flaming combustion burned smaller diameter woody litter, soil organic horizons, and surface fuels including shrubs, grasses, and herbaceous vegetation. In contrast, smoldering combustion samples were necessarily obtained from residual fuel, often larger woody debris after the initial passage of the fire front. The source of the microbial materials was therefore different between the two phases of combustion; we cannot isolate the effect of the fuel source from that of the phase of combustion. That the different phases of combustion did not produce significant results in the laboratory burns using Idaho mixed-conifer O horizon samples, along with the lack of a temperature effect, implies that the energetic differences between the combustion phases were not significant for the microbes we were able to culture.

Heating from wildfire and prescribed fire events have poorly understood physiotemporal effects on soil microbial populations (Pingree and Kobziar 2019). In forests devoid of regular fire disturbances, prescribed burning employed as a restoration effort may negatively impact ecosystem processes. For example, soil heating may be substantially increased where organic soil horizons are deeper, as in the Long-Unburned Florida site, leading to prolonged heating and increased temperatures (Varner et al. 2005), and increased potential for greater numbers of microbes to be aerosolized. Indirectly, the exclusion of frequent, low-severity fires may favor the proliferation of a soil microbial community with lower temperature thresholds and disturbance adaptability compared to a frequently burned forest soil community (Hart et al. 2005). These altered microbial communities may also be transported and relocated via aerosolization or particle-mediated transport in smoke with unknown consequences for adjacent ecosystems. Efforts to measure and characterize wildland fire effects on microbial species can help to improve management of sensitive and rare ecosystems where recurrent fire and adapted microbial species are closely coupled with ecosystem function (Glassman et al. 2016).

Societal impacts of smoke-transported living microbes could be both indirect (e.g., ecosystem services) and direct (human health). Microorganisms provide integral functions in forest ecosystems including decomposition and C cycling, nutrient cycling, production and consumption of greenhouse gases, development of soil structure and maintenance, and effects on other soil biota. Understanding the fate of specific pathogenic and beneficial microbes could help direct broader restoration efforts for the conservation of affected ecosystems (Klopfenstein et al. 2010). Theoretically, managers could retard spreading of detrimental pathogens and promote dissemination of beneficial mycorrhizae or nitrogen-fixing bacteria, or other microbes that would benefit society.

The viability and composition of microbes transported by smoke may have significant implications for forest health. For example, the fungal pathogen *Cronartium ribicola* (J.C. Fisch.), which causes white pine blister rust and threatens the endangered whitebark pine (*Pinus albicaulis* Engelm.), was spread to new hosts in the western United States via long-distance dispersal by atmospheric transport (Frank et al. 2008). If this pathogen is viable in smoke, disease spread may be vectored by smoke as well. It is currently unknown what role smoke and wildfire play in the transport of forest pathogens. These consequences may in fact be an undesired impact of management practices. For example, a recommended practice to dispose of biomass infected with plant pathogens (e.g., *Phytophthora ramorum*,

which causes Sudden Oak Death) is to burn the material (Agrios 2005). Such attempts at pathogen control may actually disperse pathogens depending on environmental conditions (e.g., as has been shown in a study of wheat field burning; Roux and O'Brien 2001). With additional knowledge, managers could plan burns when conditions are unlikely to transport pathogens present in a stand to uninfected areas.

Relationships between microbial transport and smoke composition may thereby help guide smoke management decisions with significant consequences (Bowman and Johnston 2005). Smoke plumes from wildland fires impact natural resource management decision-making, public opinion, and public safety and have catalyzed immense planning and coordination efforts by multiple stakeholders (Hardy et al. 2001). Future investigation into targeted species of special concern to human health impacts is also warranted, because aerosolized microbes are well known to aggravate patients with asthma and even cause illness in otherwise healthy individuals (Griffin 2007).

Conclusions

The addition of viable organisms to the atmosphere may alter bioaerosol species composition, activity, and growth, with effects on biogeochemical cycling, atmospheric cloud development, and weather (Morris et al. 2013, Krumins et al. 2014). Our study provides an initial foundation for a broad spectrum of future inquiries. Under what conditions does wildland fire smoke transport and deposit active plant pathogens to adjacent or distant locations, and what are the potential consequences? Can human pathogens and allergens be transported in smoke to such an extent that they affect sensitive populations, and more immediately, wildland fire personnel? With these and other questions in mind, the establishment of appropriate conceptual and methodological guidelines for this line of inquiry is needed. The approach should be grounded in the established principles and theory of aerobiology, which emphasize the importance of addressing the complete aerobiological pathway. Ultimately, pyroaerobiology should take into consideration the source, characteristics of aerosolization (i.e., launch), atmospheric transport, deposition, and direct and indirect subsequent ecological impacts (Edmonds 1979).

We suggest the following considerations be applied to future studies:

1. PAB sampling systems should sample smoke plumes from multiple fire types at increasing heights and distances from the source using mobile platforms in smoke columns; these would enable smoke communities to be sampled specifically, the influence of ambient air entrainment to be characterized, and the degree of transport to be quantified;
2. PAB sampling should incorporate a wide range of microbiological diversity assessments including community sequencing, various media, and baiting. Phylogenetic analyses coupled with physiological/morphological examinations of the species identified may shed light on the evolution of the pyroaerobiome.
3. PAB sampling strategies should integrate environmental and aero-habitat conditions (e.g., PM levels, relative humidity, temperature, UV exposure) concurrently with sample extraction in order to characterize and compare aero-habitats;
4. A variety of sampling techniques (e.g., impaction, filtering, impingement) and durations need to be laboratory- and field-tested for maximum capture of all viable organisms to determine appropriate sampling duration and volume for the unique habitat of wildland fire smoke;
5. To link these effects with predictive models of smoke transport and effects, an understanding of fire behavior, source substrates, and how they interact to aerosolize microbes is needed. PAB must include all sub-disciplines to address the questions of relevance to ecological systems and processes, as well as potential human health impacts.

Acknowledgments

We wish to acknowledge the personnel of the University of Florida's Austin Cary Forest for assistance with prescribed burning operations, and the contributions of University of Idaho's IFIRE combustion laboratory personnel. The INBRE program supported one of the study's authors through a summer internship associated with the Environmental Science

program in the University of Idaho's College of Natural Resources. This work was supported in part by grant no. IDAZ-MS-0115/project accession no. 1009933 from the USDA National Institute of Food and Agriculture. Thanks to Dr. Jennifer Andrew for initial contributions. Publication of this article was funded by the University of Idaho Open Access Publishing Fund.

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International Journal of Wildland Fire **2010**, *19*, 803–817

The economic cost of adverse health effects from wildfire-smoke exposure: a review

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Abstract. The economic costs of adverse health effects associated with exposure to wildfire smoke should be given serious consideration in determining the optimal wildfire management policy. Unfortunately, the literature in this research area is thin. In an effort to better understand the nature of these economic costs, we review and synthesise the relevant literature in three areas: studies that estimated the health-related economic costs of wildfire-smoke exposure; epidemiology studies related to the health risk of wildfire smoke; and general economic studies that estimated the monetary value of preventing the specific adverse health outcomes. Based on the findings from this literature review, we identify the need for a better understanding of the effect of wildfire smoke on major and minor adverse health outcomes. It would also be useful to know more about averting behaviours among residents exposed to smoke during a wildfire event. Finally, we suggest investigating the unique health effects of wildfire smoke compared with conventional air pollution to determine whether it is appropriate to extrapolate from previously estimated conventional pollution dose–response functions.

Additional keywords: epidemiology studies, forest fires, health damage, non-market valuation, particulate matter.

Introduction

The economic costs associated with the adverse health effects of wildfire-smoke exposure can be an important consideration in wildfire management. For example, concerns about adverse health effects from 2008 wildfires in northern California prompted the USDA Forest Service to actively suppress all wildfires in California. However, despite the emphasis placed on reducing the health risk, the science demonstrating health effects from wildfire-smoke exposure is incomplete and at times contradictory. In addition, there are few monetary estimates of the economic costs associated with the adverse health effects of wildfire-smoke exposure.

Evaluating the health-related economic costs of wildfire smoke involves two steps. First, the total adverse health outcomes associated with a wildfire event are quantified (such as 10 excess deaths or 100 excess hospital admissions for a particular illness during wildfire events). The quantified adverse health outcomes are then monetised by multiplying each health outcome by per-unit cost. In this paper, we review and synthesise the literature related to the health-related economic costs of wildfire-smoke exposure in an effort to understand the nature of this cost, to provide a comprehensive list of available studies in related fields, and to identify the key issues worthy of future investigation. We summarise three research literatures: studies that estimated the health-related economic cost of wildfire-smoke exposure; epidemiology studies related to the health effect of wildfire smoke; and general economic studies that estimated the monetary value of preventing the specific adverse health outcomes (premature mortality and various cardio-respiratory symptoms). We also discuss how the health risk of wildfire smoke could be considered in wildfire management decisions.

The rest of the article is organised into six sections. The first section describes the study methodology. The second section reviews studies that estimated the health-related economic costs of wildfire-smoke exposure. The third section summarises the epidemiology literature related to health effects of wildfire smoke. Specifically, we compare and contrast studies that examined the health effect of particulate matter (PM) exposure from industrial sources (urban PM exposure) with studies that

10.1071/WF09077 1049-8001/10/070803

examined the health effect of wildfire-smoke exposure.[A] The fourth section reviews the economic valuation literature on health outcomes related to air pollution. The fifth section highlights some potential policy considerations, and the last section provides a conclusion.

Study methodology

Systematic literature reviews were conducted in economics, epidemiology and wildfire policy research fields to identify relevant published and unpublished papers. Searching the published literature was straightforward as there are many web-based databases such as Econlit and Medline. We also relied on previously published review papers and references from recent articles in the related fields. In addition, we contacted individuals who have published in the relevant literature to inquire about recent publications and unpublished papers. On-line search engines, such as Google, were also used. For the epidemiology literature, we only summarised studies that conducted tests to determine the statistical significance of the results.

Health-related economic cost of wildfire-smoke exposure

Studies that estimated health-related economic costs of wildfire-smoke exposure are sparse. Our literature search turned up six relevant studies worldwide. The magnitude of estimated health-related economic costs depends on the scale of the wildfire event, demographic characteristics of the exposed population (residents of developed or developing countries), and the type of adverse health outcomes considered. Table 1 summarises the location of wildfires, the measured adverse health outcomes, the type of dose–response function used to quantify the level of adverse health outcomes, and estimated economic costs in each study. The economic costs are estimated either by a willingness to pay (WTP) approach or a cost of illness (COI) approach. The WTP approach measures the comprehensive economic cost, whereas the COI approach measures only direct cost associated with illness. The characteristics of each approach are discussed further in the fourth section.

When estimating health-related economic costs, the selection of adverse health outcomes to be quantified is somewhat subjective. The United States Environmental Protection Agency (US EPA) has identified an extensive list of possible health effects from PM, one of the major pollutants associated with wildfire smoke, that ranges from acute minor symptoms to premature mortality (US EPA 1999). As shown in Table 1, common adverse health outcomes considered in previous studies are hospital admission for respiratory and cardiac symptoms, hospital outpatient visits for respiratory symptoms, work loss days and restricted-activity days. Only Rittmaster *et al.* (2006, 2008) included the economic cost of premature mortality due to wildfire-smoke exposure.[B] This estimated cost of premature mortality is substantially larger than any of the other costs reported in Table 1, while the number of estimated excess deaths is very small (0.4–0.5 estimated excess deaths). Although the method employed in Rittmaster *et al.* (2006, 2008) to quantify the number of excess deaths from a wildfire event has its limitations, and could cause overestimation of the mortality impact of wildfire smoke, the studies of Rittmaster *et al.* suggest that the omission of mortality costs in the other studies summarised in Table 1 may results in substantial underestimates of total health costs.

Work days lost, restricted-activity days, and minor restricted-activity days contribute substantially to total morbidity-related costs, and account for 36 to 74% of total estimated health costs in the studies that did not consider premature mortality. The studies summarised in Table 1 also suggest that hospital admissions, respiratory symptoms and self-treatment are major health-cost components.

The approach used to quantify adverse health impacts from wildfire-smoke exposure could have important implications for the validity of the estimates. There are two approaches to quantify the level of adverse health outcomes. One approach is to use original health data such as vital statistics or hospital discharge data to measure the adverse impact of a wildfire event. The other approach is to extrapolate existing dose–response functions between air pollution and adverse health outcomes. The latter approach is commonly used in policy analysis by the US EPA (1999, 2005) because it only requires air pollution data to estimate the level of adverse health effects of any event.

Martin *et al.* (2007) and Rittmaster *et al.* (2006, 2008) use existing dose–response functions based on urban air pollution PM and adverse health outcomes. Although wildfire smoke contains substantial amounts of PM, the problem of this approach is that exposure to PM from wildfire smoke may result in different health effects. Most existing PM dose–response functions, including ones employed in Martin *et al.* (2007) and Rittmaster *et al.* (2006, 2008), are based on low to moderate concentration levels of PM exposure from urban air pollution sources such as fossil-fuel burning (hereafter referred as conventional PM studies). Wildfires often result in short-lived, but very high levels of PM from vegetation burning. As discussed later, some researchers have argued that the different chemical properties and circumstances of urban air pollution and wildfire smoke may result in different health effects. Therefore, although it is convenient to use conventional PM studies to estimate the level of adverse health outcomes of wildfire smoke, it is not clear that this approach is appropriate. In the next section, we compare the findings from conventional PM and wildfire-specific epidemiology studies in an effort to understand whether it is appropriate to extrapolate results from conventional PM studies to estimate health effects from wildfire-smoke exposure.

Epidemiology studies: urban air pollution *v.* wildfire smoke

In this section, we review and summarise the findings from conventional PM studies and wildfire-specific epidemiology

[A]Particulate matter (PM) is categorised as PM_{10}, which is particles less than 10 μm in diameter, and $PM_{2.5}$, which is particles less than 2.5 μm in diameter.
[B]The estimate of mean health-related cost in Rittmaster *et al.* (2006) was corrected in Rittmaster *et al.* (2008).

Table 1. Previous economic analysis of wildfire-smoke-induced health damage

COI denotes values obtained from Cost of Illness approach, WTP denotes values obtained from willingness to pay approach, $PM_{2.5}$ denotes particulate matter with particles less than 2.5 μm in diameter, and PM10 denotes particulate matter with particles less than 10 μm in diameter. Total cost may not be same as sum of all cost due to rounding

	Name of fire and description	Health outcomes	Source of dose–response function	Estimated economic cost (central estimate unless specified, 2007 $US value)
Cardoso de Mendonça *et al.* (2004)	Fires in Amazon between 1996 and 1999	In-patient treatment (respiratory)	Original	$2 100 000 (annual average COI) $9 400 000 (annual average WTP)
Martin *et al.* (2007)	Hypothetical prescribed fire in Kaibab National Forest in USA	Hospital admissions (respiratory and cardiac)	Conventional dose–response functions	$52 490 (COI)
		Lower respiratory symptoms (children)		$25 510 (WTP) (both children's health effects)
	Affected city: Flagstaff	Acute bronchitis (children)		
		Work-loss days (WLD)		$106 996 (WLD and RAD: COI; MRAD: WTP)
	Worst-case scenario: total 1935 $\mu g\,m^{-3}$ $PM_{2.5}$ increase	Restricted-activity days (RAD)		
		Minor restricted-activity days (MRAD)		
		Total		**$185 002 (COI and WTP mix)**
Rittmaster *et al.* (2006, 2008)[A]	2001 Chisholm fire in Canada	Mortality	Conventional dose–response function	$2 292 224 (WTP)
		Hospital admissions (respiratory)		$1901 (COI)
		Hospital admissions (cardiac)		$2038 (COI)
	7 days fire, 2 days of heavy smoke	Emergency room visits		$761 (COI)
		Restricted activity days		$289 306 (WTP, COI)
		Asthma symptom days		$17 341 (WTP)
	Peak $PM_{2.5}$: 55 $\mu g\,m^{-3}$ daily average; 261 $\mu g\,m^{-3}$ hourly average	Bronchitis admissions		$9580 (COI)
		Acute respiratory symptom events		$93 630 (WTP)
		Total		**$2 706 782 (COI and WTP mix)**
Shahwahid and Othman (1999)	1997 Asian Haze in Malaysia	Hospital admissions	Original	$610 725 (COI)
	Smoke period: August–November 1997. Air pollution index reached dangerous level. Declared state of emergency for 10 days	Outpatient		$1 820 346 (COI)
		Self-treatment		$768 754 (COI)
		Work-loss days		$294 003 (COI)
		Restricted activity days		$1 928 967 (COI)
		Total		**$5 422 798 (COI)**
		Adjusted total[B]		**$10 845 597 (WTP)**
Hon (1999)	1997 Asian Haze in Singapore	Hospital admissions	Original	$123 010–$512 544 (COI)
	14 days unhealthy 24-h average air quality level	Outpatient (less severe)		$642 549–$1 606 372 (COI)
		Outpatient (severe)		(all outpatient and self-treatment)
		Self-treatment (less severe symptoms)		
		Work-loss days		$1 964 550–$6 527 998 (COI)
		Total		**$2 730 109–$8 646 916 (COI)**
		Adjusted total[B]		**$5 460 220–$17 293 831 (WTP)**
Ruitenbeek (1999)	1997 Asian Haze in Indonesia. PM_{10} in Jambi recorded 1864 $\mu g\,m^{-3}$ (Kunii *et al.* 2002)	Hospitalisation	Shahwahid and Othman (1999)	$380 160 000 (all medical costs)
		Outpatient		
		Self-treatment		
		Work-loss days		$215 820 000 (COI)
		Total		**$595 980 000 (COI)**
		Adjusted total[B]		**$1 191 960 000 (WTP)**

[A]Canadian dollar is converted to US dollar using 1996 average exchange rate (CAN$: US$ = 1 : 0.73).

[B]Adjusted total is obtained by multiplying the total cost estimated from the COI approach and WTP/COI ratio of 2.

Table 2. Summary of conventional particulate matter (PM) health impacts

Weighted average is obtained in the following manner. First, we obtain the average relative risk and 95% confidence interval for each selected study. Then we calculate weighted average from $\bar{x} = \sum_{i=1}^{n} x_i / \text{s.e.}_i / \sum_{i=1}^{n} \text{s.e.}_i$ where x is estimated relative risk, s.e. is standard error of estimated relative risk and i denotes different study

Health outcomes	50-μg m^{-3} increase of daily PM_{10} (weighted average)	25-μg m^{-3} increase of daily $PM_{2.5}$ (weighted average)	Source
Mortality	1.1–8.3% (2.9%)	1.5–9.7% (3.5%)	Based on US EPA (2004)
Respiratory	13.9%	5.5%	
Chronic obstructive pulmonary disease (COPD)	5.5–9.9% (7.0%)	–	
Pneumonia	11.5–16.5% (14.0%)	–	
Cardiovascular	2.2–9.7 (5.5%)	2.6–19.1% (4.6%)	
Cardiorespiratory	–	5.1–6.2% (5.6%)	
Hospital admission			
Respiratory	5.8%	2.8–4.6% (3.78%)	
COPD	5–8.8% (6.8%)		
Pneumonia	2.9–18.6% (8.2%)	10.1–10.5 (10.3%)	
Asthma	9.5–16.2% (12.9%)	1.4–8.7% (2.4%)	
Cardiovascular (>65 years old)	2.7–5.0% (4.0%)	2.9–3.9% (3.4%)	
Heart failure	3.9%	6.8–8% (7.4%)	
Emergency department visit			
Cardiovascular		6.1%	
Asthma	13.2–34.7% (21.1%)		
		1-μg m^{-3} increase of daily $PM_{2.5}$	
Restricted-activity days		1.58%	Ostro and Rothschild (1989)
Minor restricted-activity days		0.82%	

literature. Most published conventional PM studies find significant health effect of PM in terms of mortality and morbidity. If urban PM and wildfire smoke PM exposures have same health effect, we expect to find a significant health effect from wildfire smoke as outdoor PM concentration levels generally increase substantially during the wildfire period. We focus our review to examine the following: (1) if wildfire-specific epidemiology studies found significant health effects associated with wildfire-smoke exposure, and (2) if the findings in wildfire-specific epidemiology studies are consistent with the findings in conventional PM studies. First, we review the findings from the conventional PM studies, followed by wildfire-specific studies, and discuss the potential uniqueness of wildfire-smoke-specific health effects.

Conventional PM studies

Table 2 summarises the results from selected conventional PM studies that were reviewed by the US EPA (2004).[C] Conventional short-term PM studies estimate the marginal effect of PM on adverse health outcomes using a daily time-series model. They generally find a small but statistically significant impact of short-term exposure to PM on the levels of mortality, cardiorespiratory-related hospital admissions and emergency department visits. For example, a 50-μg m^{-3} increase in coarse particles, PM_{10}, is associated with a 2.9% increase of mortality risk, a 5.8% increase of respiratory-related hospital admission and a 21% increase of asthma-related emergency department visits.[D] Also, Ostro and Rothschild (1989) found that a 1-μg m^{-3} increase of fine particles, $PM_{2.5}$, resulted in a 1.58% increase in respiratory-related restricted-activity days and a 0.82% increase in minor restricted-activity days among a sample of 18- to 65-year-olds.[E]

Wildfire health impact studies

This section first summarises the findings from epidemiology studies that examine the health effects of wildfire smoke, and then discusses the consistency with findings from conventional PM studies. Tables 3, 4 and 5 summarise studies that examined the relationship between wildfire smoke and the levels of mortality, hospital admissions, and emergency department visits

[C]US EPA (2004) reviewed the PM-health studies published after 1996 to re-evaluate the relevance of the PM standard established in 1996. Table 2 summarises US studies that met selection criteria outlined in US EPA (2004) and had statistically significant results.

[D]All values are based on the weighted average of mean estimate among selected studies listed in US EPA (2004). The weighted average was calculated by our research group and is detailed in Table 2.

[E]A respiratory-related restricted-activity day is defined as 'any day on which a respondent was forced to alter his or her normal activity and an acute respiratory condition was reported. It includes days of work lost or bed disability as well as more minor restriction'. A minor restricted-activity day is defined as 'a restricted-activity day that does not result in either work loss or bed disability and therefore involves more minor conditions and reductions in activity' (Ostro and Rothschild 1989, p. 239).

Table 3. Wildfire-induced mortality impact summary (ordered by maximum PM_{10} level)

Study	Location	Name and studied period of wildfire	Maximum PM_{10} average level ($\mu g\,m^{-3}$)	Effect on mortality
By historical control method				
Emmanuel (2000)	Singapore	1997 Asian Haze Sep–Oct 1997	100 (monthly)	No change
Phonboon *et al.* (1999)	Hatyai, Thailand	1997 Asian Haze Sep–Oct 1997	218 (daily)	No change
By time-series method				
Cardoso de Mendonça *et al.* (2006)	Amazon, Brazil	Burning of Amazon forest between 1996 and 1999	PM level not available. Substitute with acres of area burned	No change
Vedal and Dutton (2006)	Colorado, USA	Hayman Fire 9 and 18 June 2002	91 (daily) 372 (hourly)	No change
Morgan *et al.* (2010)	Sydney, Australia	32 bushfire days between 1994 and 2002	117 (daily)	No change
Sastry (2002)	Kuala Lumpur, Malaysia	1997 Asian Haze Sep–Oct 1997	423 (daily) Number of low visibility days = 14	Increase of PM by $100\,\mu g\,m^{-3}$ is associated with relative risk of total mortality 1.07 High risk: 65–74-year-old age groups
Sastry (2002)	Kuching, Malaysia	1997 Asian Haze Sep–Oct 1997	Number of low visibility days = 33	Cardiorespiratory related mortality: Significant only for age >75 years

Table 4. Wildfire-induced morbidity impact (hospital admission) summary ordered by maximum PM_{10} level

Respiratory symptoms include general respiratory symptoms, upper respiratory infection or obstructive respiratory disease. NA indicates that estimate is not available

Study	Location	Name of wildfire	Maximum PM_{10} level ($\mu g\,m^{-3}$)	Effect on asthma	Effect on respiratory symptoms	Effect on cardiovascular system
By historical control method						
Phonboon *et al.* (1999)	Thailand	1997 Asian Haze	218 (daily) 69 (monthly)	No change	7% increase (net)	No change
	Thailand (Hatyai)	1997 Asian Haze	218 (daily) 69 (monthly)	No change	49% increase (net, bronchitis and chronic obstructive pulmonary disease)	NA
Duclos *et al.* (1990)	California, USA	Large fire in Aug–Sep 1987	237 (daily)	No change	NA	NA
Delfino *et al.* (2009)	Southern California	2003 southern California fire	>240 (daily $PM_{2.5}$)	26–33% increase during post-wildfire period	48–58% increase during post-wildfire period	6% increase during post-wildfire period
Emmanuel (2000)	Singapore	1997 Asian Haze	100 (monthly)	NA	No change	NA
By time-series method						
Cardoso de Mendonça *et al.* (2004)	Brazil, Amazon	Burning of Amazon forest in 1996–99	Not reported	NA	1 unit of increased burned area is associated with 0.2961 cases of hospitalisation (panel model)	NA
Chen *et al.* (2006)	Brisbane, Australia	Bushfires in July 1997–Dec 2000	60 (daily)	NA	Increased PM_{10} from low to medium, or to high is associated with 9 and 19% increase of admissions respectively	NA
Johnston *et al.* (2007)	Darwin, Australia	Bushfires in 2000, 2004 and 2005	70 (daily)	13% increase associated with 10 $\mu g\,m^{-3}$ increase of PM_{10}	8% increase associated with 10-$\mu g\,m^{-3}$ increase of PM_{10}	No change
Cançado *et al.* (2006)	Piracicaba, Brazil	Sugarcane burning in April 1997–March 1998	87.7 (daily average) during burning period	NA	Children: increased (10% significance level) Elderly: increased	NA
Morgan *et al.* (2010)	Sydney, Australia	32 bushfire days between 1994 and 2002	117 (daily)	5.02% increase of adult asthma associated with 10 $\mu g\,m^{-3}$ increase of PM_{10}	1.24% increase associated with 10-$\mu g\,m^{-3}$ increase of PM_{10}	No change
Phonboon *et al.* (1999)	Thailand (region-wide)	1997 Asian Haze	218 (daily) 69 (monthly)	1 $\mu g\,m^{-3}$ increase of monthly average PM_{10} is associated with 13 excess admissions per month	1-$\mu g\,m^{-3}$ increase of PM_{10} is associated with 85 excess admissions (significant at 10% level)	NA
Delfino *et al.* (2009)	Southern California	2003 Southern California Fire	>240 in San Diego (daily $PM_{2.5}$)	4.8% increase associated with 10 $\mu g\,m^{-3}$ increase of $PM_{2.5}$	2.8% increase associated with 10-$\mu g\,m^{-3}$ increase of $PM_{2.5}$	No change
Shahwahid and Othman (1999)	Malaysia	1997 Asian Haze	Air pollution index = 831	NA	1 unit of increased air pollution index measure is associated with 0.000055 cases per 10 000 people[A]	NA
Mott *et al.* (2005)	Kuching, Malaysia	1997 Asian Haze	852 (daily)	All ages: increased	All ages: increased	No change

[A]The original classification of disease is 'cardiorespiratory illness'.

respectively.[F,G] In these tables, we only include studies that tested for the statistical significance of the results.[H] All the studies use either the time-series method or historical control method. The historical control method is used to evaluate the health effects of a particular event at an aggregate level, such as the total or average levels, by comparing the levels of adverse health outcomes during wildfire period with an appropriate control period.[I] The studies summarised in Tables 3, 4 and 5 are listed by estimation method and the maximum PM level recorded during the wildfire events (from the lowest to the highest).

In contrast to conventional PM studies, wildfire studies were less likely to find a significant positive mortality effect in spite of the substantial increases in PM levels during the wildfire period (Table 3). Only two of the seven studies found a significant mortality effect. [J] Table 4 lists studies that examine the impact of a wildfire event on hospital admissions related to asthma, general respiratory symptoms, and cardiovascular symptoms. Studies found consistent increases of general respiratory-related and asthma-related admissions during wildfire events. Twelve out of the thirteen relevant studies for general respiratory symptoms, and six out of the nine relevant studies for asthma-related admissions found a significant increase during wildfire events. However, only one of the six relevant studies found a significant increase in the number of cardiovascular-related admissions during wildfire events.

Morbidity effects can also be measured by the number of visits to hospital emergency departments.[K] Table 5 summarises studies that examine the impact of wildfire-smoke exposure on the number of asthma, general respiratory symptoms and cardiovascular symptoms-related emergency department visits. A significant increase in the number of emergency department visits was found in seven of the thirteen studies that considered asthma-related effects, nine out of the thirteen studies that considered respiratory-related symptoms, and none of the three studies that considered cardiovascular symptoms.

In summary, significant adverse health effects from wildfire smoke were consistently found in limited health outcomes, such as respiratory-related hospital admissions. The adverse health effects of wildfire smoke on respiratory-related emergency department visit were found but less consistently. Very few studies found a significant positive association between wildfire smoke and mortality or cardio-related morbidity outcomes. Even among the studies that found a significant adverse health effect from wildfire-smoke exposure, the findings are somewhat inconsistent with conventional PM studies. For example, Sastry (2002) found a positive association between levels of PM_{10} and mortality among the elderly in Malaysia during the 1997 South-east Asian Haze. The magnitude of this mortality effect is consistent with the mortality effect found in conventional PM studies. However, the mortality effect was found to be significant only after very high pollution days (daily PM $>200\,\mu g\,m^{-3}$), whereas conventional PM studies find significant mortality effects at lower levels of PM. Johnston *et al.* (2002) also found non-linear health effects associated with PM exposure during wildfire periods.[L]

However, there are studies that show a higher adverse health effect of wildfire smoke than non-wildfire-related PM exposures. For example, Chen *et al.* (2006), Cançado *et al.* (2006) and Delfino *et al.* (2009) found a higher marginal effect of PM on the level of respiratory-related hospital admissions during wildfire event periods than non-wildfire event periods.

Differences between conventional and wildfire PM studies

Contrary to expectations based on the findings from conventional PM studies, significant adverse health effects of wildfire have been found consistently only with the limited respiratory-related morbidity outcomes, and not with mortality or cardio-related morbidity outcomes. However, studies that examined respiratory-related hospital admissions indicated that wildfire-smoke exposure imposed more health risk than conventional PM exposure. Five reasons have been put forth as the possible causes of the differences in observed health effects from conventional PM studies and wildfire smoke studies (Lipsett *et al.* 1994; Kunii *et al.* 2002; Künzli *et al.* 2006; Vedal and Dutton 2006). In this section, we discuss briefly each of the five reasons.

Reason 1. The choice of the statistical model

Conventional PM studies typically use daily time-series models with a long period of observation. This large sample size likely enables researchers to detect a small health effect

[F]In these tables, 'No change' means that there was no statistically significant increase of adverse health outcomes during a wildfire event at the 5% significance level.

[G]Naeher *et al.* (2007) also provide a comprehensive review of epidemiology studies of vegetation fires as well as controlled laboratory studies of wood smoke, health effects of residential wood burning, toxicology, and the chemical and physical nature of wood smoke. Our study expands their epidemiology literature review of mortality, hospital admission and emergency room visits by adding studies that are not included as well as by adding the analytical structure.

[H]The related health studies that were excluded from these tables owing to the lack of statistical tests or the examination of other types of health outcomes include: Frankenberg *et al.* (2005), Kunii *et al.* (2002), Künzli *et al.* (2006), Mott *et al.* (2002), Moore *et al.* (2006), Mott *et al.* (2003), Ovadnevaitė *et al.* (2006), Shusterman *et al.* (1993) and Sorensen *et al.* (1999).

[I]Studies categorised under 'historical control analysis' in this paper include studies that control confounding factors by selecting appropriate reference period using sample design or through econometric modelling.

[J]We count the estimate from a different location or from a different estimation method (historical control or time-series model) in the same study as separate studies in Tables 3–5.

[K]For convenience, we categorise emergency department, health centres, or urgent-care and outpatient facilities as 'emergency departments'.

[L]Kunii *et al.* (2002) also report a weaker mortality impact from wildfire smoke than urban air pollution. They attributed 527 deaths during the 1997 South-east Asian Haze episode to wildfire smoke, while they predicted 15 000 deaths based on the conventional PM-mortality study. Kunii *et al.* (2002) is not included in Table 3 owing to a lack of statistical tests.

Table 5. Wildfire-induced morbidity impact (emergency department (ED) or other outpatient facility visit) summary ordered by maximum PM_{10} level

Respiratory symptoms include general respiratory symptoms, upper respiratory infection, or chronic obstructive pulmonary disease (COPD). NA indicates that estimate is not available. (P) indicates that analysis was conducted using proportion of the number of patients in a certain diagnosed category over total patients

Study	Location	Name of wildfire	Maximum PM_{10} level ($\mu g\,m^{-3}$)	Patient type	Effect on asthma	Effect on respiratory symptoms	Effect on cardiovascular symptoms
By historical control method							
Churches and Corbett (1991)	Sydney, Australia	Burning of firebreaks, May 1991	Nephelometry reading = 7.5 (hourly)	ED	No change	NA	NA
Cooper *et al.* (1994)	Sydney, Australia	Bushfire, Jan 1994	Nephelometry reading = 2.3 (daily)	ED	No change	NA	NA
Smith *et al.* (1996)	Sydney, Australia	Bushfire, Jan 1994	250 (hourly)	ED	No change	No change	NA
Phonboon *et al.* (1999)	Thailand (region-wide)	1997 Asian Haze	218 (daily) 69 (monthly)	Outpatient	NA	8% increase (net)	No change
Davidson *et al.* (2003)	Colorado	Hayman Fire	91 (daily) 372 (hourly)	ED Urgent care Outpatient	No change 22% decrease (P) No change	40% increase (P) No change NA	NA NA NA
Phonboon *et al.* (1999)	Thailand (Hatyai)	1997 Asian Haze	218 (daily) 69 (monthly)	Outpatient	No change	7% increase (net, all respiratory disease)	No change
Duclos *et al.* (1990)	CA, USA	Large fire, Aug–Sep 1987	237 (daily)	ED	40% increase (P)	20–50% increase (P)	NA
Viswanathan *et al.* (2006)	CA, USA	2003 southern CA fire	294 (daily)	ED (chief complaints)	Increased by 5% (P)	Increased by 4–5% (P)	No change
Kene *et al.* (2008)	San Diego, USA	2003 southern CA fire (San Diego)	Not reported	ED (chief complaints)	NA	Increased (P)	NA

By time-series method							
Smith *et al.* (1996)	Sydney, Australia	Jan 1994 (2 weeks)	250 (hourly)	ED	No change	No change	NA
Johnston *et al.* (2002)	Darwin, Australia	Long bushfire between 1 April and 31 October 2000	70 (daily)	ED	Linear model: 18% increase with 10-$\mu g\,m^{-3}$ increase of PM_{10} Categorical model: from base ($<10\,\mu g\,m^{-3}$) to high ($>40\,\mu g\,m^{-3}$) PM_{10} level is associated with 92–156% increase of admissions	NA	NA
Chew *et al.* (1995)	Singapore	1994 forest fire in Indonesia	More than 158 (daily)	ED, children	Significant increase	NA	NA
Phonboon *et al.* (1999)	Thailand (region-wide)	1997 Asian Haze	218 (daily) 69 (monthly)	Outpatient	NA	No change	NA
	Thailand (Hatyai)	1997 Asian Haze	218 (daily) 69 (monthly)	Outpatient	NA	1-$\mu g\,m^{-3}$ increase of daily average PM_{10} is associated with 0.2 excess visits	NA
Emmanuel (2000)	Singapore	1997 Asian Haze	100 (monthly)	Outpatient	19% increase with 100-$\mu g\,m^{-3}$ increase of PM_{10} ($50\,\mu g\,m^{-3}$ to $150\,\mu g\,m^{-3}$)	12% increase with 100-$\mu g\,m^{-3}$ increase of PM_{10} ($50\,\mu g\,m^{-3}$ to $150\,\mu g\,m^{-3}$)	NA
Shahwahid and Othman (1999)	Malaysia	1997 Asian Haze, Aug–Oct 1997	Air pollution index = 831	Outpatient	NA	1 unit of increased air pollution index measure is associated with 0.0125 cases per 10 000 people[A]	NA

[A]The original classification of disease is 'cardiorespiratory illness'.

from short-term PM exposure.[M] In contrast, the historical control method that is often employed in wildfire studies compares aggregate adverse health outcome levels between the study and control periods. This method is not ideal for detecting relatively small health impacts (Vedal and Dutton 2006). However, despite the drawbacks of the historical control method, using a time-series model to evaluate the health effects of wildfire smoke is generally problematic. Smoke from wildfires does not typically last for a long period of time, particularly in the USA. Thus the wildfire event period is too short to have sufficient statistical power for a time-series analysis to be performed.[N]

Reason 2. Urban air pollution and wildfire smoke have chemical differences

Another possible explanation for the observed difference between findings in conventional PM and wildfire smoke studies is the chemical differences between urban air pollution and wildfire smoke. Vedal and Dutton (2006) argue that fossil-fuel combustion usually contains toxic particles such as metal, and may be more hazardous than vegetation burning. However, Wegesser *et al.* (2009) found that PM samples collected during a wildfire event were more toxic than the same amount of PM from normal ambient air.[O]

Reason 3. Non-linearity of the PM dose–response function

Wildfires usually result in large, but short-lived increases in PM levels. In contrast, urban air pollution is often less intense. The US EPA (2004) concludes that the PM dose–response function is linear at low to moderate levels. However, several wildfire studies (Sastry 2002; Chen *et al.* 2006; Martin *et al.* 2007) suggest that the dose–response function is non-linear for higher levels of PM exposure. If this is the case, using a dose–response function derived from low- to moderate-level PM exposure to estimate the health effects of wildfire-smoke exposure would give biased estimates.

Reason 4. Averting behaviour might be different for urban air pollution than for wildfire smoke

Vedal and Dutton (2006) and Kunii *et al.* (2002) suggest the possibility of different averting behaviours among residents in smoke-affected areas during wildfire events as a potential cause of discrepancy between findings of conventional PM studies and wildfire studies. Bresnahan *et al.* (1997) and Künzli *et al.* (2006) found that individuals, particularly those who are sensitive to air pollution, take averting measures when the air pollution level is high. As large wildfire events are highly publicised, and smoke is clearly visible, individuals may take more measures to avoid air pollution from wildfires than from other sources. If that is the case, we would expect fewer observed adverse health outcomes for a given level of PM during a wildfire.[P]

Reason 5. Perceptions about the health risk

People may perceive that air pollution from wildfires imposes a greater health risk than pollution from other sources. Lipsett *et al.* (1994) found that approximately four times as many people without physical evidence of illness visited an emergency department during a large urban fire event than usual. Although the perception of wildfire smoke as a more serious health threat is unlikely to affect the levels of mortality or hospital admissions, it may result in more minor adverse health outcomes such as emergency department visits and perceived symptoms of cardiorespiratory illness.

Future research

There is still significant uncertainty about the health effects of wildfire smoke. Many mortality and cardio-related morbidity studies and some respiratory-related emergency department visit studies found no significant health effect due to wildfire events, in contrast to what would be predicted based on conventional PM studies.

Given the different study design, sample and limited information available about each study, it is difficult to rigorously compare the findings from these two types of studies. More studies are needed that use time-series analysis to understand the potentially unique health effects of wildfire-smoke exposure, as this method allows researchers to compare the health effect of air pollution during wildfire event and non-wildfire event periods using the same study design and sample.[Q] Another benefit of using time-series analysis is that the dose–response function obtained from this method could easily be extrapolated to evaluate the health impact of different wildfire events.

More research is also needed to investigate the impact of wildfire smoke on minor adverse health outcomes that do not require hospital visits, such as coughs and headaches that restrict daily activity. Such studies are sparse in general, and particularly in the area of health impact of wildfire smoke. The per-unit cost of these symptoms may be small, but the potential number of people who experience these health outcomes could be large. As a result, the total cost of minor symptoms may be substantial.

[M] See also Vedal and Dutton (2006) for the discussion of potential bias in conventional time-series PM models. Naeher *et al.* (2007) also provide a discussion about the limitation of several studies with short-observation and few reference periods.

[N] Studies that implemented a time-series approach and found a significant increase in mortality and emergency department visits during a wildfire event tended to involve long observation periods. For example, Sastry (2002) uses 13 to 33 smoke days and Chen *et al.* (2006) use 452 smoke days.

[O] A related issue of this topic is that wildfire smoke has different PM sizes than urban air pollution. According to Ward (1999), wildfire smoke mainly contains $PM_{2.5}$. Some studies suggest that $PM_{2.5}$ is more hazardous than PM_{10} (US EPA 2004). If this is the case, wildfire smoke would be more hazardous than urban air pollution.

[P] Vedal and Dutton (2006) and Kunii *et al.* (2002) provide a discussion of the effectiveness of averting behaviours during wildfire events.

[Q] There are only four hospital admission or mortality studies, Chen *et al.* (2006), Cançado *et al.* (2006), Delfino *et al.* (2009) and Morgan *et al.* (2010), that have taken such an analytical approach.

Finally, no studies have estimated the scale of averting behaviour during a wildfire event. Information on averting behaviour would provide a more complete picture of the health costs of wildfire and might help explain the disparity between conventional PM and wildfire PM studies.

Economic values of health effects

In this section, we review the economic valuation studies related to adverse health outcomes. Adverse health outcomes caused by wildfire smoke impose direct and indirect costs on society. Freeman (2003) divides the types of health costs into four categories: (1) medical costs, (2) labour loss, (3) averting costs, and (4) utility loss (discomfort, suffering). From an economic efficiency standpoint, the total cost associated with health damage should be estimated by the individual's WTP to avoid such health damages. Unfortunately, to our knowledge, there is no economic valuation study that estimated WTP to avoid adverse health outcomes associated with wildfire smoke. Thus we review economic studies that estimated the value of avoiding adverse health outcomes in general.

The widely cited US EPA (1999) report used the health valuation literature to estimate per-unit costs of different adverse health outcomes. In this section, we review the EPA's estimates and the more recent health valuation literature. We also discuss whether the EPA values should be revised based on the new literature or whether there is little difference between new and old estimates of health damages. Table 6 presents a summary of valuation estimates used in the US EPA report (1999).

Mortality valuation

The per-unit cost of premature mortality is measured by the value of a statistical life (VSL), which is society's aggregated willingness to pay to save one anonymous person's life. Viscusi (1992) provides one of the first comprehensive reviews of VSL literature. The US EPA (1999) uses the average value of Viscusi's selected 26 VSL estimates, US$7.6 million,[R] to evaluate the benefit of air-pollution control to prevent premature mortality. Out of 26 VSL estimates, 21 estimates are based on labour-market data, and five estimates are based on survey studies. Later, the US EPA revised the VSL to $6.8 million based solely on the labour-market studies (US EPA 2005).

Recent research suggests that labour-market studies used in the US EPA (1999, 2005) analysis overestimate VSL owing to incorrect model specifications. Kniesner *et al.* (2010) and Kochi (2006) correct this bias and report VSL estimates of $8 million–$14 million and $2 million respectively. A recent survey-based study in the USA found that the mean VSL is between $1.8 and $5.7 million (Alberini *et al.* 2004). Taken as a whole, the recent literature suggests that VSL may range between $2 million and $14 million.

Morbidity valuation

Estimating per-unit cost of morbidity is more complex than estimating per-unit cost of premature mortality, as the severity and duration of adverse health outcomes varies (for a detailed review of morbidity valuation methodologies, see Tolley *et al.* (1994) and Dickie and Gerking (2002)). The US EPA (1999) estimates morbidity costs of air pollution based on existing literature using the COI method or the contingent valuation (CV) method. The COI method is often used to value the cost of health outcomes that involve some type of medical care, such as hospitalisation or emergency department visits, and only includes the direct expenses associated with illness, such as medical costs and lost wages.[S] The CV method uses surveys to measure individuals' WTP to prevent an adverse health outcome, which includes the utility loss from illness and averting costs, as well as direct costs.

A full economic accounting of morbidity costs should be in terms of WTP, but COI is easier to measure. Consequently, a common practice is to convert individual COI to WTP using a WTP/COI ratio. Chestnut *et al.* (1999) provide a summary of four studies that estimated WTP as well as COI using the same study population and the same health endpoint.[T] When a COI estimate accounts for the cost incurred by the individual and a third party, such as a health insurance company, it is called a social COI and the estimated WTP/social COI ratio is between 1.3 to 2.4 for asthma symptoms, cataract and angina symptoms. Chestnut *et al.* (1999) recommend a conservative WTP/social COI ratio of 2.0 for non-fatal morbidity treatment except for cancers, and 1.5 for non-fatal cancer treatment.[U]

Several studies have estimated WTP to avoid relatively minor symptoms, such as acute cardiorespiratory symptoms. Dickie and Gerking (2002) and Dickie and Messman (2004) provided a list of studies and estimates. As Dickie and Messman (2004) noted, WTP values used by the US EPA (1999) are generally lower than more recently estimated values (selected results from the Dickie and Messman (2004) study are presented

[R] All dollar values are in 2007 US dollar values converted using the Consumer Price Index.

[S] Conventional Cost of Illness (COI) generally estimates only medical costs and lost wages during a hospital stay or hospital visit. However, a recent study by Chestnut *et al.* (2006) found that the time lost during recovery from a hospital stay is also an important source of cost, which increases conventional COI estimates by 9 to 32%. The COI during this recovery period includes: additional medical costs, lost wages, and lost productive and recreational activities. The COI per hospitalisation also depends on the age of the patient and category of illness. The elderly (over 65 years old) have lower COI than younger individuals owing to the smaller value of lost work days.

[T] These four studies either directly elicited the dollar value of willingness to pay (WTP) and cost of illness (COI), or they asked respondents to rate the share of COI components as a share of perceived total health cost. For example, Chestnut *et al.* (1988) asked respondent to rate each component of WTP associated with an increase in angina episode with a scale of 'bothersomeness'. WTP components include COI consequences (medical costs and labour loss), and non-COI consequences, such as less leisure and more concern.

[U] If the cost of illness (COI) estimate only accounts for the cost incurred by an individual (called individual COI), the WTP/individual COI ratio (WTP, willingness to pay) could be significantly higher than the WTP/social COI ratio. It is important to remember that WTP/COI ratios may vary significantly across different health outcomes, as Adamowicz *et al.* (2004) indicated.

Table 6. Per-unit economic value used in US EPA (1999)
Created from table 6-1, p. 70 and table H-3, pp. H-21–H-26, US EPA (1999). Monetary value adjusted to year 2007 level

	US EPA value (US$ 2007)	Dickie and Messman (2004)
Mortality	$7 600 000	
Hospital admissions		
All respiratory	$10 971	
All cardiovascular	$15 105	
Emergency department visits for asthma	$308	
Respiratory illness and symptoms		
Acute bronchitis	$71	$202 (adult) $380 (child)
Asthma attack or moderate or worse asthma day	$50	
Acute respiratory symptoms	$28	1-day symptom $90 (adult) $190 (child)
Upper respiratory symptoms	$30	
Lower respiratory symptoms	$19	
Shortness of breath, chest tightness or wheeze	$8	1-day shortness of breath $190 (child)
Work days loss	$131	
Mild restricted-activity days	$60	

in Table 6). For example, the US EPA uses $71 per acute bronchitis case, whereas Dickie and Messman (2004) estimated the median WTP to prevent a 6-day-long acute bronchitis case as $202 for an adult.[V] Similarly, the US EPA uses $28 per day for acute respiratory symptoms, whereas Dickie and Messman (2004) estimated $90 a day for an adult.

It is not clear why more recent studies report generally higher WTP estimates than older studies. However, recent studies incorporate improvements in non-market valuation methods and so may warrant more weight than older studies. Finally, many studies (Liu *et al.* 2000; Navrud 2001; Dickie and Messman 2004) consistently find that WTP estimates for the prevention of children's morbidity are substantially higher than the WTP estimates for the prevention of adults' morbidity. This underlines the importance of valuing adults' and children's morbidity impacts separately.

Wildfire policy considerations

Quantifying the health effects of wildfire smoke

If wildfire-management decisions are going to take into consideration the potential mortality impacts of wildfire smoke, we do not recommend using results from conventional PM epidemiology studies to estimate the mortality effects of wildfire. Although conventional PM studies generally show a statistically significant mortality risk of short-term PM exposure, the majority of wildfire-PM studies do not. Extrapolating mortality impact results from conventional PM studies to wildfire may substantially overestimate mortality-related costs. If a wildfire is of short duration, or results in only moderate increases in PM levels, then analysts might consider assuming no mortality effect, while noting that this assumption may underestimate true cost. If mortality effects are to be included, it should be noted that there is still great uncertainty in the VSL estimates.

Consideration of respiratory-related morbidity effects in wildfire-management decisions based on results of conventional PM studies might be reasonable if wildfire-specific study results are not available. However, this recommendation comes with the caveat that the health effects of urban air pollution may be somewhat different from wildfire smoke. The cost of severe morbidity that involves major medical care could be estimated based on the social COI method. To convert social COI to WTP, a WTP/social COI ratio of 2 is generally accepted. Dickie and Messman (2004) is a good source to find WTP to avoid less severe respiratory symptoms, as they used a relatively large USA sample. Finally, we again emphasise the importance of accounting for adults and children separately.

Consideration of health effects of wildfire smoke in policy options

During a wildfire event, there are two main ways to reduce the adverse health impacts of wildfire smoke: wildfire suppression to reduce PM emissions or temporarily moving susceptible people away from smoke-affected areas. The appropriate response depends on the impact wildfire smoke is likely to have on air quality, the number of people who will be exposed to the smoke, and the likely efficacy of wildfire suppression actions. For example, in the introduction, we noted that improving air quality was a major reason that the USDA Forest Service decided to suppress all wildfires in California during the summer of 2008. In the weeks leading up this decision, wildfires had a significant impact on air quality.[W] As total health costs are

[V] Dickie and Messman (2004) found that the average acute bronchitis symptom lasts an average of 7 days among the sample.

[W] The US EPA classifies wildfires as exceptional events and, therefore, does not penalise states if wildfire smoke causes federal air-quality standards to be breached.

determined largely by the magnitude of the population exposed to the smoke, all else equal, deploying a higher suppression effort is more likely warranted when wildfire smoke drifts into highly populated areas. Fire managers need to recognise that air pollution does not affect all segments of the population equally. People with pre-existing respiratory problems are far more likely to suffer adverse health outcomes. Therefore, moving a relatively small fraction of the population away from smoke-affected areas may significantly reduce the health impacts of a wildfire. However, if a wildfire affects a large metropolitan area, then moving even a small fraction of the affected population may be difficult and expensive. In such an urban area, higher levels of wildfire suppression may be cost-effective when compared with the cost of relocating thousands of people.

Another important factor to consider in wildfire smoke and suppression decisions is the marginal impact of suppression on air quality. During periods of severe fire weather, suppression may have little impact of fire behaviour. Under these circumstances, increased suppression effort would have little effect on PM levels and could not be justified on the basis of reducing health-related costs.

Reducing PM from wildfires and reducing conventional PM pollution differ in a crucial way. If urban PM is reduced by closing down a coal-fired power plant permanently, for example, then this does not increase the probability of PM emissions in the future (indeed, it does the opposite). However, suppressing wildfires to reduce PM may increase the potential for future PM emissions if a wildfire occurs years later. That is, if a wildfire is successfully suppressed, then the fuel that would have burned remains in the forest. This increased fuel load means that, in the future, severe wildfires – which emit more PM – are more likely. Wildfire suppression does not eliminate PM pollution; it may shift it into the future.[X] Thus true wildfire smoke prevention requires long-term fuel reduction.

The literature reviewed in this article suggests that prescribed burning, which reduces the probability of future severe wildfires, should result in a net reduction in health damages relative to wildfires for two reasons. First, prescribed fire generally burns less intensely, resulting in lower emissions than wildfires. If the dose–response function is non-linear, there are likely to be substantially smaller health effects at these lower PM concentrations. Second, prescribed burning can be conducted when the winds will not direct the smoke into densely populated areas, again minimising the health damages while reducing fuel loads and future PM emissions.

The wildfire–air pollution relationship also strengthens the rationale for mechanical fuel reduction. Forest thinning can be done to reduce the emissions per acre that would result if a wildfire were to occur. Thus health costs avoided should be included as one of the benefits of mechanical fuel reduction. Although mechanical fuel reduction costs more per acre compared with prescribed burning, in densely populated wildfire-prone areas, the total economic costs, including health costs, could be lower for mechanical fuel reduction than prescribed burning.

Conclusions

In this paper, we summarise the available literature related to economic analysis of adverse health impacts from wildfire smoke, identify the key issues to be investigated in the future to improve this research area, and discuss how concerns about the health effects of smoke could be considered in wildfire management decisions. We find that the available literature on the economic analysis of adverse health impacts from wildfire smoke and wildfire-specific epidemiology studies are still limited. We identify several potentially productive research areas. First, investigating the unique health effects of wildfire smoke compared with conventional air pollution would be helpful to better quantify the adverse health impacts of wildfire smoke, as well as to determine whether it is appropriate to extrapolate from existing conventional air pollution dose–response functions. Second, quantifying the relatively minor adverse health impacts that do not require major medical care could be very important as the total cost associated with these health outcomes could be substantial. Last, understanding averting behaviour during wildfire events could be important as the opportunity costs of avoiding wildfire smoke through evacuation, avoidance of outdoor activity, or other preventive measures may be substantial.

The health-related cost of wildfire-smoke exposure should undoubtedly be an important consideration for wildfire management policy. However, we still have limited knowledge about the nature of this cost. We encourage more research as such information will have increasing importance as the number and scale of future wildfire events is predicted to increase.

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[X]Shifting the adverse health effects of wildfire smoke into the future would provide some benefit, as these costs would be discounted.

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Manuscript received 16 July 2009, accepted 8 March 2010

Health Effects of the 2003 Southern California Wildfires on Children

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Rationale: **In late October 2003, Southern California wildfires burned more than 3,000 km^2. The wildfires produced heavy smoke that affected several communities participating in the University of Southern California Children's Health Study (CHS).**

Objectives: **To study the acute effects of fire smoke on the health of CHS participants.**

Methods: **A questionnaire was used to assess smoke exposure and occurrence of symptoms among CHS high-school students (n = 873; age, 17–18 yr) and elementary-school children (n = 5,551; age, 6–7 yr), in a total of 16 communities. Estimates of particulate matter (PM_{10}) concentrations during the 5 d with the highest fire activity were used to characterize community smoke level.**

Main Results: **All symptoms (nose, eyes, and throat irritations; cough; bronchitis; cold; wheezing; asthma attacks), medication usage, and physician visits were associated with individually reported exposure differences within communities. Risks increased monotonically with the number of reported smoky days. For most outcomes, reporting rates between communities were also associated with the fire-related PM_{10} levels. Associations tended to be strongest among those without asthma. Individuals with asthma were more likely to take preventive action, such as wearing masks or staying indoors during the fire.**

Conclusions: **Exposure to wildfire smoke was associated with increased eye and respiratory symptoms, medication use, and physician visits.**

Keywords: air pollution; asthma; sore throat; wheezing

AT A GLANCE COMMENTARY

Scientific Knowledge on the Subject

Adverse effects of fire smoke are known, but results in children are inconsistent due to a lack of large population-based studies.

What This Study Adds to the Field

The study quantifies effects of fire smoke on eye, upper, and lower respiratory symptoms. It gives first evidence of benefits of preventive actions.

In October 2003, a series of devastating wildfires burned in Southern California. The hot and dry Santa Ana winds encouraged the spread of fires across several locations to the north, east, and south of the Los Angeles metropolitan area, and dense plumes of smoke dominated much of the area for several days. Local air-quality monitors recorded hourly particulate matter concentrations approaching 1,000 μg/m^3 particles of aerodynamic diameter up to 10 μm (PM_{10}); these levels were 10 to 20 times the typically observed ambient levels (1, 2). The fires occurred over a wide geographic area, over a 480-km swath affecting six Southern California counties (Ventura, Los Angeles, San Bernardino, Riverside, Orange, and San Diego). The fires consumed more than 3,100 km^2 (750,000 acres) and destroyed 3,640 homes, 33 commercial properties, and 1,141 other structures (including several regional air-monitoring stations).

Most wildfire investigations focus on short-term changes in hospital admissions or on segments of the population believed to be especially sensitive to respiratory stress, such as patients with chronic obstructive lung disease (COPD) or asthma, or on those individuals especially prone to exposure, such as firefighters (3, 4). Medical surveillance data from San Diego County revealed significant increases in hospital emergency room visits for asthma, respiratory problems, and eye irritation during the 2003 fire period (5). Population-based investigations of the acute respiratory health effects of fire smoke on children's health have been limited and based on small samples. The lack of data may be contributed, in part, to the logistical challenge of implementing population-based studies during fire emergencies. Australian researchers investigated the health effects of bush fires and reported increased evening wet cough among a panel of 32 children with asthma but nonsignificant results for wheeze and β-agonist use (6). PM_{10} peaks were much lower (130 μg/m^3) than in the 2003 California fires. Associations of fire smoke and evening peak flow were also not conclusive (7). In Asia, the large 1997 fires resulted in an increased use of health services (4) and higher mortality rates both among infants and adults (8).

The Southern California fires offered a unique opportunity to conduct a population-based, large-scale investigation of the health consequences of the smoke from wildfires on children's health. The region affected by the wildfires included several communities participating in a long-term ongoing health study of California schoolchildren, the University of Southern California Children's Health Study (CHS) (9, 10). The goal of the CHS is to understand the contribution of long-term or lifetime exposure to ambient air pollution to children's respiratory health (9–11). Initial cohorts of children were recruited (1993 and 1996) from 12 communities across six Southern California counties. In 2002, an additional cohort of kindergarten and first-grade children

(*Received in original form April 12, 2006; accepted in final form September 10, 2006*)

Supported by the South Coast Air Quality Management District (AQMD contracts 04073 and 04182), the National Institute of Environmental Health Sciences (5P01ES11627, 3P01ES011627-03S2, 5P30 ES07048), and the Hastings Foundation.

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This article has an online supplement, which is accessible from this issue's table of contents at www.atsjournals.org

Am J Respir Crit Care Med Vol 174. pp 1221–1228, 2006
Originally Published in Press as DOI: 10.1164/rccm.200604-519OC on August 31, 2006
Internet address: www.atsjournals.org

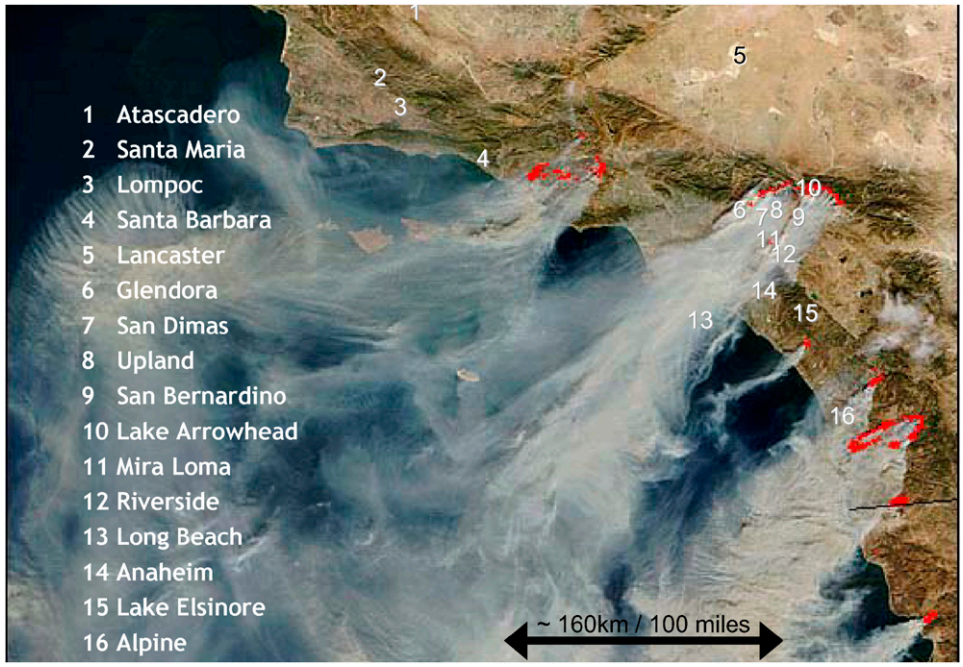

Figure 1. Satellite image of Southern California taken on October 24, 2003, showing the smoke plumes from numerous fires. Locations of the 16 Children's Health Study communities participating in the fire study are *highlighted*. Image courtesy of MODIS Rapid Response Project at NASA/GSFC.

(aged 5 to 6 yr) were enrolled from 13 partly overlapping communities (10). At least 12 of the 16 cohort communities were either directly affected by the fire (i.e., the community was the site of fire damage and human evacuation) or indirectly affected (by dense smoke covering the community). Figure 1 presents the cohort study towns in a satellite image of the wildfire areas taken in late October 2003.

To assess the effects of the wildfires, we implemented a questionnaire-based investigation of fire smoke exposure and symptoms for two of the existing and accessible study cohorts (12th-grade high-school students, and first- and second-grade elementary-school children). The availability of extensive sociodemographic and health data among this large sample of children offered a unique opportunity to efficiently investigate and quantify the health consequences of fire smoke exposure in both children with asthma and nonasthmatic children. Some of the results of this study have been previously reported in the form of an abstract (12).

METHODS

The CHS methods have been published elsewhere. Details about the fire study are provided online. In brief, the CHS consists of repeated annual health assessments to monitor the course of respiratory health. The fire questionnaire study focused on participants of two ongoing CHS cohorts, including one cohort of high-school students (17 to 18 yr old during the fire), originally enrolled in 1996, and a cohort of elementary-school children (aged 6 to 7 yr), recruited in 2002. The older student cohort included high schools from the 12 (9, 10) original CHS communities and the elementary-school cohort involved 13 communities (nine of which were the same) (10). The study protocol was approved by the institutional review board for human studies at the University of Southern California, and written, informed consent was provided by participating students and a parent or legal guardian of minors.

The 2003 Southern California fires peaked between October 20 and November 2. During November–December 2003, the high-school students and the parents of the elementary-school children received the fire questionnaire by mail (*see* online supplement) and/or during the first 6 mo in 2004 as an annex activity of the ongoing CHS. The reporting period referred to the "two weeks of the October 2003 fire period." Although the first page of the fire questionnaire asked about health-related problems, the second page referred to exposure to fire smoke and personal measures taken to modify this exposure (including evacuation, wearing of masks, reduction in time spent outdoors, and changes in physical activity). To quantify exposure duration, questionnaire response categories included the following: "not at all," "1–2 d," "3–5 d," "6–10 d," or "all days" (i.e., up to 2 wk).

Objective smoke measurements (i.e., PM_{10} [U.S. Environmental Protection Agency–approved Federal Reference Methods to quantify PM_{10}]) were available only on the community rather than on an individual level. PM_{10} was the strongest marker of fire smoke pollution (1, 2). High concentration periods lasted approximately 5 d; thus, we used the 5-d mean PM_{10} level to characterize fire smoke. Missing air-quality data required estimation procedures of the 5-d average PM_{10}. Five-day average PM_{10} concentrations were estimated for 5 of the 16 communities. San Dimas, Glendora, and Anaheim had all 5 d (October 24–28) estimated, and San Bernardino had 4 d (October 25–28) estimated. Because Alpine was directly affected by the fires from October 26 to 28, PM_{10} concentrations at Alpine were averaged over the 3 fire days with 2 d estimated (October 27–28). For more details, *see* text and Table E1 in the online supplement and Reference 1.

Statistical Analyses

To investigate the association between fire smoke exposure and symptoms, we chose multilevel approaches to distinguish within-community differences in exposure from the contrasts between communities. We used the reported "smell of fire smoke indoors" as the primary measure of exposure. We created two components of reported exposure response. The first was a between-community measure, derived from the community-specific mean response. The second was a within-community response, created by subtracting the community mean from the individual response, using a mixed-effects model with a logistic link. As described in the online supplement, the five exposure categories were combined into three levels, providing comparison across the following groups of "fire smoke smelled": no fire smoke, fire smoke smelled 1 to 5 d, and fire smoke smelled 6 d or more.

The first set of analyses was based on the reported levels of fire smoke smelled at home indoors, reflecting the change in symptoms due to an increase in the duration of (perceived) fire smell. In a second set of models, we replaced the reported community mean fire smoke response with the ambient 5-d mean PM_{10}. Thus, these between-community estimates reflected the change in symptoms for a change in ambient PM_{10} during the 5 most extreme days of fire smoke.

TABLE 1. HIGH-SCHOOL STUDENTS AND PARENTS (ELEMENTARY-SCHOOL CHILDREN) RESPONDING TO THE FIRE QUESTIONNAIRE WITHIN 8 WEEKS OF THE FIRE (EARLY RESPONSE, NOVEMBER/DECEMBER 2003) AND TOTAL RESPONSE (INCLUDING JANUARY TO JUNE 2004 DURING CHILDREN'S HEALTH STUDY HEALTH VISITS)

	High-School Students (17–18 yr old)			Elementary-School Children (6–7 yr old)		
Community	Baseline Population	Early Response (2003), n (%)	Total Response (2003/04), n (%)	Baseline Population	Early Response (2003), n (%)	Total Response (2003/04), n(%)
Alpine	75	35 (46.4)	70 (93.3)	397	165 (41.5)	299 (75.3)
Anaheim	—	—	—	419	90 (21.4)	251 (59.9)
Atascadero	74	68 (91.8)	70 (94.5)	—	—	—
Glendora	—	—	—	466	228 (48.9)	374 (80.2)
Lake Arrowhead	70	28 (40.5)	67 (95.7)	401	163 (40.6)	301 (75.0)
Lake Elsinore	66	23 (35.3)	62 (93.9)	386	254 (65.8)	254 (65.8)
Lancaster	64	27 (41.5)	61 (95.3)	—	—	—
Lompoc	80	32 (40.0)	78 (97.5)	—	—	—
Long Beach	85	35 (41.6)	79 (92.9)	366	87 (23.7)	239 (65.3)
Mira Loma	64	51 (78.4)	62 (96.8)	510	280 (54.9)	286 (56.0)
Riverside	69	53 (76.8)	67 (97.1)	439	150 (34.1)	285 (64.9)
San Bernardino	—	—	—	410	94 (22.9)	255 (62.1)
San Dimas	74	39 (52.7)	74 (100)	393	169 (43.0)	213 (54.1)
Santa Barbara	—	—	—	468	166 (35.4)	360 (76.9)
Santa Maria	66	25 (39.0)	62 (93.9)	470	125 (26.5)	311 (66.1)
Upland	86	39 (46.4)	82 (95.3)	426	198 (46.4)	347 (81.4)
Total	873 (100)	455 (52.4)	834 (95.5)	5,551 (100)	2,169 (39.0)	3,775 (68.0)

The final models included those covariates that were independent predictors and/or confounders in the models of at least one symptom, namely sex, ethnicity, educational level of parents, asthma status before the fire (physician-diagnosed asthma), and cohort (high-school vs. elementary-school cohort). A p value of 0.05 or less was considered statistically significant. In addition, all analyses were stratified by asthma status. All analyses were conducted with the statistical software SAS/STAT, version 9 (2002; SAS Institute, Cary, NC).

RESULTS

Table 1 summarizes the study populations and participation. High-school students' participation rates during the first 8 wk (2003) reached 52.4%, whereas only 39.0% of the parents (younger cohort) returned the mail-in questionnaire. The extended distribution of the fire questionnaire during 2004 strongly improved response rates, ultimately reaching 95.5% in the older and 68.0% in the younger cohort.

Table 2 summarizes the distribution of reported fire exposure and the ambient levels of measured or estimated PM_{10} (*see* Methods). Both the subjective and objective measures of fire smoke showed that communities not directly affected by local fires suffered substantial smoke exposure (e.g., Mira Loma, Riverside, and Anaheim).

Table 3 summarizes the prevalence of the reported outcomes, by cohort and asthma status. As expected, prevalence rates were much higher among individuals with asthma. Dry cough, medication, and physician visits were more frequently reported by parents of elementary-school children, whereas high-school students were more likely to report eye symptoms. Home loss due to fire was reported by 35 (0.75%) study participants. In Alpine and

TABLE 2. PREVALENCE OF REPORTED SMELL (%) OF FIRE SMOKE INDOORS (BY COHORT), 5-DAY MEAN PM_{10} DURING THE FIRE PERIOD,* AND LONG-TERM AMBIENT PM_{10} IN THE 16 COMMUNITIES

	High-School Students (*n = 834*)				Elementary-School Children (*n = 3,775*)				PM_{10} in $\mu g/m^3$	
Town	Not at All	1–2 d	3–5 d	⩾ 6 d	Not at All	1–2 d	3–5 d	⩾ 6 d	5-d Mean (fire period)	1992–2003, Mean
Alpine	27.1	21.4	20.0	31.4	21.1	23.8	19.1	33.6	201	25.3
Anaheim	—	—	—	—	64.4	10.2	6.4	13.6	132	36.9
Atascadero	97.1	1.4	0	0	—	—	—	—	52	21.3
Glendora	—	—	—	—	54.4	20.9	8.4	13.9	158	32.5
Lake Arrowhead	63.6	14.6	12.1	10.6	57.7	20.1	10.4	9.4	172	19.8
Lake Elsinor	64.5	17.7	3.2	9.7	59.0	16.1	10.8	11.7	104	35.6
Lancaster	45.9	29.5	11.5	9.8	—	—	—	—	45	29.0
Lompoc	88.5	2.6	1.3	5.1	—	—	—	—	32	14.4
Long Beach	63.3	17.7	11.4	5.1	62.2	15.9	5.2	11.6	135	36.8
Mira Loma	54.1	16.4	13.1	16.4	47.1	13.2	13.2	23.2	250	66.3
Riverside	52.2	13.4	14.9	16.4	47.1	16.4	12.9	16.4	172	42.3
San Bernardino	—	—	—	—	24.2	15.3	13.3	41.1	199	51.0
San Dimas	55.6	19.4	11.1	12.5	45.5	15.8	18.7	16.8	191	36.7
Santa Barbara	—	—	—	—	80.3	9.7	2.9	2.6	30	28.2
Santa Maria	90.3	6.5	0	1.6	90.8	2.6	0.3	1.6	51	22.0
Upland	26.6	7.6	24.1	39.3	20.1	18.6	20.4	39.0	252	40.7

* Rows do not add up to 100% due to rounding and a few "don't know" answers.

TABLE 3. PREVALENCE (%) OF SYMPTOMS REPORTED FOR THE FIRE PERIOD, BY STUDY COHORT AND BY ASTHMA STATUS (BASED ON THE LAST CHILDREN'S HEALTH STUDY QUESTIONNAIRE AVAILABLE PRIOR TO THE FIRE)

	High-School Students (*n = 834*)			Elementary-School Children (*n = 3,775*)			Both Cohorts (*n = 4,609*)		
Symptom	No Asthma (*n = 616*)	Asthma (*n = 218*)	All	No Asthma (*n = 3,287*)	Asthma (*n = 488*)	All	No Asthma (*n = 3,903*)	Asthma (*n = 706*)	All
Itchy/watery eyes	41.1	47.7	42.8	29.9	51.6	32.8	31.7	50.4	34.6
Irritated eyes	41.6	50.9	44.0	30.9	51.8	33.6	32.6	51.5	35.5
Sneezing/blocked nose	38.6	49.3	41.4	37.6	65.8	41.3	37.7	60.7	41.3
Cold	26.0	27.5	26.4	24.4	33.9	25.7	24.7	31.9	25.8
Sore throat	32.3	41.3	34.6	30.8	42.5	32.3	31.0	42.1	32.7
Dry cough at night	14.3	22.5	16.4	24.1	49.3	27.4	22.6	41.0	25.4
Dry cough first in morning	13.0	19.3	14.6	20.7	43.5	23.7	19.5	36.0	22.0
Dry cough other times	17.5	28.4	20.3	19.3	43.8	22.4	19.0	39.0	22.0
Wet cough	13.7	16.2	14.5	12.9	24.0	14.3	13.0	21.6	14.3
Wheeze/general	7.3	18.9	10.4	6.8	39.9	11.0	6.8	33.3	10.9
Wheeze/disturbed sleep	2.3	7.0	3.5	3.5	21.9	5.8	3.3	17.3	5.4
Wheeze/ limited speech	1.0	1.9	1.2	0.9	4.3	1.3	0.9	3.5	1.3
Asthma attack	1.0	11.0	3.6	1.3	17.4	3.3	1.2	15.4	3.4
Bronchitis	3.3	2.8	3.1	3.7	9.9	4.5	3.6	7.7	4.2
Medication*	12.9	23.6	15.7	23.7	50.6	27.2	22.0	42.3	25.1
Visit a doctor*	5.6	9.7	6.7	9.8	22.0	11.4	9.2	18.2	10.6
Missed school*	9.8	14.7	11.1	11.8	24.8	13.5	11.5	21.7	13.1

* For above problems.

Lake Arrowhead, more than 3% of study participants lost their homes (n = 15 and 10, respectively).

The main results are summarized in Table 4. Six or more days of fire smell indoors was significantly associated with all outcomes, and the smaller risk estimates for 1 to 5 d of exposure reached statistical significance in all but two outcomes (asthma attacks and bronchitis). Having fire smoke smell indoors for more than 6 d was associated with more than fourfold higher rates of eye symptoms, approximately threefold increased rates of dry cough and sneezing, and more than twofold higher rates of cold, sore throat, wet cough, medication use, physician visits, and missed school due to symptoms. The three types of wheezing (general, sleep-disturbing, and speech-limiting) occurred 3.5, 4.9, and 5.5 times more often, respectively, among those with 6 or more days of fire smell indoors. Asthma attacks increased 63%. The trend across the different levels of fire smell duration was highly significant for all outcomes except for asthma attacks ($p = 0.12$).

The between-community comparisons were analyzed with two different metrics, namely PM_{10} and the community mean

TABLE 4. MAIN EFFECT OF FIRE SMOKE ON ALL OUTCOMES (ODDS RATIOS AND 95% CONFIDENCE INTERVALS)

	Within-Community (reported)				Between-Community (PM_{10})	
Symptom	OR 1–5 d	95% CI	OR ⩾ 6 d	95% CI	OR 210	95% CI
Itchy/watery eyes	**2.26**	1.90–2.68	**4.11**	3.36–5.02	**2.97**	2.00–4.40
Irritated eyes	**2.38**	2.01–2.82	**4.42**	3.61–5.41	**3.13**	2.15–4.55
Sneezing; runny/blocked nose	**1.98**	1.68–2.33	**2.79**	2.30–3.39	**1.94**	1.44–2.61
Cold	**1.50**	1.25–1.81	**2.13**	1.73–2.63	0.92	0.67–1.25
Sore throat	**1.81**	1.53–2.14	**2.50**	2.05–3.05	**1.79**	1.45–2.20
Dry cough at night	**2.25**	1.87–2.71	**3.35**	2.71–4.15	**1.92**	1.38–2.67
Dry cough first thing morning	**2.24**	1.85–2.72	**2.91**	2.33–3.63	**1.93**	1.36–2.73
Dry cough other times	**2.67**	2.20–3.24	**3.27**	2.61–4.09	**2.49**	1.86–3.33
Wet cough	**1.42**	1.13–1.79	**2.15**	1.67–2.77	1.01	0.72–1.41
Wheezing or whistling	**2.15**	1.63–2.83	**3.53**	2.62–4.75	1.37	0.86–2.20
Wheeze/disturbed sleep	**2.29**	1.56–3.37	**4.94**	3.33–7.33	0.89	0.56–1.42
Wheeze/limited speech	**2.23**	1.03–4.83	**5.49**	2.63–11.48	0.78	0.29–2.10
Asthma attack	1.32	0.84–2.07	**1.63**	1.00–2.67	1.03	0.58–1.80
Bronchitis	1.33	0.87–2.02	**2.23**	1.45–3.43	0.79	0.39–1.59
Medication for above problems	**1.82**	1.51–2.19	**2.33**	1.89–2.88	**1.38**	1.03–1.84
Visit a doctor for above problems	**1.33**	1.02–1.74	**2.03**	1.53–2.71	0.81	0.59–1.12
Missed school for above problems	**1.59**	1.25–2.02	**2.24**	1.72–2.91	0.96	0.72–1.27

Definition of abbreviations: CI = confidence interval; OR = odds ratio.

Within-community ORs are based on individually reported smell of fire smoke indoors (no fire smell = reference, OR = 1.0; not shown; 1-5 d; and ⩾ 6 d of fire smell). Between-community ORs show the associations scaled to the contrast in PM_{10} between the communities with the highest and lowest levels, respectively ($\sim$ 210 vs. 30 $\mu g/m^3$). Models are adjusted for baseline asthma, ethnicity, parental education, and study cohort. Statistically significant estimates ($p \leqslant 0.05$) are in bold type.

response to the fire smell question. Results are presented for the former only (Table 4) as they were similar for both metrics. The community mean of the reported level of fire smoke indoors and the estimates of the 5-d mean PM_{10} were highly correlated ($r = 0.81$) in both high-school and elementary-school students. Comparing highest with lowest community exposures, the between-community results were statistically significant and similar for both metrics in case of dry cough, eye, nose, and throat symptoms, as well as for medication. Eye symptoms were approximately three times as frequent in the communities most affected by fires as compared with lesser-affected communities. The between-community estimate for wheezing was significant only with the mean reported smoke (odds ratio [OR], 1.37 per unit change) but not with PM_{10} (Table 4). The other outcomes were not significantly associated with either community-level metric.

The model presented in Table 4 also adjusted for asthma status before the 2003 fire period ("physician-diagnosed asthma"). Therefore, the model also estimates the contribution of physician-diagnosed asthma to symptom frequency in the end of October 2003, independent of the fire smoke. We present these effects (ORs) in Table E3 to highlight the much higher symptom rates among children with asthma (*see also* Table 3). Children with asthma were two to three times more likely to report symptoms than nonasthmatic children. Thus, the effect of having asthma was similar to the effects of fire smoke. In the case of wheezing, asthma status was more strongly related to the symptom (OR = 7.4; *see* Table E3.) than fire smoke (OR = 3.5; *see* Table 4).

The effect of fire smoke was, however, not restricted to children with asthma. Results of Table 4, stratified by asthma status, are presented in the online supplement (Tables E4 and E5). In fact, among nonasthmatic children, coefficients were either very similar or stronger (wheezing) than in children with asthma (n = 706) in whom point estimates tended to be smaller and not statistically significant for speech-limiting wheezing, asthma attacks, bronchitis, cold, wet cough, physician visits, and missed school. The between-community estimates followed a similar pattern as in nonasthmatic children, with significant associations among nine questionnaire items.

To evaluate the joint effects of fire and asthma status on reported symptoms, we examined five indicator variables for the combinations of fire smell (none, 1–5 d, ⩾ 6 d) and asthma status (yes/no) using nonasthmatic children without fire exposure as the reference group. Figure 2 presents the effects of fire smoke among children with and without asthma.

Preventive Action and Fire-related Health Outcomes

We distinguished those who took action such as wearing masks, spending less time outdoors, or using air conditioners for at least 1 to 2 d from those not reporting preventive strategies. Those taking action also reported higher rates in almost all outcomes, and in many cases, these differences were statistically significant. For example, those reporting "wearing a mask" had symptom rates more than twice as high as those not using masks, whereas those reporting the use of air conditioners or spending "less time outdoors" during the fire had 1.2- to 1.6-fold rates in symptoms. Of particular interest is the interaction between preventive actions and reported duration of fire smell indoors (*see* Table 5). As a general pattern, we observed larger risk gradients related to fire smoke among those who did not take preventive action as compared with those who did. The interaction term reached statistical significance in several models (*see* Table 5). Compared with those who reported no fire smell, subjects with 1 to 5 d of smoke smell indoors who did not wear a mask were twice as likely to report sneezing (OR = 2.02 [1.7–2.4]). For those who did wear a mask (and reported 1–5 d of smoke), sneezing rates were only 25% higher. In the most exposed subgroup (> 6 d of smoke), those without masks had an OR of 2.8 [2.3–3.5], whereas the OR among those with a mask was only 1.67.

DISCUSSION

To our knowledge, this is the largest investigation of acute effects of wildfire smoke on children's health. We confirmed very substantial effects of wildfire smoke exposure on eyes as well as upper and lower respiratory symptoms, in both children with asthma and nonasthmatic children. The study was population based; thus, findings may be generalized more broadly to other comparable populations. Our findings are consistent with other studies conducted after wildfire outbreaks and occupational studies among firefighters, which suggest that wildfire smoke leads to acute exacerbations of respiratory and eye symptoms and increased demand for health services (13). Like ambient urban air pollution, wildfire smoke contains numerous primary and secondary pollutants, including particles, polycyclic aromatic hydrocarbons, carbon monoxide, aldehydes, organic acids, organic compounds, gases, free radicals, and inorganic materials with diverse toxicologic properties (14), which may explain the wide range of acute symptoms observed in our survey (15).

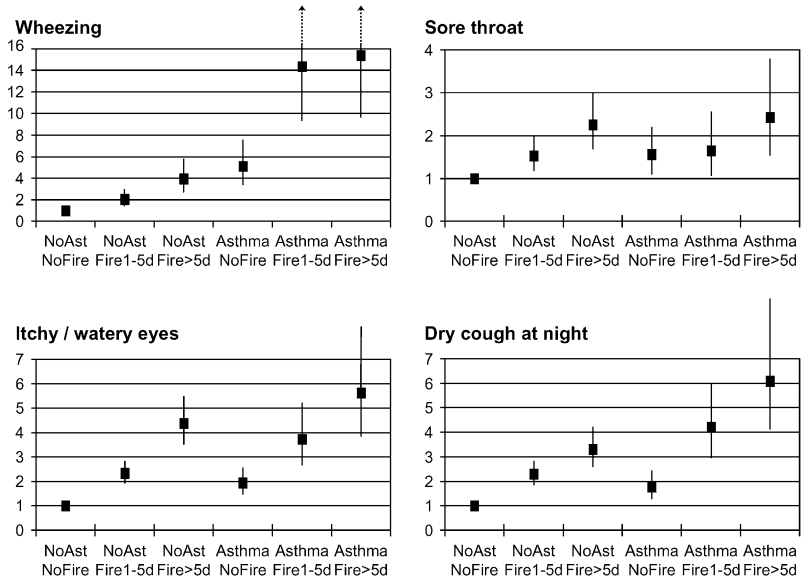

Figure 2. Effect of reported smell of fire smoke indoors (during 1–5, > 5 d, respectively) for four symptoms among children with and without asthma. Odds ratios and 95% confidence intervals from models with interaction terms for asthma and fire, adjusted for sex, ethnicity, educational level of parents, and cohort. No asthma (No Ast), no fire = reference.

TABLE 5. ODDS RATIOS FOR SYMPTOMS AMONG THOSE WITH AND WITHOUT PREVENTIVE ACTIONS AND WITH NO REPORTED SMOKE EXPOSURE (REFERENT GROUP), 1–5, OR ⩾ 6 DAYS OF FIRE SMOKE SMELL INDOORS

	Use of Mask		Air Conditioner Use		Less Outdoors	
Symptom Exposure Level	No OR (*95% CI*)	Yes OR (*95% CI*)	No OR (*95% CI*)	Yes OR (*95% CI*)	No OR (*95% CI*)	Yes OR (*95% CI*)
Sneezing or runny/blocked nose, n	3,673	396	3,158	911	1,371	2,698
No fire smell (referent)	1.00	1.00	1.00	1.00	1.00	1.00
1–5 d fire smell	2.01 (1.70–2.39)	1.58 (0.86–2.91)	2.14 (1.77–2.59)	1.75 (1.26–2.43)	1.84 (1.25–2.73)	1.76 (1.46–2.11)
6 or more days fire smell	2.81 (2.27–3.47)	2.30 (1.22–4.31)	3.05 (2.42–3.85)	2.23 (1.52–3.25)	2.54 (1.60–4.01)	2.47 (1.98–3.09)
Wheezing, n	3,630	387	3,111	906	1,357	2,660
No fire smell (referent)	1.00	1.00	1.00	1.00	1.00	1.00
1–5 d fire smell	2.05 (1.51–2.79)	1.50 (0.68–3.31)	2.29 (1.64–3.18)	1.79 (1.79–3.07)	4.80 (2.51–9.20)	1.76 (1.28–2.42)
6 or more days fire smell	3.47 (2.49–4.85)	2.23 (1.52–3.25)	3.46 (2.41–4.98)	3.00 (1.71–5.27)	7.65 (3.74–15.63)	2.91 (2.06–4.09)

For definition of abbreviations, *see* Table 4.

Total n varies between 4,017 and 4,069 due to varying number of "don't know" answers). The interactions of fire smell and preventive actions were statistically significant (likelihood ratio test, $p < 0.05$) for mask and air conditioner use in case of sneezing/blocked nose, and for "less outdoors" in case of wheezing. Note that in each exposure category, those taking preventive action had higher symptom rates than those not taking action (*see* text).

Biases require particular attention in the interpretation of these findings. Because many parents and students completed the fire questionnaire several months after the fire (from 1 to 7 mo later), and because both exposure and outcome are reported by participants, the study may be subject to interrelated reporting, recall, and selection biases. Due to the lack of individual-level PM_{10} data, we were able to compare effects of objective (PM_{10}) and subjective (reported) markers of exposure in the between-community comparison only. We used the community mean of reported fire smoke as the subjective aggregate exposure.

For reported fire smoke, the estimates for individual and community mean were similar for most outcomes. However, between-community estimates using the mean reported fire smell were not entirely consistent with those based on PM_{10}. The latter showed no clear association with cold, cough, asthma symptoms, physician visits, and missing school. There are several possible reasons for these inconsistencies.

First, the exposure metrics are inherently different and measure different domains of exposure. PM_{10} estimates the average concentration during the 5 most extreme days. In contrast, the questionnaire-based approach relates to the duration (i.e., number of days of observed smoke) rather than the level of the smoke in the community. Duration may characterize the true contrasts in exposure better than the 5-d average PM_{10} because some communities experienced fire smoke for longer or shorter periods.

Second, PM_{10} levels had to be estimated for five fire communities (*see* METHODS and online supplement). The unknown errors in these estimates may lead to under- or overestimation in the between-community effects. Thus, the results based on "objective" measures of community-level exposure are not necessarily unbiased.

Third, PM_{10} community levels are not sensitive to spatial differences in smoke densities that may have occurred within communities. Therefore, PM_{10} concentrations at the monitor may not represent the mean of the true, but unknown, home outdoor PM_{10} levels. We have no objective data to validate the reported diversity on the individual level. Wu and colleagues estimated PM_{10} distributions all across the Southern California area during the wildfire period, using PM measurements, light extinction, meteorologic data, and smoke information from satellite images (1) (*see* Figure E1). We used these results to investigate the range of daily mean PM_{10} concentrations for small areas representing size and location of several CHS communities. For example, the PM_{10} concentration estimates for a 1×1–km grid within a 10-km buffer around San Dimas indicated substantial temporal differences during the fire period, with daily means ranging from 115 $\mu g/m^3$ (October 28) to 220 $\mu g/m^3$ (October 26) as well as large spatial gradients across the grid points. For example, on October 25, the point estimates ranged from 54 to 250 $\mu g/m^3$, and from 90 to 337 $\mu g/m^3$ the next day, with spatial standard deviations up to 50% of the daily means (*see* Figure E1). Although these PM_{10} estimates may be associated with significant uncertainties at the neighborhood scale, they demonstrated substantial spatial heterogeneity, which corroborates the notion that smoke concentrations may vary substantially within communities. The distribution of reported smoke—and thus the community mean of the reported conditions—may reflect these distinct spatial gradients that are influenced by topography and wind patterns (1, 2).

Fourth, the community-level PM_{10} does not take into account PM_{10} levels in locations to which the children might have been evacuated, nor does it account for other individual preventive action taken during the fire period. Thus, the monitor PM_{10} value may again be offset from the true, but unknown, mean PM_{10} across children.

Fifth, the reported fire smell related to the indoor environment where most of the time was spent, whereas outdoor PM_{10} levels are not sensitive to differences among children's indoor environments.

The community mean of the reported fire smell was highly correlated with measured PM_{10}. However, others have shown that community mean reported annoyance of ambient air pollution correlates highly with objective measurements, whereas individual scores may poorly correlate with the home outdoor NO_2 measurements (16). Reporting was associated with health status and sex. A recent review also concluded that reported exposure to traffic was poorly associated with objective data (17). It is not clear whether findings for reported ambient air pollution also apply to fire smoke perception. Reporting of fire smoke may be less affected by personal attitudes than reported ambient air pollution, given the strong smell of fire smoke, the visibility of the problem, and the exceptional situation of the fire period. Reporting was also associated with sex. Eye symptoms, cold, medication, and physician visits were significantly more often reported among girls, whereas boys were more likely to report wheezing (data not shown). However, sex did not confound nor modify the main effects of fire smoke.

In conclusion, although it is neither possible to dismiss the possibility of biases nor to quantify their effects on our results,

we believe that the arguments outlined above support the questionnaire-based results.

Effect of Fire Smoke on Children with Asthma

With the exception of bronchitis, we consistently observed larger coefficients of reported fire smoke among the nonasthmatic children, a general pattern also true for the between-community comparison. However, the smaller effect sizes in children with asthma must be seen in light of the much higher baseline rates for all symptoms among these children (*see* Tables 3 and 4). Therefore, a small increase in the relative risk may constitute a much larger effect in the children with asthma than in the nonasthmatic children. This is apparent in Figure 2. Symptom rates among children with asthma with no fire smoke were generally as high as those among nonasthmatic children with 1 to 5 d of fire smoke.

Children with asthma were usually treated and may have had better access to medical treatment. A Centers for Disease Control and Prevention surveillance reported increased over-the-counter sales of medication after this 2003 fire period (13). The fire questionnaire did not ask about specific treatments such as steroids.

We also have evidence that children with asthma were more likely to change their behavior (data not shown). For example, 15% of children with asthma reported to have worn a mask for at least some days, whereas only 2% of nonasthmatic children reported taking this personal protective measure. More children with asthma reported reduction in time spent outdoors, outdoor sports, and indoor physical activity due to the fire than did nonasthmatic children. This is in line with results from a previous California fire study indicating that those with preexisting conditions were more likely to follow public advisories to prevent smoke exposure (18). The use of air cleaners in that study was twice as high among those with preexisting health problems.

During a 1987 fire period in California, emergency room visits due to asthma and a range of upper and lower respiratory problems increased significantly beyond the expected rates (19). This was also observed in San Diego County during this 2003 wildfire (5). Large fires in Lithuania also affected crude rates of asthma exacerbation (20). Australian scientists found inconclusive results in their investigation of bushfire effects among a panel of 32 children with asthma. Only evening wet cough was associated with fire smoke (21). Interaction with medication use was not assessed (7), but statistical power may have been a major limitation.

We conclude that the much higher background rates of symptoms was the major reason for the weaker effect estimates observed among children with asthma, and that limitations in the assessment of asthma activity, severity, and medication added further random error to the assessment of effects in children with asthma.

We did investigate effects of fire on boys and girls separately (data not shown). Although baseline frequencies differed by sex for some symptoms, sex did not confound nor did it modify the effects of fire smoke.

Long-term Ambient Air Pollution and Fire Smoke

Some of the CHS communities with high long-term ambient pollution were heavily affected by fire; thus, we investigated confounding by long-term exposure to air pollution (data not shown). Communities with high long-term pollution had significantly higher reporting of "bronchitis" and "missed school." However, the long-term mean ambient PM did not confound the association between fire smoke and fire-related outcomes. Regular exposure to wood smoke has been reported to be a risk factor for chronic respiratory diseases (22). Tan and colleagues (23) and van Eeden and colleagues (24) have shown that acute exposure to wildfire smoke was associated with the stimulation of the bone marrow to release polymorphonuclear leukocytes in men, which reflects a systemic response that may be relevant to subsequent lung injury. However, the long-term relevance of a single wildfire exposure is not clear. Follow-up of the CHS fire study participants may allow an investigation of the long-term consequences of this unusual episode.

We stratified the analyses by cohort to investigate age-related differences in the effect of fire smoke. Results among the (larger) cohort of elementary-school children were more often statistically significant than in the cohort of high-school students. Coefficients tended to be larger in the latter, however, in particular for the between-community estimates (data not shown). It is difficult to assign these differences to age, given the differences in the study methods, with parents reporting for their young children and high-school students self-reporting symptoms.

Participation rates immediately after the fires in 2003 were low in some cities, so the presence of possible selection bias based on exposure and/or symptoms might have been an issue. However, survey administration efforts during 2004 resulted in increased response rates. We evaluated the effect of time elapsed since the fire on reported symptom prevalence. For some symptoms, the likelihood of reporting steadily decreased as time elapsed between the fire and answering the questionnaire (results not shown). Reporting of eye-related symptoms increased with elapsed time. This analysis demonstrates the importance of obtaining symptom-related information from study subjects in as timely a manner as possible after an unexpected natural event or emergency. Attempts to maximize early responses are important strategies for future studies. Inclusion of some control outcome not believed to be affected by fire smoke (e.g., stomach or digestive complaints) could have enhanced the assessment of reporting biases.

Our study suggests there was a beneficial effect of wearing masks, spending less time outdoors, and/or using air conditioning—actions that were recommended during the fire by public health agencies and the media. As recently shown in a fire smoke intervention study conducted in Colorado, ventilation patterns including the use of air filters can have substantial effects on the indoor levels of fire-related PM (25); thus, our results are plausible. However, because our assessment of exposure, symptoms, and preventive action were cross-sectional and self-reported, caution is appropriate in the interpretation of these results.

In summary, this investigation indicates substantial effects of fire smoke on children's health. The study provides suggestive evidence for protective health benefits of simple strategies, such as staying indoors, wearing a mask, or the use of air conditioners during wildfire smoke periods.

Conflict of Interest Statement: None of the authors has a financial relationship with a commercial entity that has an interest in the subject of this manuscript.

Acknowledgment: The project of Dr. R.J. Delfino (South Coast Air Quality Management District 04182) provided crucial information regarding the PM_{10} concentration in five fire communities. The authors acknowledge the hard work of the administrative and research staff in the USC Division of Environmental Health.

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Environment International 139 (2020) 105668

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

The delayed effect of wildfire season particulate matter on subsequent influenza season in a mountain west region of the USA

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ARTICLE INFO

Handling Editor: Dr. Hanna Boogaard

Keywords:
Air pollution
MODIS
$PM_{2.5}$
Respiratory health
Influenza
Wildfire Smoke

ABSTRACT

Particularly in rural settings, there has been little research regarding the health impacts of fine particulate matter ($PM_{2.5}$) during the wildfire season smoke exposure period on respiratory diseases, such as influenza, and their associated outbreaks months later. We examined the delayed effects of $PM_{2.5}$ concentrations for the short-lag (1–4 weeks prior) and the long-lag (during the prior wildfire season months) on the following winter influenza season in Montana, a mountainous state in the western United States. We created gridded maps of surface $PM_{2.5}$ for the state of Montana from 2009 to 2018 using spatial regression models fit with station observations and Moderate Resolution Imaging Spectroradiometer (MODIS) aerosol optical thickness data. We used a seasonal quasi-Poisson model with generalized estimating equations to estimate weekly, county-specific, influenza counts for Montana, associated with delayed $PM_{2.5}$ concentration periods (short-lag and long-lag effects), adjusted for temperature and seasonal trend. We did not detect an acute, short-lag $PM_{2.5}$ effect nor short-lag temperature effect on influenza in Montana. Higher daily average $PM_{2.5}$ concentrations during the wildfire season was positively associated with increased influenza in the following winter influenza season (expected 16% or 22% increase in influenza rate per 1 $\mu g/m^3$ increase in average daily summer $PM_{2.5}$ based on two analyses, $p = 0.04$ or 0.008). This is one of the first observations of a relationship between $PM_{2.5}$ during wildfire season and influenza months later.

1. Introduction

The last two decades have seen a dramatic increase in wildfire activity across much of the western United States (US), a trend that has been attributed to decreasing summer precipitation and increasing temperatures (Westerling et al., 2006; Abatzoglou and Williams, 2016; Holden et al., 2018). Communities impacted by smoke from nearby and distant wildfires experience high episodic exposures to fine particulate matter (aerodynamic diameter < 2.5 μm; $PM_{2.5}$) with concentrations often exceeding 24-hour ambient air quality standards for extended periods (Liu et al., 2015). While recent studies have shown air quality improving for the contiguous US from the reduction of industrial and vehicular emissions (McClure and Jaffe, 2018; O'Dell et al., 2019), air pollution in wildfire-prone areas, particularly in the mountain west region of the US, has increased and is projected to further worsen due to climate-mediated increases in wildfire activity (Yue et al., 2013; Liu et al., 2016; Ford et al., 2018).

$PM_{2.5}$ is widely known to have significant adverse effects on human health (US EPA 2009; Anderson et al., 2012; Kim and Kabir, 2015), and several studies of $PM_{2.5}$ during wildfires have found similar positive

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https://doi.org/10.1016/j.envint.2020.105668
Received 29 July 2019; Received in revised form 12 March 2020; Accepted 16 March 2020
Available online 31 March 2020

associations with respiratory effects, including increased hospitalization and medication use for asthma, increased urgent care visits for cardiopulmonary outcomes and increased mortality (for reviews see Liu et al., 2015; Reid et al., 2016; Adetona et al., 2016). Adverse respiratory outcomes associated with wildfire exposure also include hospitalizations and urgent care visits for respiratory infections, pneumonia and bronchitis (Reid et al., 2016). To date, all studies of wildfires have focused on acute health effects with analyses typically not extending beyond a few days lag period.

Wintertime influenza offers an opportunity to evaluate the potential for longer-lag delayed effects of $PM_{2.5}$ associated with smoke from wildfire events. Traditionally, meteorological factors in temperate countries, such as low temperatures and humidity, have been shown to contribute to the risk of influenza outbreaks (Tamerius et al., 2013) and are well correlated with the seasonal changes in the US (Shaman et al., 2010). Recent studies have begun to investigate the associations of $PM_{2.5}$ and influenza. For example, a study in Beijing, China, reported the association between the delayed impact of short-term exposure of $PM_{2.5}$ and monthly influenza cases (Liang et al. 2014). A follow up study found correlations between $PM_{2.5}$ exposure and daily influenza risk by age group in Beijing, China, suggesting a 1-day optimal lag effect (Feng et al., 2016). A more recent study showed consistently increased odds of healthcare encounters for influenza for elevated $PM_{2.5}$ exposure estimates averaged across several lag periods, 0–28 days (Horne et al., 2018). However, to date and particularly in rural settings, there has been little research regarding the health impacts of $PM_{2.5}$ during the wildfire season smoke exposure period on influenza occurrence months later.

While the burden of influenza can vary from season to season, it is estimated that between 9 and 49 million cases of influenza occur each year in the United States. Of these, an estimated 140,000–960,000 hospitalizations and up to 79,000 deaths due to influenza occur each year (www.cdc.gov/flu/about/burden/index.html). In addition, a 2007 review of the economic burden of influenza determined that direct medical costs average around $10.4 billion (Molinari et. al., 2007). In the western US state of Montana, approximately 10,000 cases of influenza are reported each season (approximately October – May; https://www.cdc.gov/flu/about/season/flu-season.htm), but it is likely that the actual number is higher as not all individuals who are infected will seek medical care (MT DPHHS data). In Montana, influenza is associated with approximately 900 hospitalizations and 60 deaths each year (www.cdc.gov/flu/about/burden/index.htm). Since influenza cases are monitored closely by state and federal agencies and the periods between wildfire activity and influenza transmission are offset by weeks to months, we have the opportunity to investigate the potential of $PM_{2.5}$ exposure from wildfire season months to impart impacts weeks to months after exposure.

Wildfires have been identified as the dominant source of elevated surface $PM_{2.5}$ across the Northern Rocky Mountain region (Idaho, Montana, and Wyoming) during the western US wildfire season (Liu et al., 2016; Brey et al., 2018). Here, we define wildfire season as July 1 -September 30. This time period accounts for greater than 90% of annual wildfire emissions across the region (Urbanski et al., 2017, 2018). Inter-annual differences in wildfire activity and wildfire $PM_{2.5}$ emissions result from variability within this time window. Even in the most active fire years, fire burned area and emissions outside July – September are a minimal fraction of the total in the Northern Rocky Mountain region (Urbanski et al., 2018). In Montana, wildfires are the primary $PM_{2.5}$ emission source during the western US wildfire season (Urbanski et al., 2018). The dominant non-wildfire emissions of primary annual $PM_{2.5}$ within Montana were dust from agriculture and unpaved roadways (53%) and prescribed fires (27%), while residential fuel combustion accounted for ~2%, according to the Environmental Protection Agency (EPA) triennial National Emission Inventories (NEI) of 2011 and 2014 (www.epa.gov/air-emissions-inventories/national-emissions-inventory-nei).

The major contributor to short-lag $PM_{2.5}$ exposures during flu season in Montana is biomass smoke exposure generated from wood stoves used for heating throughout the winter months. $PM_{2.5}$ source apportionment modeling has identified wood smoke contributions to be between 56 and 77% of the ambient wintertime $PM_{2.5}$ in multiple communities throughout western Montana (Ward and Lange 2010). Other contributions include dust (1–4%), ammonium nitrate from heavily fertilized agricultural fields and livestock waste (10–20%), sulfate (0–5%), diesel (0–5%), automobiles (0–4%), and unexplained sources (0–4%) (Ward and Lange 2010).

The objective of this study was to evaluate associations between $PM_{2.5}$ and influenza counts at the county level in Montana. Specifically, we developed spatio-temporal $PM_{2.5}$ maps to estimate $PM_{2.5}$ effects for two different time frames: (1) $PM_{2.5}$ exposure 1–4 weeks before influenza cases, hereafter referred to as short-lag $PM_{2.5}$ and (2) $PM_{2.5}$ during the wildfire season 1–10 months before influenza cases, hereafter referred to as long-lag $PM_{2.5}$. We then evaluated associations between the delayed effects of $PM_{2.5}$ for each time frame on influenza counts for counties in Montana for 2010–2018, adjusted for temperature and seasonal trend, in a quasi-Poisson model framework.

2. Methods

2.1. Influenza data

In this time-series analysis, influenza counts in Montana were provided by the Montana Department of Public Health and Human Services. The data used in this study are weekly county-level case counts of positive diagnoses of influenza from all reporting sources, including laboratory confirmations, hospitalizations, and clinical diagnoses. Influenza cases in those of all ages are reported. The Centers for Disease Control do not state the estimated under-reporting of influenza, but do acknowledge that it is largely under-reported (www.cdc.gov/flu/about/burden/how-cdc-estimates.htm). Six small population counties (Musselshell, Petroleum, Judith Basin, Wheatland, Golden Valley, and Fergus) were grouped into what is known as the 'Central Montana Health District' (CMHD). The CMHD and all 50 other Montana counties were included in this study for a total of 51 regions which will be referred to as counties for simplicity. In total, the influenza data for Montana produced 51 counties over 8 years or 408 'clusters' of time series. Mean, minimum, and maximum case counts of each week for all counties are shown in Fig. 1A. Fig. 1B depicts flu incidence per 1,000 in each county for an example flu season period (October 1, 2015 – April 30, 2016) for each county in Montana.

In temperate climates and during the northern latitude summer months (May-August), influenza counts are at their minimum, whereas winter months are the predominant season for infection due to cold temperatures, low humidity, and increased indoor crowding (Finkelman et al. 2007; Cauchemez et al. 2008; Tamerius et al. 2013). We considered two datasets for our modeling purposes. For our first dataset, we excluded flu counts from May 1 – August 30, as these primarily contained either unreported cases or 0 counts. This dataset hereafter referred to as the 'complete' dataset (n = 12,474) had an average flu season length of 31 weeks. Our second dataset accounted for the start and end of each flu season, further reducing the zero counts in the flu data. The second dataset hereafter referred to as the 'reduced' dataset (n = 6,308) was subset into vectors of flu counts with a single leading zero and trailing zero for each flu season within each year and each county. The reduced data set had an average flu season length of 15.5 weeks.

2.2. $PM_{2.5}$ model for Montana

We used daily $PM_{2.5}$ measurements from air quality monitoring stations, combined with satellite retrievals of aerosol optical thickness from the Moderate Resolution Imaging Spectroradiometer (MODIS) to

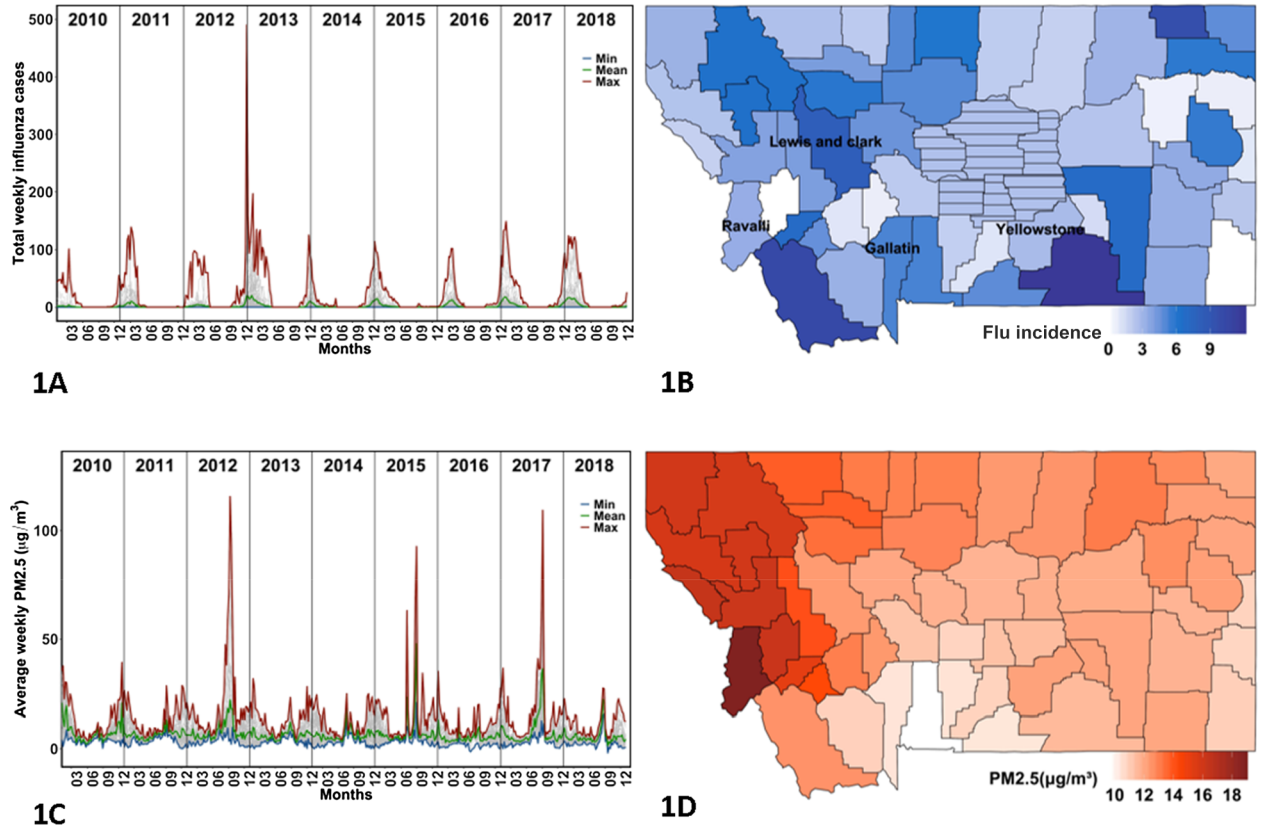

Fig. 1. (A) Total weekly influenza cases plotted for all Montana counties, 2010–2018. (B) Flu season incidence (per 1,000) for each county in Montana, 2015–2016 with the 'Central Montana Health District' (CMHD) shown with dash. (C) Average weekly $PM_{2.5}$ (ug/m^3) plotted for each county in Montana, 2010–2018. (D) Average $PM_{2.5}$ during the wildfire season (July 1 – September 30) for each county in Montana, 2015.

produce gridded maps of daily $PM_{2.5}$ concentrations across the state of Montana from 2009 − 2018. Mean daily $PM_{2.5}$ were retrieved from the EPA (https://www.epa.gov/outdoor-air-quality-data) and used as a response variable in the models. One hundred and seven stations within a domain bounded by −100 to −120 degrees longitude and 42 – 49 degrees latitude were used for model fitting. Counts of daily observations available for each station varied from 1% to 100%, with a mean of 21% and counts of the number of available observations by day ranged from 10 to 57, with a mean of 38. The 1 km MODIS aerosol optical thickness (AOT) product, developed using the MAIAC algorithm (Lyapustrin et al. 2018) was used as predictor. Quality assurance layers provided for each image were used to screen pixels containing snow or clouds, and only the highest quality observations were retained for analysis. Fig. A1 shows the correlation of the AOT data with the $PM_{2.5}$ EPA station data. Use of remotely sensed products is particularly challenging in Montana, due to image contamination by clouds and snow. More than 90% of data were missing during winter months (Fig. A2). Therefore, satellite AOT data were only used in the models from June-October (but see comparison of models for November-May in Appendix). We infilled missing observations in the June-October AOT data using probabilistic Principal Components Analysis (Stacklies et al. 2007).

We used Bayesian spatial linear models implemented in the 'spBayes' package (spLM; Finley et al. 2015) in R version 3.5.3 (R Core Team 2019) to estimate daily $PM_{2.5}$ concentrations. We fit a unique model for each day, and predicted the fitted model to a 12 km grid. Aerosol optical thickness was used as a predictor in this model along with an exponential covariance model. Uninformative priors were used for the regression parameters, a uniform prior was used for the spatial decay parameter with support for effective ranges between 5% and 90% of the maximum inter-point distance, and uninformative inverse gamma priors were used for the covariance sill and nugget (σ^2 and τ^2) parameters. For November-May days, we used thin plate spline regression (TPS) in the R package 'fields' (Nychka et al., 2017) to interpolate daily $PM_{2.5}$ observations without any spatial covariates. This decision was made because of the high proportion of missing MODIS data in winter. Here, a spline regression model was fit for each date using the latitude and longitude of each station and predicted the model to a 12 km grid. We evaluated both the spLM and TPS models using a leave-one-out cross validation approach. Here, for every model date each observation was withheld. A model was fit using the remaining observations, and then predicted to the withheld observation. This procedure was repeated for each observation and each date and error statistics were retained for all days. The overall model fit for the summer wildfire season model was reasonably strong, with Mean Absolute Error of 2.79 (µg/m^3) and r^2 = 0.66 (Fig. A3a). The accuracy of the thin plate spline model, fit without the benefit of MODIS AOT data was not as strong as expected, with Mean Absolute Error of 3.11 (µg/m^3) and r^2 = 0.37 (Fig. A3b). Daily gridded $PM_{2.5}$ estimates were combined into weekly grids using the mean, and then extracted for county in Montana also using the mean.

The resulting weekly $PM_{2.5}$ time series for all counties is given in Fig. 1C. For this study, we are primarily interested in evaluating two possible functions of $PM_{2.5}$ in relation to influenza: (1) a long-lag effect experienced primarily from $PM_{2.5}$ during wildfire season, and (2) a short-lag effect experienced primarily from biomass smoke exposure (e.g., Ward and Lange, 2010). We tested multiple such functions to express these different kinds of $PM_{2.5}$ periods and effects as summarized in Table 1. For the long-lag effects, we used the average daily $PM_{2.5}$ concentration for the months July 1 – September 30 preceding the flu-season, when $PM_{2.5}$ density spikes due to wildfires, with the intent of estimating average $PM_{2.5}$ concentrations during wildfire season. An

Table 1
Short-lag and long-lag $PM_{2.5}$ variables considered.

		Variable Name	Description
$PM_{2.5}$ Variables Tested	**Long-lag Effects**	Daily average $PM_{2.5}$ during wildfire season	Total $PM_{2.5}$ from months preceding flu-season (July 1 – September 30) divided by wildfire season total days (91 days)
	Short-lag Effects	(A) *n* week lag (*n = 1,2,3,4)*	Lag $PM_{2.5}$ up to *n* weeks before current week of influenza
		(B) *n* week moving window sum (*n = 2,3,4)*	Total $PM_{2.5}$ up to *n* weeks before current week of influenza divided by *n* weeks
		(C) Daily average $PM_{2.5}$ during flu season	Total $PM_{2.5}$ over entirety of flu-season (October 1 – April 30) divided by flu-season total days

Table 2
Model summary for each variable and dataset.

Complete Dataset (n = 12,474)				Reduced Dataset (n = 6,308)			
Term	Estimate	Robust SE	pval	Term	Estimate	Robust SE	pval
Sine, Cosine	Varies	Varies	$< 10^{-5}$	*Sine, Cosine*	Varies	Varies	$< 10^{-5}$
Temperature Lag	−0.0014	0.0030	0.650	*Temperature Lag*	0.0001	0.0030	0.979
Daily Long-Lag $PM_{2.5}$	0.1995	0.0752	0.008	*Daily Long-Lag $PM_{2.5}$*	0.1470	0.0724	0.042
Daily Short Lag $PM_{2.5}$	−0.0459	0.0464	0.323	*Daily Short Lag $PM_{2.5}$*	−0.0407	0.0483	0.399

example long-lag $PM_{2.5}$ for each Montana county in 2015 is shown in Fig. 1D. Fig. A4 shows how severe the wildfire seasons were each year in Montana using total area burned. For the short-lag effects, we looked at three different $PM_{2.5}$ variables: (A) a lag in $PM_{2.5}$ up to *n* weeks before current week of influenza, (B) a moving window daily average $PM_{2.5}$ up to *n* weeks before current week of influenza, and (C) a daily average $PM_{2.5}$ concentration over the entirety of flu-season.

2.3. Statistical analysis

The associations between weekly counts of influenza cases and the different $PM_{2.5}$ effects were examined using the following quasi-Poisson regression model weighted by county population for all counties simultaneously:

$$\log(\mu_{t,k}) = \log(Population_{t,k}) + \beta_0 + \sum_{i=1}^{6} \beta_i F_i(t,k) + \beta_7 Temperature_{t-1,k} + \beta_8 LongLagPM2.5_{t,k} + \beta_9 ShortLagPM2.5_{t,k} + \sum_{i=10}^{60} \beta_i County_k \quad (1)$$

where *t* is the week index, $t = 1, 2, \ldots, 435$ weeks from 2010-Jan-03 to 2018-May-31, excluding weeks outside of the flu season, *k* is the county index, $k = 1, 2, \ldots, 51$ counties as earlier defined, $\mu_{t,k}$ is the expected influenza count at time *t* in county *k*, assuming $\mu_{t,k} \sim$ Exponential Family(θ), $Population_{t,k}$ is the population in county *k* in week *t*, entering the model as an offset allowing an influenza rate response, $Temperature_{t-1,k}$ is the maximum daily temperature (in deg. Celsius) extracted using 250 m resolution gridded temperature data (Holden et al., 2018) in county *k* in week *t*-1, $LongLagPM2.5_{t,k}$ is the long-lag $PM_{2.5}$ daily average from the previous wildfire season as described in the previous section for county *k* relative to week *t*, $ShortLagPM2.5_{t,k}$ is the short-lag $PM_{2.5}$ effect as described in Table 1 for county *k* relative to week *t*, $F_i(t,k)$ is the i^{th} Fourier seasonal term ($i = 1,2,3$ for Sine and $i = 4,5,6$ for Cosine) for county *k* in week *t*, and $County_k$ is a county indicator (1 if county *k* and 0 otherwise). Notice that the first seven terms in the model (parameterized by $\beta_1, \beta_2, \ldots, \beta_7$) are all variables regularly associated with influenza dynamics (e.g., Imai et al., 2015).

A generalized estimating equation (GEE) was used to estimate the model parameters for this quasi-Poisson generalized linear model to address any residual temporal autocorrelation and uncertainty in the covariance structure of the flu counts. An autoregressive (AR(1)) covariance structure is assumed for the GEE to account for the weekly dependence in flu counts within each county-year cluster (51 counties × 8 years = 408 county-year clusters). The model for influenza rate (specified as influenza count with population as an offset) given in Eq. (1) was applied simultaneously to all counties in Montana and for the two datasets described in the previous section (complete and reduced), and basic statistical inference performed on the coefficients using Huber-White robust standard error estimates to account for uncertainty in the quasi-Poisson correlation structure. All analyses were performed using the geem and glm function in R (version 3.5.3; R Development Core Team) with a quasi-Poisson family to account for overdispersion, and each case weighted by the county population.

3. Results

3.1. Long-lag $PM_{2.5}$ impacts on influenza

Average daily long-lag $PM_{2.5}$ concentration (averaged over the period July 1 – September 30 during the previous wildfire season) was positively associated with increased influenza rate for both the complete and reduced datasets ($p = 0.008$ and $p = 0.042$, respectively) as shown in the model summary provided in Table 2. The estimated model coefficients are 0.1995 (SE = 0.0752) and 0.1479 (SE = 0.0724) for the complete and reduced datasets, respectively. For the complete dataset, we expect influenza incidence to increase by a factor of exp (0.1995) = 1.22 per 1 μg/m^3 elevation in average daily wildfire season $PM_{2.5}$ exposure (95% CI: (1.05, 1.41)). For the reduced dataset, we expect influenza incidence to increase by a factor of exp (0.1470) = 1.16 per 1 μg/m^3 elevation in average daily wildfire season $PM_{2.5}$ exposure (95% CI: (1.01, 1.33)). We note that these estimated parameters reflect population-based changes in influenza rate taken on average.

3.2. Short-lag $PM_{2.5}$ impacts on influenza

Moving window daily average short-lag $PM_{2.5}$ two weeks prior to the current week showed no association with influenza rate for the complete dataset ($p = 0.323$) and the reduced dataset ($p = 0.399$) (Table 2). Regardless of short-lag method chosen for the model (Table 1), no association was observed. Individual county-specific models indicated a positive association in 13–23 of the 51 counties, depending on which short-lag $PM_{2.5}$ variable was used, but no overall effects were observed (Table A1; Figs. A5–A6).

3.3. Temperature impacts on influenza

The average maximum temperature (degrees Celsius) in the previous week showed no association with influenza rate for the complete dataset ($p = 0.650$) and the reduced dataset ($p = 0.979$) (Table 2). Individual county-specific models indicated that 39 out of 51 Montana counties show a negative albeit insignificant relationship between temperature and influenza counts (Fig. A7), the direction of which is consistent with all previous literature on this topic (e.g., Tamerius et al. 2013).

3.4. Residual autocorrelation

There was strong evidence of temporal autocorrelation within the county-year clusters in the influenza count model residuals (Breusch-Godfrey test, $p = 0.0004$). To address this residual autocorrelation, robust Huber-White standard errors under an AR(1) covariance structure were used in the GEE modeling framework to assess the significance of model predictors.

4. Discussion

We found that higher average $PM_{2.5}$ concentrations during the wildfire season positively associated with increased influenza in Montana counties in the following winter flu season. Individual county-specific models further support this result, showing long-lag $PM_{2.5}$ positively associating with wintertime influenza in 50 out 51 counties (Table A1; Figs. A5–A7). Although there are studies that report the correlation between short-lag exposure of $PM_{2.5}$ and influenza cases (Liang et al., 2014; Feng et al., 2016; Horne et al., 2018), our study suggests one of the longest lag associations observed for communities impacted by wildfires. Wildfire season for Montana (and much of the intermountain West) occurs between July – September with corresponding peak levels of $PM_{2.5}$. Flu season spans October – April, with peak flu cases typically occurring in January. Thus, average daily $PM_{2.5}$ concentrations during wildfire season months was observed to be positively associated with flu 1–10 months later, even after accounting for seasonal, temperature, and autocorrelative factors.

Past studies modeling the effects of $PM_{2.5}$ during wildfire episodes on respiratory outcomes have typically looked at lagged associations of less than 5 days (Liu et al., 2015; DeFlorio-Bake et al., 2019). However, a recent study showed consistently increased odds of healthcare encounter for influenza for elevated $PM_{2.5}$ exposure estimates averaged across several lag periods, 0–28 days (Horne et al., 2018). We also included a short-lag variable in our model using different methods to account for the effect of $PM_{2.5}$ immediately preceding influenza rates (1–4 weeks; Table 1). Surprisingly, these results were less consistent than the long-lag $PM_{2.5}$ variable, and we found little support for a short-lag $PM_{2.5}$ effect on influenza. Individual county-specific models run with each different short-lag $PM_{2.5}$ variable in Table 1 are compared in the Appendix (Table A1; Figs. A5–A7) and further corroborate this finding. Depending on which $PM_{2.5}$ short-lag variable used, 13–23 of the 51 counties indicated a positive association with short-lag $PM_{2.5}$ and influenza counts with at most 7 counties being significant ($p < 0.05$). The inability to separate out the various contributing factors to short-lag $PM_{2.5}$ in the winter months (i.e., woodsmoke, other industrial pollutants) could be one reason our model was not able to find a short-lag $PM_{2.5}$ association with flu. Several studies have evaluated specific PM components and cardio-respiratory outcomes, but findings have been inconsistent in linking isolated PM factors or sources to specific outcomes (Stanek et al., 2011). To our knowledge no such studies have evaluated PM component or PM source with respect to influenza, and this would be a potential area for further exploration.

Although our modeling was able to partially explain effects of long-lag $PM_{2.5}$ concentrations on Montana's counties, finer scale data could help reveal more spatially resolved details. Our modeling used $PM_{2.5}$ maps produced at relatively coarse (12 km) spatial resolutions and aggregated to the county level. Future research should explore variation at finer resolution. Of particular concern in western rural states is the scarcity of air quality monitoring stations, which provide the data needed to deliver accurate respiratory health warnings and predictions to the public, as well as to provide the data to better understand the role air pollution has on respiratory diseases. In the intermountain west, the sparsity of air quality monitoring stations is further complicated by the region's complex terrain which likely contributes to significant heterogeneity in air pollution levels across communities (Armstrong, 1998). Furthermore, it is likely that during the wildfire season inversions and drainage flows may lead to highly variable smoke exposure. Many areas of Montana and the intermountain west in the wintertime experience an increased risk of poor air quality due to cold-air inversions, trapping air pollutants in mountain valleys where most towns and residents are located (Ward and Lange 2010; Holden et al., 2011). Air quality monitoring stations are often located to represent worst-case exposures for the largest concentration of people or sited to capture background exposure. For example, in 2018 there were 19 sites in the Montana network that monitored $PM_{2.5}$ (13 = Population Exposure, 5 = Background Exposure, and 1 = Source Impact; https://www3.epa.gov/ttnamti1/files/ambient/pm25/qa/vol2sec06.pdf). Regardless, it is unlikely that the single air monitor sites in many Montana communities provide an accurate representation of pollution exposure and could be missing much of the spatial patterning in $PM_{2.5}$, as suggested by other urban area focused studies (Tunno et al. 2016). Thus, improved spatially resolved maps of $PM_{2.5}$ would enhance understanding of particulate matter impacts on public health during both the winter and wildfire season. Such maps would also provide the spatial and topographically resolved data needed to identify fine scale $PM_{2.5}$ effects on specific respiratory diseases, such as influenza.

There are several potential factors that are relevant to influenza risk that were not addressed in our study. For example, previous studies included sociodemographic factors and one study from Australia found that wealthier communities with lower levels of unemployment experienced greater flu activity than those less advantaged areas (Huang et al., 2017). Other studies have found that school calendars may play a role in influenza outbreaks, suggesting that closing schools could be effective in limiting the spread of influenza outbreaks (Chu et al., 2017), also contributing to the hypotheses on indoor crowding and increased person-to-person contact (Cauchemez et al., 2008). Furthermore, our model did not include vaccination rates (e.g., Baselga-Moreno et al., 2019), influenza strain, distance to airports (e.g., Hooten et al., 2010), and possible important determinants influenza in for rural states, such as healthcare access, or any other sociodemographic or economic variables, all of which could influence influenza transmission. Future studies could explore the interactions of virus-specific, climate, sociodemographic, and $PM_{2.5}$ variables. Finally, we note that our models did not explicitly account for uncertainty in the particulate matter model. This uncertainty was higher in winter months, where satellite data were unavailable, and may have contributed to the lack of any relationship between short-lag PM exposure and influenza.

While our study supports a link between long-lag $PM_{2.5}$ during wildfire season and wintertime influenza, the mechanisms underlying this relationship are complex, and beyond the scope of this study. Future work using in vivo and in vitro studies could be conducted to explore these underlying mechanisms for either viral etiology and/or host susceptibility. For example, some animal studies have looked at wood smoke particles' sustained immune suppression effect (Samuelson et al, 2009; Migliaccio et al, 2013), showing this type of PM having a 24 h sustained effect on respiratory bacterial infections. Other animal models suggest that the role of lagged PM exposure on influenza risk could occur via diminished capacity of pulmonary macrophages to secrete IL-6 and IFN-β (Ma et al. 2017). Ma et al. 2017 provide support for a 2-week exposure to outdoor $PM_{2.5}$ from Shanghai, China, leading to decreased resistance to influenza via altered immune responses.

Predicting influenza outbreaks based on climatic and environmental factors, such as $PM_{2.5}$, may be important for both short- and long-term public health planning. In the short-term, models may help predict outbreaks days to weeks in advance, giving public health officials an opportunity to target prevention messages and vaccine efforts. In the long-term, models linking climatic or environmental variables to influenza outbreaks may provide a picture for what populations can expect with ongoing climate change or extreme seasonal conditions. For example, Ford et al. (2018) projected change in $PM_{2.5}$ based on prognostic land-fire models for the continental US with the worst areas in Montana forecasted to have a 5 $\mu g/m^3$ increase in the annual average per decade. Moreover, identifying the predictors of influenza, such as long-lag $PM_{2.5}$ effects, and improving upon the predictive models for influenza, will be important for the population health, as influenza is associated with approximately 900 hospitalizations and 60 deaths in Montana each year (www.cdc.gov/flu/about/burden/index.htm).

CRediT authorship contribution statement

Erin L. Landguth: Conceptualization, Methodology, Writing - original draft, review & editing. **Zachary A. Holden:** Methodology, Writing - review & editing. **Jonathan Graham:** Methodology, Writing - review & editing. **Benjamin Stark:** Formal analysis. **Elham Bayat Mokhtari:** Visualization. **Emily Kaleczyc:** Data curation. **Stacey Anderson:** Data curation, Writing - review & editing. **Shawn Urbanski:** Writing - review & editing. **Matt Jolly:** Writing - review & editing. **Erin Semmens:** Conceptualization, Writing - review & editing. **Dyer A. Warren:** Formal analysis. **Alan Swanson:** Formal analysis. **Emily Stone:** Writine - review & editing. **Curtis Noonan:** Conceptualization, Supervision, Writing - review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

This research was supported by the National Institute of General Medical Sciences of the National Institutes of Health (NIH), United States [Award Numbers P20GM130418 and P20GM103474] and the Office of the Director, NIH [Award Number 8UG1OD024952]. MODIS Aerosol optical thickness and gridded PM2.5 data generated for this study is available at https://topofire.dbs.umt.edu/helmsdeep1/public_data/air_quality.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2020.105668.

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Review

J Prev Med Public Health 2016;49:205-219 • http://dx.doi.org/10.3961/jpmph.16.037

pISSN 1975-8375 eISSN 2233-4521

Journal of Preventive Medicine & Public Health

Short-term Effect of Fine Particulate Matter on Children's Hospital Admissions and Emergency Department Visits for Asthma: A Systematic Review and Meta-analysis

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Objectives: No children-specified review and meta-analysis paper about the short-term effect of fine particulate matter ($PM_{2.5}$) on hospital admissions and emergency department visits for asthma has been published. We calculated more precise pooled effect estimates on this topic and evaluated the variation in effect size according to the differences in study characteristics not considered in previous studies.

Methods: Two authors each independently searched PubMed and EMBASE for relevant studies in March, 2016. We conducted random effect meta-analyses and mixed-effect meta-regression analyses using retrieved summary effect estimates and 95% confidence intervals (CIs) and some characteristics of selected studies. The Egger's test and funnel plot were used to check publication bias. All analyses were done using R version 3.1.3.

Results: We ultimately retrieved 26 time-series and case-crossover design studies about the short-term effect of $PM_{2.5}$ on children's hospital admissions and emergency department visits for asthma. In the primary meta-analysis, children's hospital admissions and emergency department visits for asthma were positively associated with a short-term 10 μg/m^3 increase in $PM_{2.5}$ (relative risk, 1.048; 95% CI, 1.028 to 1.067; I^2=95.7%). We also found different effect coefficients by region; the value in Asia was estimated to be lower than in North America or Europe.

Conclusions: We strengthened the evidence on the short-term effect of $PM_{2.5}$ on children's hospital admissions and emergency department visits for asthma. Further studies from other regions outside North America and Europe regions are needed for more generalizable evidence.

Key words: Particulate matter, Asthma, Child, Review, Meta-analysis, Hospitalization

Received: April 7, 2016 Accepted: July 14, 2016

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INTRODUCTION

The adverse health effects of air pollution on respiratory and cardiovascular diseases are well known to the public. Regulation and monitoring of air pollution are performed at both the national and international levels. Particulate matter (PM) is one type of air pollutant. It is not a specific chemical entity, unlike other commonly known pollutants such as ozone, sulphur dioxide, and nitrogen dioxide. It is a physical category of

dust with different components mixed together [1]. The particle size determines the different categorizations: PM_{10} (less than 10 μm aerodynamic diameter) and $PM_{2.5}$ (less than 2.5 μm aerodynamic diameter). $PM_{2.5}$ is also known as fine PM.

$PM_{2.5}$ has been reported to play a major role in increasing the chance of mortality due to cardiovascular diseases because it can penetrate the capillary vessel of the lungs and reach the alveoli [2,3]. Extensive research has been conducted on the association between $PM_{2.5}$ and respiratory diseases including asthma. Asthma is a syndrome in which reversible respiratory obstruction occurs and is characterized by hypersensitiveness to allergens. When stimulated, a person experiences wheezing and dyspnea. In most cases, asthma is caused by a genetic predisposition and is triggered by environmental allergens.

The prevalence rate of asthma is high in children. In the case of South Korea (hererafter Korea), the prevalence rate of asthma in children steadily increased due to urbanization and westernization. In 2010, a national study based on the International Study of Asthma and Allergies in Childhood questionnaire found that 10.1% of elementary school students and 8.5% of middle school students had experienced symptoms of asthma in the past 12 months [4]. These numbers should not be ignored.

Recently published systematic reviews and meta-analyses reported the pooled relative risk (RR) of the number of hospital admissions and emergency department (ED) visits due to asthma as 1.023 (95% confidence interval [CI], 1.015 to 1.031, per 10 $\mu g/m^3$ increase) when examining the effects of $PM_{2.5}$ on the total population, and 1.025 (95% CI, 1.013 to 1.037, per 10 $\mu g/m^3$ increase) when the subject was confined to children only [5]. Another review that examined the effects of $PM_{2.5}$ on ED visits due to asthma reported a pooled RR of 1.036 (95% CI, 1.018 to 1.053, per 10 $\mu g/m^3$ increase) [6]. However, existing studies contain several limitations. These studies were not focused on childhood asthma and only presented pooled effect estimates in children as subgroup analysis. Moreover, most of the relevant studies were conducted in North America and Europe [7-28], and although studies conducted in other regions exist [29-32], they did not consider the varying effects of $PM_{2.5}$ according to different regions. The design of the study, the background $PM_{2.5}$ mean concentration and variation of the region where the study was conducted, and the time of study may change the effects as well, but these factors were not adequately considered in existing studies.

In addition to the two reviews mentioned above, seven new relevant papers have recently been published [22-28]. Of these, the time-series studies assessed the exposure to air pollution by using the exposure value of the population-weighted average in between the measuring points of air pollution [22,24,27], and the case-crossover design studies used the method of matching individual addresses with the $PM_{2.5}$ measures [25,26], which yielded more accurate results. Therefore, by including these recent developments, we tried to calculate more accurate pooled effect estimates of the effects of $PM_{2.5}$ on childhood asthma and assess the variations of effects induced by differences in some factors such as region or date of research, which have been not adequately examined yet.

METHODS

Selection Criteria

We first determined some criteria for selecting relevant studies. They are as follows:

1) The subject of study was limited to children and adolescents under the age of 20.
2) Study results were limited to computerized records of hospital admissions and ED visits. Outpatient visits were excluded. Hospital admissions confirmed through interviews were not eligible. Subjective symptoms, decrease in pulmonary function, and use of emergency inhalers were not considered endpoints.
3) Effect estimates had to be presented as an odds ratio (OR) or RR.

Search Terms and Study Selection

When deciding on search terms, we minimized keywords in order to increase the sensitivity of our searches. Some of the search terms we used were child*, pediatric*, fine particulate matter*, fine particle*, $PM_{2.5}$, asthma*, hospitalization, hospitalisation, admission*, ed, er, and emergency. We searched studies to include in our meta-analysis using PubMed and EMBASE in March of 2016. Moreover, we selected the final eligible studies after having two authors each independently select references according to the criteria above and the same search terms and then comparing the two lists.

Statistical Methods

The effect size was expressed as RR. We considered the OR as a proxy to the RR. In order to have all the effect estimates

chosen from the selected studies to reflect the same 10 μg/m^3 increment of $PM_{2.5}$ concentration, we implemented meta-analyses after recalculating the β coefficient and 95% CI presented in each study. Because the purpose of this study is to combine and identify the effects from regions all over the world, generalization of heterogeneous parts of the research group was its goal. Therefore, the random effects model using the DerSimonian and Laird [33] estimation method was mainly considered, rather than the fixed effects model [33,34]. When estimating the pooled effect, the model takes into account both the between-study variation and the within-study variation and provides a greater confidence level than the fixed effects model. The I-squared value (%) was calculated in order to identify heterogeneity.

In the primary meta-analysis of this study, an effect estimate that could represent the selected studies was used. We used the same lag value that was presented in the original paper [35], but if a study presented multiple estimates from different lags, we selected the one with the largest effect size. This is because, generally, these works report the greatest effect size [36]. If a study did not have one effect estimate that could represent the research, we selected two or more values that were obtained from subjects that were mutually exclusive (that is, if a study did not present an effect estimate in whole participants but presented two or more separate values from stratified groups, we included those in the meta-analysis). In order to identify publication bias, we conducted the Egger's test and identified the degree of asymmetry through a funnel plot [37].

Moreover, we conducted category-specific meta-analyses in order to determine what factors influenced the effect of $PM_{2.5}$, if those influences were robust, and what factors contributed to the heterogeneity of effect estimates. We conducted the analyses by sorting the effect estimates into categories of age, results (records of hospital admissions or ED visits), season, design of the study, region, and the lag of exposure. We also conducted a separate analysis according to whether or not different pollutants were adjusted in the statistical model.

We hypothesized that the components of $PM_{2.5}$ would change according to the time of the study and that the size of the effect could change according to the components. In addition, we thought that the variation and the mean concentration of $PM_{2.5}$ in the region where the study was conducted might change the size of the effect. Therefore, through mixed-effects meta-regression, we derived an effect estimate of the time of the study, and the mean and standard deviation of the concentration in the study region on RR for childhood asthma.

All statistical analyses performed using R version 3.1.3 (Comprehensive R Archive Network: http://cran.r-project.org) and we carried out a series of statistical analyses described above through the meta package. All statistical analyses set a 5% significance level for the two-tailed test.

RESULTS

Selection of Relevant Studies and Extracting Effect Estimates and Their Confidence Intervals

A total of 661 references were searched using the search terms mentioned above, and of those, we first selected 56 to examine in whole by excluding overlapping studies (n=171) and reading the titles and abstracts (n=490). Then we ultimately selected 26 studies according to the selection criteria and extracted effect estimates (Figure 1). The 26 studies were published between 1999 and 2016, and we summarized each of the research outlines and the main research results in Table 1. Most of the research was conducted in North America and Europe and both time-series and case-crossover designs were almost equally represented.

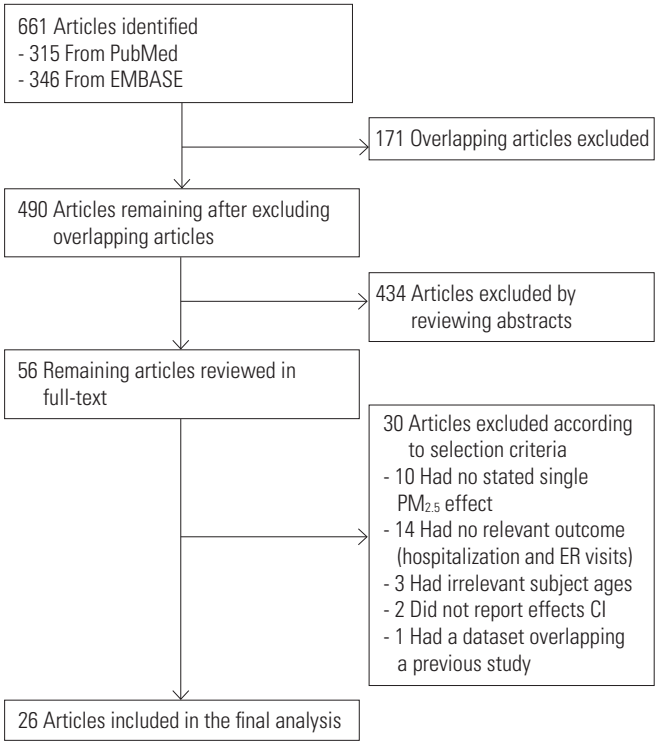

Figure 1. Selection process for systematic review and meta-analysis. $PM_{2.5}$, fine particulate matter; ER, emergency room; CI, confidence interval.

Table 1. Summary of selected studies on the association of short-term fine particulate matter ($PM_{2.5}$) exposure with pediatric HA and ED visits for asthma

Author (publication year) [Ref]	Study period	Location	Sample	Exposure assessment	Outcome	Study design	Statistical model	$PM_{2.5}$ arithmetic mean concentration ($\mu g/m^3$) (SD)	Major effect estimates (risk ratio) (95% CIs)
Norris et al. (1999) [7]	Sep 1, 1995-Dec 31, 1996	Seattle, USA	<18 y, 900 patients	3 Fixed sites; a daily arithmetic mean was calculated and used	ED visits	TS	GAM with Poisson distribution	12.0 (9.5)	Single-pollutant model 1.15 (1.08, 1.23) for 1-d lag IQR increase Multi-pollutant model with SO_2 and NO_2 1.17 (1.08, 1.26) for 1-d lag IQR increase
Lin et al. (2002) [8]	Jan 1, 1981-Dec 31, 1993	Toronto, Canada	6-12 y, 7319 (boys: 4629, girls: 2690) patients	1 Fixed site; the authors obtained data on every 6-d period from 1984 to 1990 and constructed a daily predicted value via modeling	HA	TS and CCD	GAM and conditional logistic regression	18.0 (8.5)	Single-pollutant model (a) Boys, 1.00 (0.97, 1.04) for the same day IQR increase in TS 1.01 (0.97, 1.06) for the same day IQR increase in CCD (b) Girls, 1.06 (0.99, 1.13) for 5-d average IQR increase in TS 1.04 (0.95, 1.15) for 5-d average IQR increase in CCD Multi-pollutant model with CO, SO_2, NO_2 and O_3 (a) Boys, 0.96 (0.90, 1.02) for 5-d average IQR increase in TS 0.94 (0.85, 1.03) for 5-d average IQR increase in CCD (b) Girls, 1.01 (0.93, 1.10) for 5-d average IQR increase in TS 0.96 (0.85, 1.09) for 5-d average IQR increase in CCD
Lee et al. (2006) [29]	Jan 1, 1997-Dec 31, 2002	Hong Kong, China	≤18 y, 26 663 patients	13 Fixed sites (before 2000, 11 sites); a daily arithmetic mean was calculated and used	HA	TS	GAM with Poisson distribution	45.3 (16.2)	Single-pollutant model 1.066 (1.045, 1.087) for 4-d lag IQR increase Multi-pollutant model with CO, SO_2, NO_2 and O_3 1.032 (1.009, 1.056) for 1-d lag IQR increase
Ko et al. (2007) [30]	Jan 1, 2000-Dec 31, 2005	Hong Kong, China	≤14 y, 23 596 patients	3 Fixed sites; a daily arithmetic mean was calculated and used	HA	TS	GAM with Poisson distribution	65.4 (21.1)	Single-pollutant model 1.024 (1.013, 1.034) for 5-d average 10 $\mu g/m^3$ increase
Villneneuve et al. (2007) [9]	Jan 1, 1998-Mar 31, 2002	Edmonton, Canada	2-4 y, 7247 patients; 5-14 y, 13 145 patients	3 Fixed sites; a daily arithmetic mean was calculated and used	ED visits	CCD	Conditional logistic regression	7.0[1] in Apr to Sep; 7.3[1] in Oct to Mar	Single-pollutant model: (a) 2-4 y, 1.06 (0.97, 1.15) for 5-d average IQR increase - Oct to Mar: 0.95 (0.84, 1.07) - Apr to Sep: 1.16 (1.04, 1.28) (b) 5-14 y, 1.06 (1.00, 1.12) for 5-d average IQR increase - Oct to Mar: 0.99 (0.91, 1.09) - Apr to Sep: 1.10 (1.02, 1.17)

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Table 1. Continued from the previous page

Author (publication year) [Ref]	Study period	Location	Sample	Exposure assessment	Outcome	Study design	Statistical model	$PM_{2.5}$ arithmetic mean concentration ($\mu g/m^3$) (SD)	Major effect estimates (risk ratio) (95% CIs)
Andersen et al. (2008) [10]	Oct 3, 2003-Dec 31, 2004	Copenhagen, Denmark	5-18 y, 559 patients in single pollutant model; 318 patients in two-pollutant model	1 Fixed site; a daily arithmetic mean was calculated and used	HA	TS	GLM with Poisson regression	10.0 (5.0)	Single-pollutant model 1.15 (1.00, 1.32) for 6-d average IQR increase Two-pollutant model with total number concentration of particles 1.13 (0.98, 1.32) for 6-d average IQR increase
Halonen et al. (2008) [11]	Jan 1, 1998-Dec 31, 2004	Helsinki, Finland	< 15 y, 4807 patients	Fixed monitoring site, no specific information available	ED visits	TS	GLM with Poisson regression	9.5[1]	Single-pollutant model 1.026 (0.083, 1.054) for 4-d lag IQR increase
Jalaludin et al. (2008) [31]	Jan 1, 1997-Dec 31, 2001	Sydney, Australia	1-14 y, 317 724 patients	14 Fixed sites; a daily arithmetic mean was calculated and used	ED visits	CCD	Conditional logistic regression	9.4 (5.1)	Single-pollutant model (a) 1-4 y, 1.014 (1.007, 1.021) for the same-day IQR increase - Warm months: 1.009 (1.002, 1.017) - Cool months: 1.010 (0.999, 1.024) (b) 5-9 y, 1.016 (1.005, 1.027) for the same-day IQR increase - Warm months: 1.013 (1.003, 1.024) - Cool months: 0.995 (0.976, 1.015) (c) 10-14 y, 1.012 (.0998, 1.027) for the same-day IQR increase - Warm months: 1.001 (0.987, 1.024) - Cool months: 1.017 (0.991, 1.044) Two-pollutant model with NO_2 (a) 1-4 y, 1.008 (1.001, 1.015) for the same-day IQR increase (b) 5-9 y, 1.016 (1.006, 1.026) for the same-day IQR increase (c) 10-14 y, 1.011 (0.999, 1.024) for the same-day IQR increase
Tecer et al. (2008) [12]	Dec 31, 2004-Oct 31, 2005	Zonguldak, Turkey	< 15 y, 187 patients	1 Fixed site; a daily arithmetic mean was calculated and used	HA	CCD	Conditional logistic regression	29.1 (NA)	Single-pollutant model 1.25 (1.05, 1.50) for 4-d lag 10 $\mu g/m^3$ increase 1.37 (1.06, 1.76) for 4-d lag IQR increase
Halonen et al. (2010) [13]	Jan 1, 1998-Dec 31, 2004	Helsinki, Finland	Restricted to the warm season (May to Sep) < 15 y, 1972 patients	2 Fixed sites; a daily arithmetic mean was calculated and used	ED visits	TS	GAM with Poisson distribution	8.8[1]	Two-pollutant model with O_3 1.148 (1.038, 1.270) for 5-d average IQR increase

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Table 1. Continued from the previous page

Author (publication year) [Ref]	Study period	Location	Sample	Exposure assessment	Outcome	Study design	Statistical model	$PM_{2.5}$ arithmetic mean concentration ($\mu g/m^3$) (SD)	Major effect estimates (risk ratio) (95% CIs)
Silverman et al. (2010) [14]	Jan 1, 1999-Dec 31, 2006	New York City, USA	Restricted to the warm season (Apr to Aug) <6 y - Non-ICU admission: 15 185, - ICU admission: 1141 patients 6-18 y - Non-ICU admission: 10 332, - ICU admission: 994 patients	24 Fixed sites; a daily arithmetic mean was calculated and used	HA	TS	GLM with Poisson regression	13[1]	Single-pollutant model (a) <6 y, - Non-ICU: 1.14 (1.10, 1.19) for 2-d average IQR increase - ICU: 1.03 (0.91, 1.17) for 2-d average IQR increase (b) 6-18 y, - Non-ICU: 1.19 (1.11, 1.27) for 2-d average IQR increase - ICU: 1.26 (1.10, 1.44) for 2-d average IQR increase Two-pollutant model with O_3 (a) <6 y, - Non-ICU: 1.13 (1.08, 1.18) for 2-d average IQR increase - ICU: 1.04 (0.91, 1.19) for 2-d average IQR increase (b) 6-18 y, - Non-ICU: 1.16 (1.08, 1.23) for 2-d average IQR increase - ICU: 1.23 (1.07-1.41) for 2-d average IQR increase
Strickland et al. (2010) [15]	Aug 1, 1998-Dec 31, 2004	Atlanta, USA	5-17 y, 91 386 patients	11 Fixed sites; a population-weighting average across monitors was calculated and used	ED visits	TS	GLM with Poisson regression	16.4 (7.4)	Single-pollutant model - Whole period: 1.020 (1.002, 1.039) for 3-d average IQR increase - Warm season: 1.043 (1.016, 1.070) for 3-d average IQR increase - Cold season: 1.005 (0.978, 1.031) for 3-d average IQR increase
Li et al. (2011) [16]	Jan 1, 2004-Dec 31,2006	Detroit, USA	2-18 y, 7063 patients	4 Fixed sites; a daily arithmetic mean was calculated and used	ED visits + HA[2]	TS and CCD	GAM and conditional logistic regression	15.0 (7.9)	Single-pollutant model 1.030 (1.001, 1.061) for 5-d average IQR increase in TS 1.039 (1.013, 1.066) for 5-d average IQR increase in CCD
Glad et al. (2012) [17]	Jan 1, 2002-Dec 31, 2005	Pittsburgh, USA	0-17 y, 978 patients	2 Fixed sites; a daily arithmetic mean was calculated and used	ED visits	CCD	Conditional logistic regression	NA	Single-pollutant model 1.012 (0.916, 1.118) for the same-day 10 $\mu g/m^3$ increase
Iskandar et al. (2012) [18]	May 15, 2001-Dec 31, 2008	Copenhagen, Denmark	0-18 y, 6329 patients	1 Fixed site; a daily arithmetic mean was calculated and used	HA	CCD	Conditional logistic regression	10.3 (5.4)	Single-pollutant model 1.09 (1.04, 1.13) for 5-d average IQR increase Two-pollutant model with NO_2: 1.06 (1.02, 1.11) for 5-d average IQR increase
Winquist et al. (2012) [19]	Jan 1, 2001-Jun 27, 2007	St. Louis, USA	0-1 y, - ED: 12 236 patients 2-18 y, - ED: 49 978 patients - All HA: 7095 patients	1 Fixed site; a daily arithmetic mean was calculated and used	ED visits & HA	TS	GLM with Poisson regression	14.4 (7.5)	Single-pollutant model (a) 0-1 y, - ED: 1.047 (0.999, 1.097) for 5-d average IQR increase (b) 2-18 y, - ED: 1.050 (1.021,1.080) for 5-d average IQR increase - HA: 1.052 (0.985, 1.123) for 5-d average IQR increase

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Table 1. Continued from the previous page

Author (publication year) [Ref]	Study period	Location	Sample	Exposure assessment	Outcome	Study design	Statistical model	$PM_{2.5}$ arithmetic mean concentration ($\mu g/m^3$) (SD)	Major effect estimates (risk ratio) (95% CIs)
Delfino et al. (2014) [20]	Jan 1, 2000-Dec 31, 2008	California, USA	0-18 y, 11 390 patients	Subject addresses were geocoded; using a modified, California LINE Source Dispersion Model, version. 4 to estimate pollutants at each residence	ED visits + HA[2]	CCD	Conditional logistic regression	- Warm season: 16.0 (9.5) - Cool season: 19.0 (13.8)	Single-pollutant model - Warm season: 1.079 (1.008, 1.154) for 7-d average IQR increase - Cool season: 1.162 (1.076, 1.254) for 7-d average IQR increase
Gleason et al. (2014) [21]	Jan 1, 2004-Dec 31, 2007	New Jersey, USA	3-17 y, 21 854 patients	Subject addresses were geocoded; using 12×12-km grid from the Multi-Scale Air Quality Model to estimate pollutants at each residence	ED visits	CCD	Conditional logistic regression	NA	Single-pollutant model 1.03 (1.02, 1.04) for the same day IQR increase Multipollutant model with O_3 and other pollens 0.99 (0.98, 1.01) for the same day IQR increase
Hua et al. (2014) [32]	Jan 1, 2007-Jul 31, 2012	Shanghai, China	0-14 y, 114 673 patients	1 Fixed site; a daily arithmetic mean was calculated and used	HA	TS	Polynomial distributed lag model	40.9 (27.7)	Single-pollutant model 1.04 (1.02, 1.05) for IQR increase with a maximum lag of 3-d 1.06 (1.05, 1.08) for IQR increase with a maximum lag of 5-d Multipollutant model with NO_2 and SO_2 1.03 (1.02, 1.05) for IQR increase with a maximum lag of 3-d 1.06 (1.04, 1.08) for IQR increase with a maximum lag of 5-d
Strickland et al. (2014) [22]	Jan 1, 2002-Jun 30, 2010	Atlanta, USA	2-16 y, 109 758 patients	6 Fixed sites; a population-weighting average across monitors calculated and used	ED visits	TS	GLM with Poisson regression	13.3 (5.4)	Single-pollutant model 1.032 (1.019, 1.044) for 3-d average IQR increase Two-pollutant model with O_3 1.022 (1.009, 1.035) for 3-d average IQR increase
Wendt et al. (2014) [23]	Jan 1, 2005-Dec 31, 2007	Boston, USA	0-17 y - May to Oct: 6061 patients - Nov to Apr: 7894 patients	3 Fixed sites; a daily arithmetic mean was calculated and used	HA	CCD	Conditional logistic regression	15.0 (6.0)	Single-pollutant model - May to Oct: 1.10 (1.03, 1.17) for 6-d average IQR increase - Nov to April: 1.06 (1.00, 1.14) for 6-d average IQR increase Two-pollutant model with NO_2 - May to Oct: 1.13 (1.04, 1.24) for 6-d average IQR increase - Nov to Apr: 1.00 (0.93, 1.07) for 6-d average IQR increase

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Table 1. Continued from the previous page

Author (publication year) [Ref]	Study period	Location	Sample	Exposure assessment	Outcome	Study design	Statistical model	$PM_{2.5}$ arithmetic mean concentration ($\mu g/m^3$) (SD)	Major effect estimates (risk ratio) (95% CIs)
Byers et al. (2016) [24]	Jan 1, 2007-Dec 31, 2011	Indianapolis, USA	5-17 y, 33 981 patients	3 Fixed sites; a population-weighting average across monitors calculated and used	ED visits	TS	GLM with Poisson regression	13.6 (7.1)	Single-pollutant model - All seasons: 1.007 (0.986, 1.029) for 3-d average IQR increase - Apr to Sep: 0.985 (0.934, 1.040) for 3-d average IQR increase - Oct to Mar: 0.976 (0.930, 1.025) for 3-d average IQR increase
Gleason et al. (2015) [25]	Jan 1, 2004-Dec 31, 2007	Newark, USA	3-17 y, 3675 patients	Subject addresses were geocoded; using grid from the Multi-Scale Air Quality Model to estimate pollutants at each residence	ED visits	TS and CCD	GLM and conditional logistic regression	NA	Single-pollutant model 1.00 (0.96, 1.05) for 3-d average IQR increase in TS 1.00 (0.96, 1.04) for 3-d average IQR increase in CCD Multipollutant model with O_3 and other pollens 0.93 (0.89, 0.98) for 3-d average IQR increase in TS 0.95 (0.91, 1.00) for 3-d average IQR increase in CCD
Strickland et al. (2015) [26]	Jan 1, 2002-Jun 30, 2010	Georgia, USA	2-18 y, 189 816 patients	Subject addresses were geocoded; using a two-stage model that includes land use parameters and satellite aerosol optical depth measurements at 1-km resolution to estimate pollutants	ED visits	CCD	Conditional logistic regression	12.9[1]	Single-pollutant model 1.013 (1.003, 1.023) for the same day 10 $\mu g/m^3$ increase
Alhanti et al. (2016) [27]	Jan 1, 2006-Dec 31, 2009	Dallas, USA	0-4 y, mean daily counts: 16.91 patients 5-18 y, mean daily counts: 25.75 patients	All available monitors; the monitoring data were first spatially interpolated across the study's geographic domain and then a population-weighted average across monitors calculated and used	ED visits	TS	GLM with Poisson regression	11.1 (4.7)	Single-pollutant model 0-4 y, 0.98 (0.94, 1.02) for 3-d average IQR increase 5-18 y, 0.99 (0.95, 1.03) for 3-d average IQR increase
Weichenthal et al. (2016) [28]	Jan 1, 2004-Dec 31, 2011	Ontario, Canada	Total; 127 836 patients, <9 y, NA	Fixed site in Ontario which is part of Canada's National Air Pollution Surveillance network; a daily arithmetic mean was calculated and used	ED visits	CCD	Conditional logistic regression	7.1 (6.3)	Single-pollutant model 1.072 (1.042, 1.100) for 3-d average IQR increase

Ref, reference number; HA, hospital admission; ED, emergency department; GLM, generalized linear model; GAM, generalized additive model; NA, not available; IQR, interquartile range; TS, time series; CCD, case-crossover design; PM, particulate matter; SD, standard devaition; CI, confidence interval; ICU, intensive care unit; CO, carbon monoxide; SO_2, sulfur dioxide; NO_2, nitrogen dioxide; O_3, ozone.
[1]Median value of the daily $PM_{2.5}$ distribution during the entire study period. This study doesn't present the arithmetic mean of $PM_{2.5}$.
[2]The authors regarded asthma morbidity as hospital encounters which counted both HA and ED visits.

After extracting all effect estimates and CIs from the main body of each research paper and its supplementary materials, we broke it down to a total of 244 effect estimates. Of those, we selected 33 representative effect estimates from each study to use in our primary meta-analysis.

Primary Meta-analysis

In the random effects model, we were able to find that when the concentration of $PM_{2.5}$ increased by 10 μg/m^3, the risk of a child's hospital admission or ED visit increased by 4.8% (RR, 1.048; 95% CI, 1.028 to 1.067). The I-squared value, which shows the heterogeneity of the included studies, was 95.6%, a high figure. We presented a forest plot for the included effect estimates and pooled estimates (Figure 2).

Publication Bias

To schematically examine the tendency toward publication bias, we found a relatively symmetrical shape in the funnel plot and confirmed that there was not much of a bias because there was not statistically significant (p=0.42) in the Egger's test (Figure 3).

Category-specific Meta-analyses

We found that the effects are greater on children below the age of five than on children ages 5 to 19, in warmer seasons, and in North America and Europe than in Asia. The pooled effect estimates extracted through the multi-pollutant model was also statistically significant (RR, 1.040; 95% CI, 1.022 to 1.057). According to the lags, the effect changed greatly from

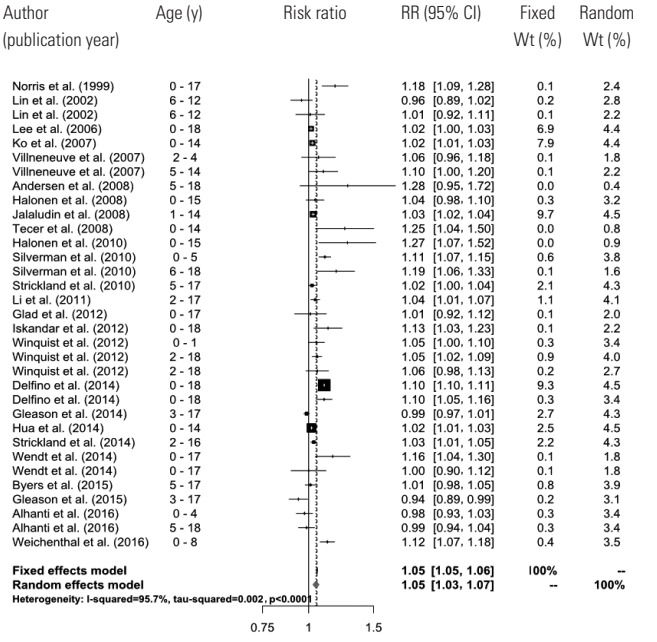

Figure 2. Forest plot for selected effect estimates in primary meta-analysis. RR, relative risk; CI, confidence interval; Wt, weight.

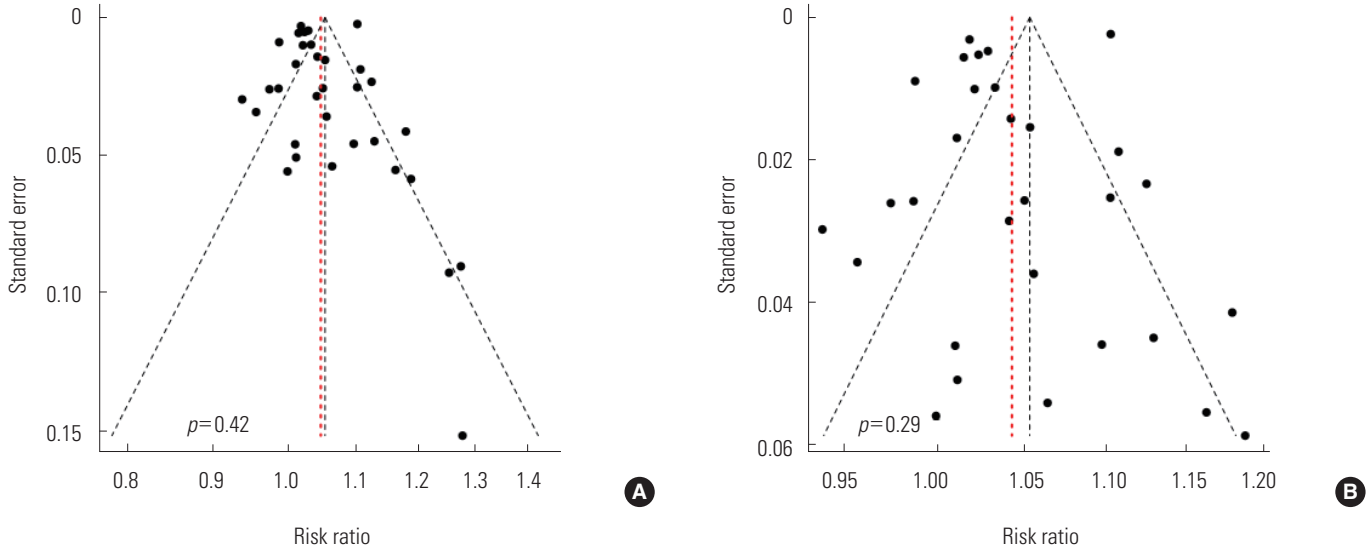

Figure 3. Funnel plot for a possible selection bias in the primary meta-analysis (A). After removing three estimates (Anderson et al. [8], Tecer et al. [12], and Halonen et al. [13]) from the right-lower area in A, still symmetrical shape is shown (B). Each black circle denotes each effect estimate of the selected studies, and the vertical red dotted line denotes the pooled random effect risk ratio in the primary meta-analysis. The p-value is derived from Egger's test.

Table 2. Results of category-specific meta-analyses

	No. of study (no. of estimate)	RR (95% CIs)[1]	I^2 (%)
Age[2]			
<5	7 (9)	1.044 (1.017, 1.071)	81.9
5-18	12 (15)	1.027 (1.011, 1.043)	76.8
Outcome			
HA	10 (15)	1.048 (1.029, 1.067)	77.7
ED visits	15 (17)	1.027 (1.011, 1.044)	79.5
Season			
Cold	7 (8)	1.015 (0.994, 1.037)	57.1
Warm	9 (11)	1.085 (1.051, 1.119)	94.8
Study design			
TS	15 (19)	1.028 (1.015, 1.041)	76.9
CCD	13 (17)	1.051 (1.020, 1.084)	96.6
Area			
North America	14 (19)	1.047 (1.019, 1.076)	96.1
Europe	8 (11)	1.075 (1.030, 1.123)	65.9
China	3 (3)	1.019 (1.013, 1.025)	0.0
Multipollutant model			
No	25 (33)	1.054 (1.037, 1.071)	96.0
Yes	13 (18)	1.040 (1.022, 1.057)	83.1
Time lag (d)			
0 (same day)	12 (14)	1.018 (1.005, 1.028)	60.9
1	11 (13)	1.018 (1.005, 1.030)	59.6
2	8 (8)	1.002 (0.984, 1.021)	84.6
3	10 (11)	1.030 (1.015, 1.045)	66.6
4	4 (4)	1.016 (0.969, 1.065)	83.1
5	5 (6)	1.019 (0.975, 1.065)	93.5
Average			
2	3 (7)	1.065 (1.020, 1.113)	81.7
3	11 (15)	1.019 (1.006, 1.033)	82.2
5	10 (14)	1.025 (1.007, 1.043)	77.4
6	3 (5)	1.029 (0.938, 1.129)	69.9

RR, relative risk; CI, confidence interval; HA, hospital admission; ED, emergency department; TS, time-series; CCD, case-crossover design.
[1]Calculated by DerSimonian and Laird random effects model [33].
[2]There are two exceptions: Silverman et al. [14] and Iskandar et al. [18]: cut-off age is six.

0.2% to 6.5%, and the effect was large for 3-day lag and 3-day average lag (Table 2).

Meta-regression Analyses

We did not find a tendency toward change in the statistically significant RR according to the time of study and the standard deviation of the background concentration of the region of study. We found a negative tendency in the mean $PM_{2.5}$ concentration by the region of study, but it was not statistically significant (β=-0.0008, p=0.14) (Figure 4).

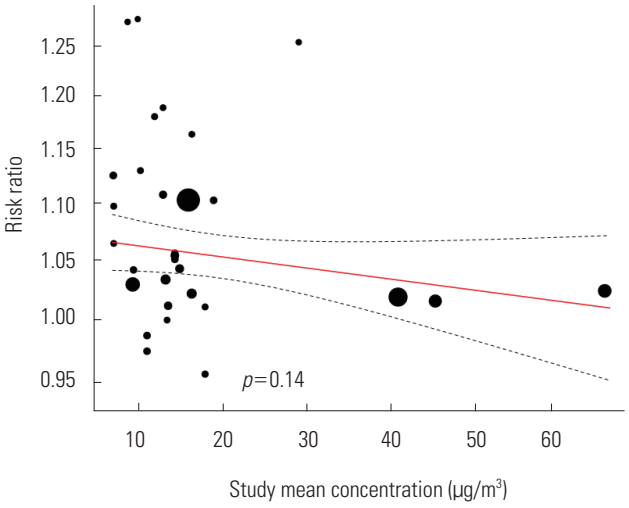

Figure 4. Bubble plot and regression line for mixed-effect meta-regression of study mean fine particulate matter ($PM_{2.5}$) concentration and effect estimate. The black circles denote each effect estimate and their sizes represent each weight. The bold red line indicates a linear relationship between study mean $PM_{2.5}$ concentration and relative risk and the black dotted lines indicate a 95% confidence interval.

DISCUSSION

In the primary meta-analysis of the effect estimates obtained from the 26 studies, we found that in the short-term, when the concentration of $PM_{2.5}$ increased 10 μg/m^3, the risk of a child's hospital admission or ED visit increased 4.8%, which is statistically significant. The effect of $PM_{2.5}$ could be considered quite robust, since the effect was maintained to 4.0% even when we pooled the estimates extracted by the multi-pollutant model in this study. This number is greater than the 2.3% found among the total population presented in the aforementioned study of Zheng et al. [5]. These results show that children are more vulnerable to air pollution because their alveoli and airways are still growing, their immune systems are underdeveloped, and they spend more time outdoors, which increases ventilation [38].

Based on known biological mechanisms, the generation of reactive oxygen species (ROS) is accelerated because of the transition metal included in $PM_{2.5}$. Oxidative stress from ROS may be related to epithelial cell destruction and allergic inflammation, and this process is known to be related to exacerbation of asthma [39]. Meanwhile, previous studies reported

that arginase may participate in a process that fine particles exacerbate childhood asthma [40]. *In vivo* studies report that the overexpression of arginase influences the hyperresponsiveness of airways [41] and that fine particles exacerbate the airway's responsiveness in asthma in murine models [42]. Human epidemiological studies have shown that the variation of the ARG1 and ARG2 genes—which are related to the manifestation of arginase in childhood asthma patients—is statistically significant [40,43].

In the preceding meta-analyses by Zheng et al. [5], they suggested 20 relevant studies on children's asthma. We found a discrepancy between the selected studies of Zheng et al. [5] and ours even aside from seven papers published more recently. They cited several studies that we excluded in the process of extracting eligible studies. On the other hand, the six studies included in this study were not cited by the preceding study. We selected studies and extracted results carefully focusing on children. Therefore, we believe that the 26 references selected for this study comprise the best selection.

We found that when the concentration of $PM_{2.5}$ increased by 10 μg/m^3, the risk of a child's hospital admission or ED visit increased by 4.8%. This value is greater than the 2.5% increase in children found in the preceding meta-analysis by Zheng, et al. [5]. The following are some reasons to explain this difference. First, the newly added original studies included several studies in which the RR exceeded 1.10 when the measure of effect estimates was converted to 10 μg/m^3 per increase [7,9,13,20,23,28]. Second, while the previous study pooled the effect estimates from the 0-day, 1-day, or 2-day average lags, we used the model with the greatest effect size out of the lags reported in the original studies.

In this study, we found a difference in RR according to the season, and during the warmer seasons, the RR was 1.085 (95% CI, 1.051 to 1.119). The studies included in our meta-analysis showed quite consistent results [9,20,22,23,31]. We thought the reason for this was that during warmer seasons, children spend more time outdoors and therefore spend more time exposed to $PM_{2.5}$. In addition, greater ventilation of buildings during these seasons makes it easier for air pollutants to penetrate inside the buildings. It was reported that the individual exposure concentration of $PM_{2.5}$ that people living in well-ventilated environments showed high correlation to the concentration of the atmosphere [44]. The difference in components of $PM_{2.5}$ according to the season may also be related, but because the extent of heterogeneity by region is too great, the evidence is not yet definitive [45-47].

In terms of the design of the studies, the pooled RRs for the time-series and the case-crossover design studies were 1.028 and 1.051, respectively. For the case-crossover design, the OR was calculated using the conditional logistic regression model. Compared to the RR, the OR has a tendency to overestimate the actual risk. However, it may be thought as a closer representation of reality than the exposure assessment of the time-series because a recently published case-crossover study more accurately matched air pollutants using the addresses of individuals [20,21,25,26]. Residential information of patients entering hospitals or visiting the ED cannot be reflected in time-series. If we suppose that $PM_{2.5}$ having an influence on exacerbating asthma as true, even in one study region, there is a possibility that the large effect in certain area with a high concentration could be diluted because of smaller effects in other area with a low concentration. We think that the actual effect is somewhere between the RRs of the time-series studies and the ORs of the case-crossover studies.

When we examine the pooled RR of each lag, we can see that there is up to a 6% difference in value depending on the type of model. The effects of both the concentration three days before (3-day lag) and the average concentration over three days (3-day average lag) were considerable. This result is somewhat difficult to interpret. We need to consider the following factors when dealing with lags: that the ethnicities of the subject of study differ by regions and an accessibility to health services could change depending on the time of study. Through meta-regression analysis, we found a negative tendency among effect sizes depending on the mean concentration of $PM_{2.5}$, but it was not statistically significant. Aside from the three studies in China which the mean concentrastion exceeded 30 μg/m^3 (Figure 4), we did not find a negative tendency in the meta-regression analysis (β=-0.0004, p=0.90). Therefore, we could not draw conclusions in this study regarding such a limited tendency. A negative tendency means that the effect on asthma is smaller for regions where the mean concentrations of $PM_{2.5}$ are higher. This means that the relationship between the mean concentration and the childhood asthma could be non-linear, or more specifically, supra-linear.

When we examine the results according to region in the category-specific analyses, the pooled RR of the three studies conducted in Shanghai and Hong Kong was 1.019, which is a smaller value than those in North America (1.047) and Europe (1.075). This is similar to the results of the previous meta-anal-

ysis that examined the short-term effects of $PM_{2.5}$ on total mortality and cardiorespiratory mortality, and found that the pooled estimate in China was lower than in the US, Europe, Japan, and Australia [48]. A hypothesis that the components of $PM_{2.5}$ in China are different from those of developed countries was raised regarding this finding. In other words, in China, the contributions of coal combustion and desert dust—rather than exhaust from automobiles—were greater than in other regions.

However, in a preceding meta-regression analysis including studies on PM_{10} and cardiorespiratory mortality conducted in China only, a statistically significant negative tendency was reported regarding the association between the mean concentrations of study regions and the effect sizes [49]. A study conducted across 27 US regions also reported that the effect of $PM_{2.5}$ was greater in regions with lower background mean concentration, even though the result was not statistically significant [50]. In a cohort study on the effect of $PM_{2.5}$ on cardiorespiratory mortality, the risks with the concentration level formed a supra-linear shape [2]. Therefore, for the regional effect variation in this study, the hypothesis that the effect was lowered in high concentrations seems more plausible, since only groups with resistance remain and detrimental effects on individuals vulnerable to $PM_{2.5}$ occur in lower concentrations.

There are many other genetic and environmental factors reported to cause childhood asthma besides $PM_{2.5}$. Another hypothesis, following hygiene theory, states that allergic reactions decrease when children are exposed to micro-organisms because immune reactions are suppressed. Since westernization is still in progress in China, the effects of $PM_{2.5}$ on asthma may be small [51,52]. There may be an objection to this statement since the three Chinese studies included in this study were conducted in Shanghai and Hong Kong, two very westernized large cities, but the infrastructure of the residences and the lifestyles of children growing up in such regions are different from those of North America and Europe.

There are some limitations of this study. First, outpatient visits, use of inhalers, and other symptom outbreaks could all be considered health effects and consequences, but we confined the results to hospital admissions and ED visits which were mainly reported in previous studies. Therefore, the pooled effect estimate reported in our study might be underestimated. But in a study that uses surveys on symptoms and use of inhalers, the period between the exposure and outbreak could be imprecise. Moreover, results from a survey could be subjective. In cases of outpatient visits, we cannot exclude periodic follow-up cases. Second, we combined the RRs with the ORs because we deemed the OR to be proxy to the RR. Because of this, we may have calculated an overestimated value rather than the actual risk. However, in the case of Korea, hospitalization due to asthma among children between the ages of zero to 19 was 0.14% in 2014 [53]. The frequency of hospital admissions or ED visits due to asthma is rare so a possible bias will be negligible. Third, we could not control the innate heterogeneity of the selected studies. Components of $PM_{2.5}$, ethnicities of the study population, and accessibility to health service as well as different age range, season, and adjusting variables or parameters in statistical models all probably affected the heterogeneity of the studies. However, we did not find a significant decrease of heterogeneity (Table 2). In order to obtain a more accurate pooled effect estimate, a meta-analysis should be conducted after an in-depth examination of the methods and quality of research.

The strength of this study is that we newly included seven recent studies in our meta-analysis. In addition, with a focus on children, we examined variations in effect of different possible factors, and presented the direction for future studies. In particular, we raised the need for an epidemiological study on regions besides China with high concentrations of $PM_{2.5}$.

CONCLUSION

We found that in the short-term, when the concentration increased by 10 $\mu g/m^3$, the risk of a child's hospital admission or ED visit increased by 4.8%. If we consider the fact that air pollution affects a vast range of regions and many populations, this is not a negligible figure. A more fundamental solution is the reduction of the matter from emission sources, so we need to conduct studies on sources that emit $PM_{2.5}$ and draft feasible environment-friendly policies for such emission sources.

ACKNOWLEDGEMENTS

This study was supported by the R&D Program for Society of the National Research Foundation funded by Ministry of Science, ICT and Future Planning, Republic of Korea (grant no. 2014M3C8A5030619).

CONFLICT OF INTEREST

The authors have no conflicts of interest associated with the material presented in this paper.

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RESIDUES OF FIRE ACCELERANT CHEMICALS

VOLUME II: LITERATURE SEARCH

Prepared for:

Intermountain Region
USDA Forest Service
Ogden, UT

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October 17, 2002

LITERATURE SEARCH: RESIDUES OF FIRE ACCELERANT CHEMICALS

Structure of Report

This report presents the results of a literature search designed to identify data points for a quantitative risk assessment of the residues remaining after the use of fire accelerants to ignite prescribed burns.

Table 1, Chemicals List, presents the fire accelerants, their chemical components, and the residues expected to remain following combustion. Each of the following sections present the literature search for one of the residues, consisting of a table of data parameters followed by abstracts or summaries of the literature cited for that chemical.

The residues evaluated are as follows:

Table 1-1. Chemicals Evaluated in Risk Assessment

Accelerant	Components	Residues*
Fusee	Strontium nitrate + Potassium perchlorate + Sulfur + Sawdust/oil binder	Strontium sulfate Strontium oxide Strontium sulfide *Nitrogen oxides* Potassium chloride Potassium hydroxide *Carbon dioxide* *Water vapor* *Sulfur dioxide*
Gasoline	Gasoline mixture + MTBE (additive)	Gasoline as a mixture MTBE
Diesel fuel	Diesel fuel mixture	Diesel fuel as a mixture
Firegel/Alumagel/Suregel/ Petrol Jel	Aluminum carboxylates	Aluminum oxide *Water vapor*
Ping-pong balls	Potassium permanganate + Ethylene glycol + Polystyrene ball	Manganese dioxide Potassium hydroxide *Carbon dioxide* *Water vapor* *Styrene* Uncombusted polystyrene
Flares propelled by launcher pistols	Aluminum + Calcium sulfate + Iron oxide + Copper oxide + Silicon + Potassium perchlorate + Lead oxide + Black powder: (Potassium nitrate + Sulfur + Charcoal)	Aluminum Aluminum oxide Calcium sulfate Iron oxide Copper oxide Silicon dioxide Potassium chloride *Carbon dioxide* *Water vapor* Lead Potassium hydroxide *Nitrogen oxides* *Sulfur dioxide*
Propane	Propane	*Carbon dioxide* *Water vapor*

*Gaseous compounds are presented in italics; they are not analyzed in this assessment.
Sources: Etiumsoft 2002, Lewis 1994a, Lewis 1994b, Sumi and Tsuchiya 1971.

Free aluminum is reactive. Following combustion, aluminum oxide will be the dominant form.

Aluminum Oxide, CAS #1344-28-1 (Al_2O_3, aluminum trioxide, alumina)

Data Point	Data Summary	Reference
Water solubility	Practically insoluble 0.000098 g/100 cc = 0.0000098 mg/L	Budavari et al. 1989 ATSDR 1999
K_{oc}		
Soil half-life	No degradation.	ATSDR 1999
BCF	BCFs are less than 300 in fish, since aluminum is highly toxic to fish species.	ATSDR 1999
Ingestion toxicity	A minimal risk level of 2.0 mg/kg/day was estimated for intermediate (15 to 364 days) oral exposure, based on the most sensitive toxicity endpoint (neurotoxicity) identified in studies in laboratory animals.	ATSDR 1999
Carcinogenicity	Chronic ingestion studies in mice and rats using aluminum potassium sulfate or aluminum phosphide led reviewers to conclude that aluminum has not demonstrated carcinogenicity in laboratory animals.	ATSDR 1999
Mammalian tox	Oral LD_{50}s are 162 and 164 mg/kg in rat and mouse, respectively.	ATSDR 1999
Avian tox	14-day LD_{50} >8,000 mg/kg in northern bobwhite and 4,997 in Japanese quail for monoethyl ester phosphonic acid aluminum salt (CAS # 39148-24-8), equivalent to >2,303 and 1,439 mg Al_2O_3/kg, respectively.	EPA 2002
Fish toxicity	96-hour LC_{50} in rainbow trout = 0.310 mg Al/L, equal to 1.17 mg Al_2O_3/L	EPA 2002
Aq. invert. tox	24-hour LC_{50}s in water fleas (Daphnia spp.) were 2.6 and 3.5 mg/L	EPA 2002
Aq. amph. tox	96-hour LC_{50} for aluminum in Jefferson salamander embryos is approximately 0.38 mg/L, equivalent to 1.4 mg Al_2O_3/L LC_{10} (NOEC) was 0.3 mg/L, 24-hour LC_{50} was 0.5 mg/L, LC_{100} was 0.7 mg/L for aluminum in common toad embryos 7-day LC_{50} for aluminum in eastern narrowmouth toad embryo-larvae was 0.05 mg/L	Pauli et al. 2000

Agency for Toxic Substances and Disease Registry. 1999. Toxicological profile for aluminum. Atlanta, GA. http://www.atsdr.cdc.gov/toxprofiles/tp22.html

ATSDR. See Agency for Toxic Substances and Disease Registry.

Budavari, S., M.J. O'Neil, A. Smith, and P.E. Heckelman, eds. 1989. *The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals.* Merck and Co., Inc. Rahway, NJ.

EPA. See U.S. Environmental Protection Agency.

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters, Hull, Québec, Canada. http://www.cws-scf.ec.gc.ca/nwrc/ratl/about_e.htm

U.S. Environmental Protection Agency. 2002. Ecotox database: Aluminum. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN. http://www.epa.gov/ecotox/

Agency for Toxic Substances and Disease Registry. 1999. Toxicological profile for aluminum. Atlanta, GA.

Report summarized by ATSDR in the form of ToxFAQs document; relevant sections follow:

ToxFAQs™ for Aluminum, CAS# 7429-90-5, June 1999

HIGHLIGHTS: Everyone is exposed to low levels of aluminum from food, air, and water. Exposure to high levels of aluminum may result in respiratory problems.

Aluminum occurs naturally and makes up about 8% of the surface of the earth. It is always found combined with other elements such as oxygen, silicon, and fluorine.

What happens to aluminum when it enters the environment? It binds to particles in the air. It can dissolve in lakes, streams, and rivers depending on the quality of the water. Acid rain may dissolve aluminum from soil and rocks. It can be taken up into some plants from soil. It is not known to bioconcentrate up the food chain.

How might I be exposed to aluminum? Eating small amounts of aluminum in food. Breathing higher levels of aluminum dust in workplace air. Drinking water with high levels of aluminum near waste sites, manufacturing plants, or areas naturally high in aluminum. Eating substances containing high levels of aluminum (such as antacids) especially when eating or drinking citrus products at the same time. Very little enters your body from aluminum cooking utensils.

How can aluminum affect my health? Low-level exposure to aluminum from food, air, water, or contact with skin is not thought to harm your health. Aluminum, however, is not a necessary substance for our bodies and too much may be harmful. People who are exposed to high levels of aluminum in air may have respiratory problems including coughing and asthma from breathing dust. Some studies show that people with Alzheimer's disease have more aluminum than usual in their brains. We do not know whether aluminum causes the disease or whether the buildup of aluminum happens to people who already have the disease. Infants and adults who received large doses of aluminum as a treatment for another problem developed bone diseases, which suggests that aluminum may cause skeletal problems. Some sensitive people develop skin rashes from using aluminum chlorohydrate deodorants.

How likely is aluminum to cause cancer? The Department of Health and Human Services, the International Agency for Research on Cancer, and the EPA have not classified aluminum for carcinogenicity. Aluminum has not been shown to cause cancer in animals.

How does aluminum affect children? Children with kidney problems who were given aluminum in their medical treatments developed bone diseases. Other health effects of aluminum on children have not been studied. It is not known whether aluminum affects children differently than adults, or what the long-term effects might be in adults exposed as children. Large amounts of aluminum have been shown to be harmful to unborn and developing animals because it can cause delays in skeletal and neurological development. Aluminum has been shown to cause lower birthweights in some animals.

Budavari, S., M.J. O'Neil, A. Smith, and P.E. Heckelman, eds. 1989. *The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals.* Merck and Co., Inc. Rahway, NJ.

359. Aluminum Oxide. Alumina. Al_2O_3; mol wt 101.94. Al 52.91%, O 47.08%. Occurs in nature as the minerals: **bauxite, bayerite, boehmite, corundum, diaspore, gibbsite**. Prepn and properties: *Mellor's* vol. V, 263-273 (1929); *Gmelin's. Aluminum* (8^{th} ed.) **35B**, pp 7-98 (1934); Becher in *Handbook of Preparative Inorganic Chemistry* vol. 1, G. Grauer. Ed. (Academic Press, New York, 2^{nd} ed., 1963) pp 822-823; Wagner, *ibid*. vol. 2 (1965) pp 1660-1663.

Approximate characteristics of native aluminum oxide: White cryst powder. d^{20}_4 4.0 mp about 2000°. Very hard, about 8.8 on Moh's scale. An electrical insulator; electrical resistivity at 300° about 1.2×10^{13} ohms-cm. Practically insol in water. Slowly sol in aq alkaline solns with the formation of hydroxides. Practically insol in non-polar organic solvents.

USE: As adsorbent, dessicant, abrasive; as filler for paints and varnishes; in manuf of alloys, ceramic materials, electrical insulators and resistors, dental cements, glass, steel, artificial gems; in coatings for metals, etc.; as catalyst for organic reactions. The minerals *corundum* (hardness = 9) and ***Alundium*** (obtained by fusing bauxite in an electric furnace) are used as abrasives and polishes; in manuf of refractories. Aluminum oxide is also used in chromatography , *see* Aluminum Oxide (Brockmann).

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters, Hull, Québec, Canada.

> The RATL (Reptile and Amphibian Toxicology Literature) database contains data extracted from the primary literature for amphibian and reptile ecotoxicology studies published up to and including 1997; there are some data from studies published in 1998 and 1999. As of September, 2000, there was approximately 2000 references in the database. Citations were gathered through searches of various literature databases, but these searches concentrated on the environmental pollution literature with the result that the bibliography cannot be considered exhaustive.

U.S. Environmental Protection Agency. 2002. Ecotox database: Aluminum. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN.

The ECOTOXicology database is a source for locating single chemical toxicity data for aquatic life, terrestrial plants and wildlife. ECOTOX integrates three toxicology effects databases: AQUIRE (aquatic life), PHYTOTOX (terrestrial plants), and TERRETOX (terrestrial wildlife). These databases were created by the U.S. EPA, Office of Research and Development (ORD), and the National Health and Environmental Effects Research Laboratory (NHEERL), Mid-Continent Ecology Division.

Scientific name, Common name	**End-point**	**Effect**	**Trend --- Effect %**	**Media Type**	**Duration --- Exp Typ**	**Conc (ug/L)**	**Signif --- Level**	**Response Site --- BCF**	**Ref #**
Daphnia magna Water flea	LC50	MOR	--------	FW	24 H ----------	F 3500		------	3936
Daphnia pulex Water flea	LC50	MOR	--------	FW	24 H ----------	F 2600		------	3936
Oncorhynchus mykiss Rainbow trout,donaldson trout	LC50	MOR	INC --------	FW	96 H ---------- S	T 310		------	14405

Calcium Sulfate, CAS #7778-18-9 ($CaSO_4$, plaster of Paris, gypsum)

Data Point	Data Summary	Reference
Water solubility	3,000 mg/L	HSDB 2002
K_{oc}	No data.	
Soil half-life	Stable. Naturally occurring compound as gypsum.	HSDB 2002
BCF	No data.	
Ingestion toxicity	Substance added directly to human food affirmed as generally recognized as safe (GRAS).	21 CFR 184.1230
Carcinogenicity	Inhalation of calcium sulfate fibers resulted in tumors in laboratory animals.	HSDB 2002
Fish toxicity	96-hour LC_{50} in bluegill sunfish >2,980 mg/L	EPA 2002
Aq. invert. tox	24-hour LC_{50} in water flea *Daphnia magna* >1,970 mg/L	EPA 2002
Aq. amph. tox	No data.	

21 CFR 184.1230. Direct food substances affirmed as Generally Recognized as Safe--Listing of Specific Substances Affirmed as GRAS. Calcium sulfate. U.S. Food and Drug Administration.

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

U.S. Environmental Protection Agency. 2002. Ecotox database: Calcium sulfate. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN. http://www.epa.gov/ecotox/

21 CFR 184.1230. Direct food substances affirmed as Generally Recognized as Safe--Listing of Specific Substances Affirmed as GRAS. Calcium sulfate. U.S. Food and Drug Administration.

TITLE 21--FOOD AND DRUGS
CHAPTER I--FOOD AND DRUG ADMINISTRATION, DEPARTMENT OF HEALTH AND HUMAN SERVICES (CONTINUED)

PART 184--DIRECT FOOD SUBSTANCES AFFIRMED AS GENERALLY RECOGNIZED AS SAFE--
Table of Contents
Subpart B--Listing of Specific Substances Affirmed as GRAS
Sec. 184.**1230** Calcium sulfate.

(a) Calcium sulfate (CaSO<INF>4,</INF> CAS Reg. No. 7778-18-9 or CaSO<INF>4</INF><t-bullet>2H<INF>2</INF>O, CAS Reg. No. 10101-41-4), also known as plaster of Paris, anhydrite, and gypsum, occurs naturally and exists as a fine, white to slightly yellow-white odorless powder. The anhydrous form is prepared by complete dehydration of gypsum, below 300 deg.C, in an electric oven.

(b) The ingredient meets the specifications of the ``Food Chemicals Codex,'' 3d Ed. (1981), p. 66, which is incorporated by reference. Copies may be obtained from the National Academy Press, 2101 Constitution Ave. NW., Washington, DC 20418, or may be examined at the Office of the Federal Register, 800 North Capitol Street, NW., suite 700, Washington, DC 20408.

(c) The ingredient is used as an anticaking agent as defined in Sec. 170.3(o)(1) of this chapter, color and coloring adjunct as defined in Sec. 170.3(o)(4) of this chapter, dough strengthener as defined in Sec. 170.3(o)(6) of this chapter, drying agent as defined in Sec. 170.3(o)(7) of this chapter, firming agent as defined in Sec. 170.3(o)(10) of this chapter, flour treating agent as defined in Sec. 170.3(o)(13) of this chapter, formulation aid as defined in Sec. 170.3(o)(14) of this chapter, leavening agent as defined in Sec. 170.3(o)(17) of this chapter, nutrient supplement as defined in Sec. 170.3(o)(20) of this chapter, pH control agent as defined in Sec. 170.3(o)(23) of this chapter, processing aid as defined in Sec. 170.3(o)(24) of this chapter, stabilizer and thickener as defined in Sec. 170.3(o)(28) of this chapter, synergist as defined in Sec. 170.3(o)(31) of this chapter, and texturizer as defined in Sec. 170.3(o)(32) of this chapter.

(d) The ingredient is used in food at levels not to exceed good manufacturing practice in accordance with Sec. 184.1(b)(1). Current good manufacturing practice results in a maximum level, as served, of 1.3 percent for baked goods as defined in Sec. 170.3(n)(1) of this chapter, 3.0 percent for confections and frostings as defined in Sec. 170.3(n)(9) of this chapter, 0.5 percent for frozen dairy desserts and mixes as defined in Sec. 170.3(n)(20) of this chapter, 0.4 percent for gelatins and puddings as defined in Sec. 170.3(n)(22) of this chapter, 0.5 percent for grain products and pastas as defined in Sec. 170.3(n)(23) of this chapter, 0.35 percent for processed vegetables as defined in Sec. 170.3(n)(36) of this chapter, and 0.07 percent or less for all other food categories.

(e) Prior sanctions for this ingredient different from the uses established in this section do not exist or have been waived.
[45 FR 6086, Jan. 25, 1980; 45 FR 26319, Apr. 18, 1980, as amended at 49 FR 5611, Feb. 14, 1984]

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD.

HSDB is a toxicology data file on the National Library of Medicine's (NLM) Toxicology Data Network (TOXNET®). It focuses on the toxicology of potentially hazardous chemicals. It is enhanced with information on human exposure, industrial hygiene, emergency handling procedures, environmental fate, regulatory requirements, and related areas. All data are referenced and derived from a core set of books, government documents, technical reports and selected primary journal literature. HSDB is peer-reviewed by the Scientific Review Panel (SRP), a committee of experts in the major subject areas within the data bank's scope. HSDB is organized into individual chemical records, and contains over 4500 such records.

The following are the human health and environmental fate summaries from HSDB:

Human Health Effects:

Human Toxicity Excerpts:

GYPSUM DUST HAS AN IRRITANT ACTION ON MUCOUS MEMBRANES OF THE RESPIRATORY TRACT & EYES, & THERE HAVE BEEN REPORTS OF CONJUNCTIVITIS, CHRONIC RHINITIS, LARYNGITIS, PHARYNGITIS, IMPAIRED SENSE OF SMELL & TASTE, BLEEDING FROM THE NOSE, & REACTIONS OF TRACHEAL & BRONCHIAL MEMBRANES IN EXPOSED WORKERS. /GYPSUM/
[International Labour Office. Encyclopedia of Occupational Health and Safety. Volumes I and II. New York: McGraw-Hill Book Co., 1971. 630]**PEER REVIEWED**

Because it hardens quickly after absorbing moisture, its ingestion may result in obstruction, particularly at the pylorus. ... To delay "setting," drink glycerin or gelatin solutions, or large volumes of water. Surgical relief may be necessary. /Plaster of Paris/
[Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. II-127]**PEER REVIEWED**

... Calcium sulfate caused no lung disease in calcium sulfate miners.
[American Conference of Governmental Industrial Hygienists, Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. Volumes I,II, III. Cincinnati, OH: ACGIH, 1991. 204]**PEER REVIEWED**

Medical Surveillance:

IT IS ADVISABLE FOR GYPSUM & GYPSUM-PRODUCTS WORKERS TO RECEIVE A PRE-EMPLOYMENT EXAMINATION FOLLOWED BY PERIODIC EXAMINATIONS EACH YR. /GYPSUM/
[International Labour Office. Encyclopedia of Occupational Health and Safety. Volumes I and II. New York: McGraw-Hill Book Co., 1971. 630]**PEER REVIEWED**

Probable Routes of Human Exposure:

WORKERS EMPLOYED IN PROCESSING OF GYPSUM ROCK MAY BE EXPOSED TO HIGH ATMOSPHERIC CONCN OF GYPSUM DUST ... FURNACE GASES & SMOKE. IN GYPSUM CALCINATING, WORKERS ARE EXPOSED TO HIGH ENVIRONMENTAL TEMP, & THERE IS ALSO THE HAZARD OF BURNS. /GYPSUM/
[International Labour Office. Encyclopedia of Occupational Health and Safety. Volumes I and II. New York: McGraw-Hill Book Co., 1971. 630]**PEER REVIEWED**

Environmental Fate & Exposure:

Natural Pollution Sources:

NATURAL FORM OF ANHYDROUS CALCIUM SULFATE IS KNOWN AS MINERAL ANHYDRITE; ALSO AS KARSTENITE, MURIACITE, ANHYDROUS SULFATE OF LIME, ANHYDROUS GYPSUM.
[The Merck Index. 9th ed. Rahway, New Jersey: Merck & Co., Inc., 1976. 216]**PEER REVIEWED**

MINERAL WITH CHEM COMPOSITION CASO4.2H2O. ... IT IS RARELY FOUND PURE & GYPSUM DEPOSITS MAY CONTAIN QUARTZ, PYRITES, CARBONATES & CLAYEY & BITUMINOUS MATERIALS. IT OCCURS IN NATURE IN 5 VARIETIES: GYPSUM ROCK; GYPSITE ... ALABASTER ... SATIN SPAR ... & SELENITE. /GYPSUM/
[International Labour Office. Encyclopedia of Occupational Health and Safety. Volumes I and II. New York: McGraw-Hill Book Co., 1971. 630]**PEER REVIEWED**

Calcium sulfate is the commonest of the natural sulfates.
[Harben PW, Bates RL; Geology of the Nonmetallics p.237 (1984)]**PEER REVIEWED**

Domestic resources are adequate but are unevenly distributed. There are no gypsum deposits on the eastern seaboard of the United States. Large deposits occur in the Great Lakes region, mid-continent region, California, and other States. /Gypsum/
[BUREAU OF MINES. MINERAL COMMODITY SUMMARIES 1986 p.67]**PEER REVIEWED**

U.S. Environmental Protection Agency. 2002. Ecotox database: Calcium sulfate. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN.

The ECOTOXicology database is a source for locating single chemical toxicity data for aquatic life, terrestrial plants and wildlife. ECOTOX integrates three toxicology effects databases: AQUIRE (aquatic life), PHYTOTOX (terrestrial plants), and TERRETOX (terrestrial wildlife). These databases were created by the U.S. EPA, Office of Research and Development (ORD), and the National Health and Environmental Effects Research Laboratory (NHEERL), Mid-Continent Ecology Division.

Scientific name, Common name	Endpoint	Effect	Trend --------- Effect %	Media Type	Duration ---------- Exp Typ	Conc (ug/L)	Signif ------- Level	Response Site ------- BCF	Ref #
Daphnia magna Water flea	LC50	MOR	INC --------	FW	24 H ---------- S	T >1970000, >1970000 - >1970000		------	18272
Lepomis macrochirus Bluegill	LC50	MOR	INC --------	FW	96 H ---------- S	T 2980000		------	5683

Copper Oxide, CAS #1317-38-0

Data Point	Data Summary	Reference
Water solubility	Practically insoluble. In its Cu(II) state, copper forms coordination compounds or complexes with both inorganic and organic ligands. At the pH values and carbonate concentrations characteristic of natural waters, most dissolved Cu(II) exists as carbonate complexes rather than as free (hydrated) cupric ions.	HSDB 2002 ATSDR 1990
K_{oc}	No data	
Soil half-life	Copper is a stable element. Copper oxide may form complexes with soil or dissolve in water, depending on the pH and organic carbon content of the specific soil.	
BCF	The bioconcentration factor (BCF) of copper in fish obtained in field studies is 10- 100, indicating a low potential for bioconcentration.	ATSDR 1990
Ingestion toxicity	The mean daily dietary intake of copper in adults ranges between 0.9 and 2.2 mg 300 mg Cu/kg/day was the LOAEL causing death in weanling rats when administered over a period of 2 to 15 weeks. Equivalent to 376 mg CuO/kg/day.	HSDB 2002
Carcinogenicity	Inadequate data to determine carcinogenicity.	EPA 1991
Avian toxicity	500 mg Cu/kg caused adverse effects in the domestic chicken, equivalent to 626 mg CuO/kg. No LD_{50} for avian species was identified.	Eisler 1998
Fish toxicity	The 96-hour LC_{50} for rainbow trout was 25.4 mg/L.	EPA 2002
Aq. invert. tox	The 48-hour EC_{50} for intoxication for the water flea *Daphnia magna* was 0.011 to 0.039 mg/L. The 48-hour EC_{50} for mortality in *Ceriodaphnia dubia* was 0.028 mg Cu/L = 0.035 mg CuO/L.	EPA 2002
Aq. amph. tox	No data.	

Agency for Toxic Substances and Disease Registry. 1990. Toxicological profile for copper. Atlanta, GA. http://www.atsdr.cdc.gov/toxprofiles/tp132.html

ATSDR. See Agency for Toxic Substances and Disease Registry.

Eisler, R. 1998. Copper hazards to fish, wildlife, and invertebrates: a synoptic review. Biological Science Report USGS/BRD/BSR--1998-0002. U.S. Geological Survey, Biological Resources Division. Laurel, MD. http://www.pwrc.usgs.gov/new/chrback.htm

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

U.S. Environmental Protection Agency. 1991. Integrated risk information system. Office of Research and Development. Cincinnati, OH. http://www.epa.gov/iris/subst/0368.htm

U.S. Environmental Protection Agency. 2002. Ecotox database: Cupric oxide. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN. http://www.epa.gov/ecotox/

Agency for Toxic Substances and Disease Registry. 1999. Toxicological profile for copper. Atlanta, GA.

Report summarized by ATSDR in the form of ToxFAQs document; relevant sections follow:

HIGHLIGHTS: Copper is an element that is found naturally in the environment. Small amounts of copper are necessary for good health; however, very large amounts can cause dizziness, headaches, diarrhea, and liver and kidney damage.

What happens to copper when it enters the environment? Copper is emitted to the air through natural processes such as windblown dust and volcanic eruptions. Human activities such as copper smelting and ore processing also result in copper being released to the air. Copper may enter the air when it is applied as a fungicide to plants, wood, fabric, and leather. Copper is released to water as a result of natural weathering of soil. It may also be released to water from discharges from industries and sewage treatment plants. Copper may also be added to lakes and ponds to control algae.

How can copper affect my health? Copper is necessary for good health. However, very large doses can be harmful. Long-term exposure to copper in the air can irritate your nose, mouth, and eyes, and cause dizziness, headaches, and diarrhea. Eating or drinking very high amounts of copper can cause liver and kidney damage and effects on the blood. Drinking water with higher than normal levels of copper can cause vomiting, diarrhea, stomach cramps, and nausea. Skin contact with copper can result in an allergic reaction in some people. This reaction is usually skin irritation or a skin rash. Animal studies have shown effects on the stomach and abnormalities in development when animals were fed a diet high in copper. Copper has not been shown to cause cancer in people or animals. The International Agency for Research on Cancer (IARC) has determined that copper is not classifiable as to human carcinogenicity.

The EPA has set a treatment technique for copper in drinking water that includes an action level of 1.3 milligrams of copper per liter of water (1.3 mg/L). The EPA has also set a secondary maximum contaminant level (SMCL) of 1 mg/L of copper in drinking water. An SMCL is a nonenforceable drinking water standard based on taste, odor, or other aesthetic considerations.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD.

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The following are the human health and environmental fate summaries from HSDB:

Human Health Effects:

Toxicity Summary:

For healthy, non-occupationally-exposed humans the major route of exposure to copper is oral. The mean daily dietary intake of copper in adults ranges between 0.9 and 2.2 mg. ... In some cases, drinking water may make a substantial additional contribution to the total daily intake of copper, particularly in households where corrosive waters have stood in copper pipes. ... All other intakes of copper (inhalation and dermal) are insignificant in comparison to the oral route. Inhalation adds 0.3-2.0 ug/day from dusts and smoke. Women using copper IUDs are exposed to only 80ug or less of copper per day from this source. The homeostasis of copper involves the dual essentiality and toxicity of the element. Its essentiality arises from its specific incorporation into a large number of proteins for catalytic and structural purposes. The cellular pathways of uptake, incorporation into protein and export of copper are conserved in mammals and modulated by the metal itself. Copper is mainly absorbed through the gastrointestinal tract. From 20 to 60% of the dietary copper is absorbed, with the rest being excreted through the feces. Once the metal passes through the basolateral membrane it is transported to the liver bound to serum albumin. The liver is the critical organ for copper homeostatis. The copper is partitioned for excretion through the bile or incorporation into intra- and extracellular proteins. The primary route of excretion is through the bile. The transport of copper to the peripheral tissues is accomplished through the plasma attached to serum albumin, ceruloplasmin or low-molecular weight complexes. ... The biochemical toxicity of copper, when it exceeds homeostatic control, is derived from its effects on the structure and function of biomolecules, such as DNA, membranes and proteins directly or through oxygen-radical mechanisms. The toxicity of a single oral dose of copper varies widely between species. ... The major soluble salts (copper(II) sulfate, copper(II) chloride) are generally more toxic than the less soluble salts (copper(II) hydroxide, copper (II) oxide). Death is preceded by gastric hemorrhage, tachycardia, hypotension, hemolytic crisis, convulsions and paralysis. ... Long-term exposure in rats and mice showed no overt signs of toxicity other than a dose-related reduction in growth after ingestion ... The effects included inflammation of the liver and degeneration of kidney tubule epithelium. ... Some testicular degeneration and reduced neonatal body and organ weights were seen in rats ... and fetotoxic effects and malformations were seen at high dose levels. ... Neurochemical changes have been reported after oral administration ... A limited number of immunotoxicity studies showed humoral and cell-mediated immune function impairment in mice after oral intakes in drinking-water ... Copper is an essential element and adverse health effects /in humans/ are related to deficiency as well as excess. Copper deficiency is associated with anemia, neutropenia and bone abnormalities but clinically evident deficiency is relatively infrequent in humans. .. Except for occasional acute incidents of copper poisoning, few effects are noted in normal /human/ populations. Effects of single exposure following suicidal or accidental oral exposure have been reported as metallic taste, epigastric pain, headache, nausea, dizziness,

vomiting and diarrhea, tachycardia, respiratory difficulty, hemolytic anemia, hematuria, massive gastrointestinal bleeding, liver and kidney failure, and death. Gastrointestinal effects have also resulted from single and repeated ingestion of drinking-water containing high copper concentrations, and liver failure has been reported following chronic ingestion of copper. Dermal exposure has not been associated with systemic toxicity but copper may induce allergic responses in sensitive individuals. Metal fume fever from inhalation of high concentrations in the air in occupational settings have been reported ... A number of groups are described where apparent disorders in copper homeostasis result in greater sensitivity to copper deficit or excess than the general population. Some disorders have a well-defined genetic basis. These include Menkes disease, a generally fatal manifestation of copper deficiency; Wilson disease (hepatolenticular degeneration), a condition leading to progressive accumulation of copper; and hereditary aceruloplasminemia, with clinical symptoms of copper overload. Indian childhood cirrhosis and idiopathic copper toxicosis are conditions related to excess copper which may be associated with genetically based copper sensitivity ... These are fatal conditions in early childhood where copper accumulates in the liver. ... Other groups potentially sensitive to copper excess are hemodialysis patients and subjects with chronic liver disease. Groups at risk of copper deficiency include infants (particularly low birth weight/preterm babies, children recovering from malnutrition, and babies fed exclusively with cow's milk), people with maladsorption syndrome (e.g., celiac disease, sprue, cystic fibrosis), and patients on total parenteral nutrition. Copper deficiency has been implicated in the pathogenesis of cardiovascular disease. The adverse effects of copper must be balanced against its essentiality. Copper is an essential element for all biota ... At least 12 major proteins require copper as an integral part of their structure. It is essential for the utilization of iron in the formation of hemoglobin, and most crustaceans and molluscs possess the copper-containing hemocyanin as their main oxygen-carrying blood protein. ... A critical factor in assessing the hazard of copper is its bioavailablity. Adsorption of copper to particles and complexation by organic matter can greatly limit the degree to which copper will be accumulated ... At many sites, physiochemical factors limiting bioavailability will warrant higher copper limits. ...
[Environmental Health Criteria 200: Copper pp. 1-11 (1998) by the International Programme on Chemical Safety (IPCS) under the joint sponsorship of the United Nations Environment Programme, the International Labour Organisation and the World Health Organization.]**PEER REVIEWED**

Environmental Fate & Exposure:

Probable Routes of Human Exposure:

Exposure may occur in copper and brass plants and during the welding of copper alloys.
[NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 97-140. Washington, D.C. U.S. Government Printing Office, 1997. 76]**PEER REVIEWED**

Natural Pollution Sources:

OCCURS IN NATURE AS MINERALS TENORITE (TRICLINIC CRYSTALS) & PARAMELACONITE (TETRAHEDRAL, CUBIC CRYSTALS).
[The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 378]**PEER REVIEWED**

U.S. Environmental Protection Agency. 1991. Integrated risk information system. Office of Research and Development. Cincinnati, OH.

Status of Data for Copper

File First On-Line: 09/07/1988
Last Significant Revision: 09/07/1988
Category Status Last Revised
Oral RfD Assessment No data
Inhalation RfC Assessment No data
Carcinogenicity Assessment On-line 08/01/1991

Weight of Evidence (1986 US EPA Guidelines):
D (Not classifiable as to human carcinogenicity)

Weight of Evidence Narrative:
There are no human data, inadequate animal data from assays of copper compounds, and equivocal mutagenicity data.

This may be a synopsis of the full weight-of-evidence narrative. See Full IRIS Summary.

Quantitative Estimate of Carcinogenic Risk from Oral Exposure
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Not Assessed under the IRIS Program.

Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure
---
Not Assessed under the IRIS Program.

U.S. Environmental Protection Agency. 2002. Ecotox database: Cupric oxide. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN.

Scientific name, Common name	Endpoint	Effect	Trend --- Effect %	Media Type	Duration --- Exp Typ	Conc (ug/L)	Signif --- Level	Response Site --- BCF	Ref #
Daphnia magna Water flea	EC50	ITX	---	FW	48 H --- S	T 11 - 39		---	10917
Oncorhynchus mykiss Rainbow trout, donaldson trout	LC50	MOR	---	FW	96 H --- S	F 25.4, 21.8 - 29.5 ppm		---	344

Diesel Fuel, CAS #68334-30-5 (Diesel fuel no. 2)

Data Point	Data Summary	Reference
Water solubility	0.00076 mg/L.	TPHCWG 1997, 1998
K_{oc}	log K_{oc} is 6.7 (K_{oc} = 5,011,872)	TPHCWG 1997, 1998
Soil half-life	40% biodegradation in 28 days = $t_{1/2}$ of 21 days	Chevron 2001
BCF	Components of gas oil have measured or calculated log K_{ow} values in the range 3.9 to greater than 6, indicating a high potential to bioaccumulate. However there is little measured data on gas oils or their components and there are major technical difficulties in measuring bioconcentration (BCF) values with complex mixtures.	CONCAWE 1996
Ingestion toxicity	Oral LD_{50} in rats = 7,400 mg/kg Doses of 125+ mg/kg for five days increased the frequency of chromosomal aberrations in the bone marrow of Sprague-Dawley rats	API 1980a, as cited in CONCAWE 1996 WHO 1996
Carcinogenicity	Not classifiable as to carcinogenicity in humans	IARC 1989
Avian toxicity	Mallard LD_{50} = 20 mg/kg	NPS 1997
Fish toxicity	96-hour LC_{50} in rainbow trout is 21 to 210 mg/L 24-hour LC_{50}s were 1.40 to 1.97 for pink salmon, 26.7 to >55.6 for coho salmon, and >23.1 to 168.4 for rainbow trout.	Chevron 2001 WHO 1996
Aq. invert. tox	48-hour EC_{50} in *Daphnia magna* is 20 to 210 mg/L	Chevron 2001
Aq. amph. tox	96-hour LC_{50} for larvae of wood frog *Rana sylvatica* is 4.2 mg/L	Hedtke and Puglisi 1982, as cited in CONCAWE 1996

Chevron Products Co. 2001. Material safety data sheet 6894: Chevron LS diesel 2. San Ramon, CA.

CONCAWE. 1996. Gas oils (diesel fuels/heating oils). Product dossier no. 95/107. Brussels, Belgium.

IARC. See International Agency for Research on Cancer.

International Agency for Research on Cancer. 1989. Diesel fuels. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 45:219. http://193.51.164.11/htdocs/monographs/vol45/45-05.htm

NPS. See U.S. National Park Service.

Total Petroleum Hydrocarbon Criteria Working Group. 1997. Volume III: Selection of representative TPH fractions based on fate and transport considerations Amherst Scientific Publishers. Amherst, MA. http://www.aehs.com/publications/catalog/contents/Volume3.pdf

Total Petroleum Hydrocarbon Criteria Working Group. 1998. Volume I: Analysis of petroleum hydrocarbons in environmental media. Amherst Scientific Publishers. Amherst, MA. http://www.aehs.com/publications/catalog/contents/Volume1.pdf

TPHCWG. See Total Petroleum Hydrocarbon Criteria Working Group.

U.S. National Park Service. 1997. Environmental contaminants encyclopedia: Diesel oil entry. Water Resources Division, Water Operations Branch. Fort Collins, CO. http://www1.nature.nps.gov/toxic/search/

WHO. See World Health Organization.

World Health Organization. 1996. Environmental health criteria 171: Diesel fuel and exhaust emissions. Geneva. http://www.inchem.org/documents/ehc/ehc/ehc171.htm

Chevron Products Co. 2001. Material safety data sheet 6894: Chevron LS diesel 2. San Ramon, CA.

ECOTOXICITY:
A series of studies on the acute toxicity of 4 diesel fuel samples were conducted by one laboratory using water accommodated fractions. The range of effective (EC50) or lethal concentrations (LC50) expressed as loading rates were: The 96-hour LC50 for rainbow trout (Salmo gairdneri) is 21-210 mg/l. The 48-hour EC50 for daphnia (Daphnia magna) is 20-210 mg/l. The 72-hour EC50 in alga (Raphidocellus subcapitata) is 2.6-25 mg/l.
ENVIRONMENTAL FATE:
On release to the environment the lighter components of diesel fuel will generally evaporate but depending on local environmental conditions (temperature, wind, mixing or wave action, soil type, etc.) the remainder may become dispersed in the water column or absorbed to soil or sediment. Diesel fuel would not be expected to be "readily biodegradable". In a modified Strum test (OECD method 301B) approximately 40% biodegradation was recorded over 28 days. However, it has been shown that most hydrocarbon components of diesel fuel are degraded in soil in the presence of oxygen. Under anaerobic conditions, such as in anoxic sediments, rates of biodegradation are negligible.

CONCAWE. 1996. Gas oils (diesel fuels/heating oils). Product dossier no. 95/107. Brussels, Belgium.

The dossier summarizes the physical and chemical properties and toxicological, health, safety and environmental information available on gas oils, these include diesel fuels and heating oils.

International Agency for Research on Cancer. 1989. Diesel fuels. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 45:219.

Overall evaluation:
Marine diesel fuel is possibly carcinogenic to humans (Group 2B).
Distillate (light) diesel fuels are not classifiable as to their carcinogenicity to humans (Group 3).

Total Petroleum Hydrocarbon Criteria Working Group. 1998. Volume I: Analysis of petroleum hydrocarbons in environmental media. Amherst Scientific Publishers. Amherst, MA.

DIESEL FUEL

Transportation diesels are manufactured primarily from distilled fractions of crude oil with some blending with cracked gas oils. The major components of diesels are similar to those present in the crude oil, but include a higher fraction of aromatics (up to 30 to 40%). Diesel fuel is essentially the same as furnace oil, but the proportion of cracked gas oil is usually less than in furnace oil. Although cracking processes also produce small alkenes as well as aromatics, the small alkenes are not in the diesel carbon range and end up in the gasoline pool. The typical carbon range for diesel #1 grades is C8 to C17 range, with the majority in the C10 to C14 range (similar to Jet A and kerosene). The typical carbon range for diesel # 2 fuels is C8 to C26 , with the majority in the C10 to C20 range (similar to fuel oil No. 2). In all cases, the majority of the fuels is 60-90% normal, branched, and cyclic alkanes.

Total Petroleum Hydrocarbon Criteria Working Group. 1997. Volume III: Selection of representative TPH fractions based on fate and transport considerations Amherst Scientific Publishers. Amherst, MA.

> For the EC >12 to 16 aliphatic fraction of petroleum hydrocarbons, the representative water solubility is 0.00076 mg/L, and the log K_{oc} is 6.7 (K_{oc} = 5,011,872).

World Health Organization. 1996. Environmental health criteria 171: Diesel fuel and exhaust emissions. Geneva.

Draws on findings from over 600 studies to evaluate the risks to human health and the environment posed by exposure to diesel fuel and diesel exhaust emissions. The two categories of exposure are evaluated in separate parts.

The evaluation of diesel fuel opens with a discussion of the complexity of these mixtures and the many variables that affect their quality and composition. An evaluation of toxicity studies in laboratory animals and in vitro test systems concludes that diesel fuel has low acute toxicity when administered via oral, dermal, and inhalation routes. Findings on embryotoxicity, teratogenicity, mutagenicity, and genotoxicity were judged to be either negative or equivocal. In view of inadequacies in the few studies of carcinogenic risks, the report concludes that the main effect of exposure on human health is dermatitis following skin contact.

The second and largest part evaluates diesel exhaust emissions. A review of the abundant data demonstrating adverse effects on the environment concludes that the major components of diesel exhaust contribute to acid deposition, tropospheric ozone formation, and global warming. The most extensive sections discuss the epidemiological studies in humans and studies in experimental animals considered useful for the assessment of risks to human health. Although a number of epidemiological studies have indicated an increased risk of lung cancer in bus and railroad workers, all studies suffered from weaknesses. The report concludes that diesel exhaust is probably carcinogenic to humans, and that inhalation of diesel exhaust contributes to both neoplastic and non-neoplastic diseases, including asthma. The report further concludes that the particulate phase has the greatest effect on human health.

Gasoline, CAS # 8006-61-9

Data Point	Data Summary	Reference
Water solubility	Insoluble	ATSDR 1995
K_{oc}	Log K_{oc} = 1.81 to 4.56 (K_{oc} = 65 to 36,300)	ATSDR 1995
Soil half-life		
BCF		
Ingestion toxicity	No NOAEL identified. Lowest LOAELs for endpoints relevant to human toxicity (body weight, gastrointestinal effects) were 2,000 mg/kg in 28-day studies in rats. The oral LD_{50} in rats was 14,063 mg/kg	ATSDR 1995
Carcinogenicity	No studies were located regarding cancer in humans or animals after oral exposure to gasoline. Gasoline is possibly carcinogenic to humans (Group 2B) by inhalation exposure.	ATSDR 1995 IARC 1989
Fish toxicity	96-hour LC_{50} in rainbow trout is 2.7 mg/l (based on values for BTEX).	Chevron 2001
Aq. invert. tox	48-hour LC_{50} in Daphnia magna is 3.0 mg/L (based on values for BTEX).	Chevron 2001
Aq. amph. tox		

Agency for Toxic Substances and Disease Registry. 1995. Toxicological profile for automotive gasoline. Atlanta, GA. http://www.atsdr.cdc.gov/toxprofiles/tp72.html

ATSDR. See Agency for Toxic Substances and Disease Registry.

Chevron Products Company. 2001. MSDS 2655: Regular unleaded gasoline. San Ramon, CA.

IARC. See International Agency for Research on Cancer.

International Agency for Research on Cancer. 1989. Gasoline. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 45:159. http://193.51.164.11/htdocs/monographs/vol45/45-03.htm

Agency for Toxic Substances and Disease Registry. 1995. Toxicological profile for gasoline. Atlanta, GA.

Report summarized by ATSDR in the form of ToxFAQs document; relevant sections follow:

ToxFAQs™ for Automotive Gasoline, CAS# 8006-61-9, September 1996

"SUMMARY: Exposure to automotive gasoline most likely occurs from breathing its vapor at a service station while filling a car's fuel tank. At high levels, automotive gasoline is irritating to the lungs when breathed in and irritating to the lining of the stomach when swallowed. Exposure to high levels may also cause harmful effects to the nervous system.

Typically, gasoline contains more than 150 chemicals, including small amounts of benzene, toluene, xylene, and sometimes lead. How the gasoline is made determines which chemicals are present in the gasoline mixture and how much of each is present. The actual composition varies with the source of the crude petroleum, the manufacturer, and the time of year.

What happens to automotive gasoline when it enters the environment? Small amounts of the chemicals present in gasoline evaporate into the air when you fill the gas tank in your car or when gasoline is accidentally spilled onto surfaces and soils or into surface waters. Other chemicals in gasoline dissolve in water after spills to surface waters or underground storage tank leaks into the groundwater. In surface releases, most chemicals in gasoline will probably evaporate; others may dissolve and be carried away by water; a few will probably stick to soil. The chemicals that evaporate are broken down by sunlight and other chemicals in the air. The chemicals that dissolve in water also break down quickly by natural processes.

Many of the harmful effects seen after exposure to gasoline are due to the individual chemicals in the gasoline mixture, such as benzene and lead. Inhaling or swallowing large amounts of gasoline can cause death. Inhaling high concentrations of gasoline is irritating to the lungs when breathed in and irritating to the lining of the stomach when swallowed. Gasoline is also a skin irritant. Breathing in high levels of gasoline for short periods or swallowing large amounts of gasoline may also cause harmful effects on the nervous system. Serious nervous system effects include coma and the inability to breathe, while less serious effects include dizziness and headaches. There is not enough information available to determine if gasoline causes birth defects or affects reproduction. The Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have not classified automotive gasoline for carcinogenicity. Automotive gasoline is currently undergoing review by the EPA for cancer classification. Some laboratory animals that breathed high concentrations of unleaded gasoline vapors continuously for 2 years developed liver and kidney tumors. However, there is no evidence that exposure to gasoline causes cancer in humans.

Chevron Products Company. 2001. MSDS 2655: Regular unleaded gasoline. San Ramon, CA.

ECOTOXICITY:
Gasoline studies have been conducted in the laboratory under a variety of test conditions with a range of fish and invertebrate species. An even more extensive database is available on the aquatic toxicity of individual aromatic constituents. The majority of published studies do not identify the type of gasoline evaluated, or even provide distinguishing characteristics such as aromatic content or presence of lead alkyls. As a result, comparison of results among studies using open and closed vessels, different ages and species of test animals and different gasoline types, is difficult.

The bulk of the available literature on gasoline relates to the environmental impact of monoaromatic (BTEX) and diaromatic (naphthalene, methylnaphthalenes) constituents. In general, non-oxygenated gasoline exhibits some short-term toxicity to freshwater and marine organisms, especially under closed vessel or flow-through exposure conditions in the laboratory. The components which are the most prominent in the water soluble fraction and cause aquatic toxicity, are also highly volatile and can be readily biodegraded by microorganisms.

The 96-hour LC50 in rainbow trout (Oncorhynchus mykiss) is 2.7 mg/l (BTEX). The 48-hour LC50 in daphnia (Daphnia magna) is 3.0 mg/l (BTEX). The 96-hour LC50 in sheepshead minnow (Cyprinodon variegatus) is 8.3 mg/l (BTEX). The 96-hour LC50 in mysid shrimp (Mysidopsis bahia) is 1.8 mg/l (BTEX).

International Agency for Research on Cancer. 1989. Gasoline. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 45:159.

In reference to inhalation exposure to gasoline, IARC concluded the following:

- There is inadequate evidence for the carcinogenicity in humans of gasoline.
- There is limited evidence for the carcinogenicity in experimental animals of unleaded automotive gasoline.
- Gasoline is possibly carcinogenic to humans (Group 2B).

Iron Oxide, CAS # 1309-37-1

Data Point	Data Summary	Reference
Water solubility	Insoluble.	HSDB 2002
K_{oc}	No data.	
Soil half-life	Stable.	
BCF	No data.	
Ingestion toxicity	Iron oxide is regulated by the FDA for use as a food coloring and in food packaging; it is generally recognized as safe. Severe toxicity may result in children following ingestion of more than 0.5 g of iron. In adults, chronic excessive ingestion may lead to toxicity, manifested by hemosiderosis, disturbances in liver function, diabetes mellitus, and possible endocrine disturbances and cardiovascular effects. EPA has established a secondary drinking water regulation of 0.3 mg/L for iron, based on aesthetic endpoints. Intraperitoneal LD_{50} is 5,400 mg/kg in mice.	21 CFR 73.200, 186.1300, and 186.1374 (Amdur et al. 1991) EPA 1992 DHHS 1987
Carcinogenicity	Not classifiable as to its carcinogenicity in humans.	IARC 1987
Fish toxicity Aq. invert. tox Aq. amph. tox	EPA set an ambient water quality criteria level of 1 mg/L for protection of aquatic life from iron, equivalent to 2.9 mg Fe_2O_3/L.	EPA 1999

Amdur, M.O., J. Doull, and C.D. Klaassen (eds.). 1991 *Casarett and Doull's Toxicology: The Basic Science of Poisons.* 4th edition. Pergamon Press, Inc. Elmsford, NY.

DHHS. See U.S. Department of Health and Human Services.

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

IARC. See International Agency for Research on Cancer.

International Agency for Research on Cancer. 1987. Haematite and ferric oxide. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Supplement 7:216. http://193.51.164.11/htdocs/monographs/suppl7/haematite.html

U.S. Department of Health and Human Services (DHHS). 1987. Registry of Toxic Effects of Chemical Substances (RTECS). DHHS NIOSH Publication No. 87-114. U.S. Government Printing Office. Washington, DC.

U.S. Environmental Protection Agency. 1992. Secondary drinking water regulations: Guidance for nuisance chemicals. EPA 810/K-92-001. Office of Water. Washington, DC.

U.S. Environmental Protection Agency. 1999. National recommended water quality criteria--Correction. EPA 822-A-99-01. Office of Water. Washington, DC.

Amdur, M.O., J. Doull, and C.D. Klaassen (eds.). 1991 *Casarett and Doull's Toxicology: The Basic Science of Poisons.* 4th edition. Pergamon Press, Inc. Elmsford, NY.

> Acute iron toxicity is nearly always due to accidental ingestion of iron-containing medicines, and most often occurs in children. ... Severe toxicity occurs after ingestion of more than 0.5 g of iron or 2.5 g of ferrous sulfate. ... Chronic toxicity or iron overload in adults is a more common problem. ... The pathologic consequences of iron overload are similar regardless of basic cause. The body iron content is increased to between 20 and 40 g. Most of the extra iron is hemosiderin. Greatest concentrations are in parenchymal cells of liver and pancreas, as well as endocrine organs and heart. ... Further clinical effects may include disturbances in liver function, diabetes mellitus, and even endocrine disturbances and cardiovascular effects.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD.

HSDB is a toxicology data file on the National Library of Medicine's (NLM) Toxicology Data Network (TOXNET®). It focuses on the toxicology of potentially hazardous chemicals. It is enhanced with information on human exposure, industrial hygiene, emergency handling procedures, environmental fate, regulatory requirements, and related areas. All data are referenced and derived from a core set of books, government documents, technical reports and selected primary journal literature. HSDB is peer-reviewed by the Scientific Review Panel (SRP), a committee of experts in the major subject areas within the data bank's scope. HSDB is organized into individual chemical records, and contains over 4500 such records.

The following are the human health and environmental fate summaries from HSDB:

Human Health Effects:

Evidence for Carcinogenicity:

Classification of carcinogenicity: 1) evidence in humans: inadequate; 2) evidence suggesting lack of carcinogenicity in animals. Overall summary evaluation of carcinogenic risk to humans is Group 3: The agent is not classifiable as to its carcinogenicity to humans. /From table/
[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. S7 216 (1987)]**PEER REVIEWED**

A4; Not classifiable as a human carcinogen. /Iron oxide dust and fume (Fe2O3), as Fe/
[American Conference of Governmental Industrial Hygienists. TLVs & BEIs: Threshold limit Values for Chemical Substances and Physical Agents andBiological Exposure Indices for 2002. Cincinnati, OH. 2002. 37]**QC REVIEWED**

Human Toxicity Excerpts:

HEMATITE DUST CAUSES A BENIGN PNEUMOCONIOSIS ... .
[The Merck Index. 9th ed. Rahway, New Jersey: Merck & Co., Inc., 1976. 525]**PEER REVIEWED**

IT IS CLEAR ... THAT UNDER CONDITIONS OF HEAVY EXPOSURE TO HEMATITE DUST, PULMONARY CLEARANCE MECHANISMS MAY BE OVERWHELMED. ... UPPER LOBES & UPPER PARTS OF LOWER LOBES TEND TO BE MORE AFFECTED BY FIBROSIS THAN LOWER PARTS OF LOWER LOBES. ALSO, PERIPHERIES OF LUNGS TEND TO BE MORE AFFECTED THAN CENTRAL REGIONS.
[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. V1 32]**PEER REVIEWED**

... 1 CASE OF BRONCHIAL CARCINOMA & 1 GROSS PULMONARY TUBERCULOSIS /WERE REPORTED/ AMONG GROUP OF HEMATITE MINERS.
[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. V1 32]**PEER REVIEWED**

... EXCESSIVE INCIDENCE OF BRONCHIAL CANCER /WAS REPORTED/ AMONG IRON-ORE MINERS OF LORRAINE BASIN. ... 64 CASES OF DISEASE /WERE REPORTED/

AMONG 10000 ... MINERS ... COMPARED WITH 28 CASES AMONG 10000 WORKERS FROM IRON WORKS IN SAME DISTRICT. CO-EXISTENCE OF LUNG CANCER & SILICOSIS ... NOTED IN 10 EX-MINERS ON PENSION.
[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. V1 33]**PEER REVIEWED**

OCCLUSIVE & OBLITERATIVE VASCULAR CHANGES IN HEMATITE LUNG ARE REALLY THOSE OF SILICOSIS. ONLY FEATURE PECULIAR TO HEMATITE LUNG IS INTENSE ACCUM OF IRON-DUST IN & AROUND PULMONARY BLOOD VESSELS.
[HEATH D ET AL; BR J DIS CHEST 72 (2): 88-94 (1978)]**PEER REVIEWED**

LUNG FUNCTION TESTS IN 14 WORKERS EXPOSED ON AVG OF 10 YR TO PURE IRON OXIDE DUST SEEMS TO SUPPORT OPINION THAT PURE IRON OXIDE IS NOT FIBROGENIC IN LUNG.
[TECULESCU D, ALBU A; INT ARCH ARBEITSMED 31 (2): 163-70 (1973)]**PEER REVIEWED**

Eight of 25 welders exposed chiefly to iron oxide for an average of 18.7 (range 3 to 32) years had reticulonodular shadows on chest x-rays consistent with siderosis but no reduction in pulmonary function; exposure levels ranged from 0.65 to 47 mg/cu m. In another study, 16 welders with an average exposure of 17.1 (range 7 to 30) years also had x-rays suggesting siderosis and spirograms which were normal; however, the static and functional compliance of the lungs was reduced; some of the welders were smokers. The welders with the lowest compliance complained of dyspnea.
[Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981.]**PEER REVIEWED**

... Some electric arc welders exposed mainly to iron oxide fume showed generalized discrete densities in their chest X-ray films. None of these welders, however, showed any demonstrable clinical disability.
[American Conference of Governmental Industrial Hygienists, Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. Volumes I,II, III. Cincinnati, OH: ACGIH, 1991. 803]**PEER REVIEWED**

... Workers exposed to iron oxide fume and silica amy develop a "mixed dust pneumoconiosis."
[American Conference of Governmental Industrial Hygienists, Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. Volumes I,II, III. Cincinnati, OH: ACGIH, 1991. 803]**PEER REVIEWED**

... Little or no physical disability was associated with the presence of iron oxide fume and dust in the lungs ... The deposition and collection of iron oxide in the lung ... has been termed "siderosis". ... Siderosis is considered a benign condition and does not progress to fibrosis. Six to 10 years of exposure to iron oxide fume is generally required in order to produce siderosis. Little or no clinical changes are found upon physical examination of workers diagnosed with siderosis.
[American Conference of Governmental Industrial Hygienists, Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. Volumes I,II, III. Cincinnati, OH: ACGIH, 1991. 803]**PEER REVIEWED**

Studies in foundry workers exposed to iron oxide have shown an increase in lung cancer incidence among these workers.
[American Conference of Governmental Industrial Hygienists, Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. Volumes I,II, III. Cincinnati, OH: ACGIH, 1991. 804]**PEER REVIEWED**

... Three cases of severe pulmonary changes related to iron oxide exposure from welding fumes /was reported/. The three men in this study suffered from cough and shortness of breath, X-ray examination revealed diffuse fibrosis.
[Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986. 286]**PEER REVIEWED**

... The lung function of 16 welders exposed to iron oxide fumes /was compared/ with 13 non-exposed men of similar age, height and smoking habits. Static and functional lung compliance of exposed men was found to be significantly different from that of controls. Silica was stated to be absent in ... /the/ study.
[Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986. 287]**PEER REVIEWED**

HIGHER CONCN OF SILICA & IRON ... FOUND IN LUNGS OF HEMATITE MINERS WITH FIBROSIS (ASSOC OR NOT WITH PULMONARY TUBERCULOSIS) OR ... BRONCHIAL CARCINOMA THAN IN ... THOSE WITH NO SUCH PATHOLOGY ... LEVELS IN THOSE WITH CARCINOMA WERE NOT HIGHER THAN ... IN THOSE WITH FIBBROSIS (ACCOMPANIED OR NOT BY TUBERCULOSIS) BUT NO CARCINOMA ... .
[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. V1 32]**PEER REVIEWED**

ON BASIS OF EPIDEMIOLOGICAL EVIDENCE, EXPOSURE TO HEMATITE DUST MAY BE REGARDED AS INCR RISK OF LUNG CANCER DEVELOPMENT IN MAN. RISK IS MANIFEST IN UNDERGROUND WORKERS BUT NOT SURFACE WORKERS ... NOT KNOWN WHETHER EXCESS RISK IS DUE TO RADIOACTIVITY IN AIR OF MINES, INHALATION OF FERRIC OXIDE OR SILICA, OR COMBINATION ... .
[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. V1 36]**PEER REVIEWED**

MOST COMPREHENSIVE EPIDEMIOLOGICAL STUDY...DEATH CERTIFICATES /WERE STUDIED/ OF 5811 MALE RESIDENTS OF HEMATITE MINING REGION OF CUMBERLAND, ENG WHO DIED BETWEEN 1948 & 1967. ... 36 LUNG CANCER DEATHS AMONG UNDERGROUND HEMATITE WORKERS AS COMPARED WITH CA 21 EXPECTED ON BASIS OF EITHER LOCAL NON-MINER DEATHS OR NATIONAL AVG.
[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. V1 33]**PEER REVIEWED**

/IN STUDY OF 5811 HEMATITE WORKERS' DEATH CERTIFICATES IN CUMBERLAND, ENG/ NO EXCESS MORTALITY FROM LUNG CANCER WAS FOUND AMONG SURFACE IRON-ORE MINERS, & FOR IRON MINERS IN GENERAL MORTALITY FROM CANCERS OF SITES OTHER THAN LUNG WAS CLOSE TO NATIONAL AVG.
[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. V1 33]**PEER REVIEWED**

... STUDY OF IRON-ORE MINERS IN LORRAINE BASIN OF FRANCE, COMPARED INCIDENCE OF BRONCHOGENIC CARCINOMA IN 1095 IRON-ORE MINERS & 940 NON-MINERS (ALL MALES ...): 3.3% INCIDENCE IN FORMER IS SIGNIFICANTLY HIGHER (P= 0.01) THAN IN LATTER (1.5%). ... USE OF TOBACCO IS COMMON AMONG THEIR IRON WORKERS.

[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. V1 33]**PEER REVIEWED**

10 MG/CU M FOR IRON OXIDE FUME IS SUGGESTED TO PREVENT DEVELOPMENT OF X-RAY CHANGES IN LUNGS ON LONG-TERM EXPOSURE. ... MORE INFORMATION IS ALSO NEEDED ON RELATIONSHIP OF IRON DEPOSITS IN LUNGS TO CONCOMITANT EXPOSURE TO OTHER INDUSTRIAL DUSTS.
[American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values for Substances in Workroom Air. Third Edition, 1971. Cincinnati, Ohio: AmericanConference of Governmental Industrial Hygienists, 1971. (Plus supplements to 1979) 136]**PEER REVIEWED**

Medical Surveillance:

/Protect/ from exposure those individuals with pulmonary diseases.
[ITII. Toxic and Hazardous Industrial Chemicals Safety Manual. Tokyo, Japan: The International Technical Information Institute, 1988. 284]**PEER REVIEWED**

The following medical procedures should be made available to each employee who is exposed to iron oxide fume at potentially hazardous levels: ... A complete history and physical examination ... Examination of the respiratory system should be stressed; 14" X 17" chest roentgenogram; ... FVC and FEV (1 sec). ... The aforementioned medical examinations should be repeated on an annual basis, except that an x-ray is considered necessary only when indicated by the results of pulmonary function testing.
[Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981.]**PEER REVIEWED**

Populations at Special Risk:

... Individuals with pulmonary diseases.
[ITII. Toxic and Hazardous Industrial Chemicals Safety Manual. Tokyo, Japan: The International Technical Information Institute, 1988. 284]**PEER REVIEWED**

Probable Routes of Human Exposure:

... FOLLOWING OCCUPATIONS ... /ENTAIL/ RISK OF INHALATION OF DUST & FUMES OF IRON & ITS VARIOUS ALLOYS & CMPD: IRON-ORE MINERS; ARC WELDERS; GRINDERS: POLISHERS; SILVER FINISHERS; METAL WORKERS. ... /ALSO/ BOILER MAKERS ... .
[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. V1 30]**PEER REVIEWED**

Environmental Fate & Exposure:

Natural Pollution Sources:

Alpha-form occurs in nature as the mineral hematite; gamma-form occurs as the mineral maghemite
[Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989. 632]**PEER REVIEWED**

International Agency for Research on Cancer. 1987. Haematite and ferric oxide. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Supplement 7:216.

Overall evaluation
Ferric oxide is *not classifiable as to its carcinogenicity to humans (Group 3)*.
Haematite is *not classifiable as to its carcinogenicity to humans (Group 3)*.
Underground haematite mining with exposure to radon is *carcinogenic to humans (Group 1)*.

U.S. Environmental Protection Agency. 1992. Secondary drinking water regulations: Guidance for nuisance chemicals. EPA 810/K-92-001. Office of Water. Washington, DC.

The U.S. Environmental Protection Agency (EPA) has established National Primary Drinking Water Regulations that set mandatory water quality standards for drinking water contaminants. These are enforceable standards called "maximum contaminant levels" or "MCLs", which are established to protect the public against consumption of drinking water contaminants that present a risk to human health. An MCL is the maximum allowable amount of a contaminant in drinking water which is delivered to the consumer .

In addition, EPA has established National Secondary Drinking Water Regulations that set non-mandatory water quality standards for 15 contaminants. EPA does not enforce these "secondary maximum contaminant levels" or "SMCLs." They are established only as guidelines to assist public water systems in managing their drinking water for aesthetic considerations, such as taste, color and odor. These contaminants are not considered to present a risk to human health at the SMCL.

Table I. Secondary Maximum Contaminant Levels

Contaminant	**Secondary MCL**	**Noticeable Effects above the Secondary MCL**
Aluminum	0.05 to 0.2 mg/L*	colored water
Chloride	250 mg/L	salty taste
Color	15 color units	visible tint
Copper	1.0 mg/L	metallic taste; blue-green staining
Corrosivity	Non-corrosive	metallic taste; corroded pipes/ fixtures staining
Fluoride	2.0 mg/L	tooth discoloration
Foaming agents	0.5 mg/L	frothy, cloudy; bitter taste; odor
Iron	0.3 mg/L	rusty color; sediment; metallic taste; reddish or orange staining
Manganese	0.05 mg/L	black to brown color; black staining; bitter metallic taste
Odor	3 TON (threshold odor number)	"rotten-egg", musty or chemical smell
pH	6.5 - 8.5	*low pH:* bitter metallic taste; corrosion *high pH:* slippery feel; soda taste; deposits
Silver	0.1 mg/L	skin discoloration; graying of the white part of the eye
Sulfate	250 mg/L	salty taste
Total Dissolved Solids (TDS)	500 mg/L	hardness; deposits; colored water; staining; salty taste
Zinc	5 mg/L	metallic taste
** mg/L is milligrams of substance per liter of water*		

U.S. Environmental Protection Agency. 1999. National recommended water quality criteria--Correction. EPA 822-A-99-01. Office of Water. Washington, DC.

SUMMARY: EPA is publishing a compilation of its national recommended water quality criteria for 157 pollutants, developed pursuant to section 304(a) of the Clean Water Act (CWA or the Act). These recommended criteria provide guidance for States and Tribes in adopting water quality standards under section 303(c) of the CWA. Such standards are used in implementing a number of environmental programs, including setting discharge limits in National Pollutant Discharge Elimination System (NPDES) permits. These water quality criteria are not regulations, and do not impose legally binding requirements on EPA, States, Tribes or the public.

Lead, CAS #7439-92-1

Data Point	Data Summary	Reference
Water solubility	Insoluble.	ATSDR 1999
K_{oc}	Most lead is retained strongly in soil, and very little is transported into surface water or groundwater. Lead is strongly sorbed to organic matter in soil, and although not subject to leaching, it may enter surface waters as a result of erosion of lead-containing soil particulates.	ATSDR 1999
Soil half-life	Stable.	
BCF	Median BCF = 42 in fish.	Eisler 1988
Ingestion toxicity	EPA's reference dose workgroup concluded it was inappropriate to develop a reference dose, or an acceptable daily intake, for lead because some of lead's adverse effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. A lowest lethal dose of 1,400 mg/kg was estimated for lead oxide in dogs, equivalent to 1,307 mg/kg lead.	EPA 1993 ATSDR 1999
Carcinogenicity	Lead is a probable human carcinogen, but a quantitative estimate of risk is not appropriate given current data.	EPA 1993
Avian toxicity	5-day dietary LC_{50} in Japanese quail >5,000 ppm in food, equivalent to approximately 875 mg/kg.	HSDB 2002
Fish toxicity	96-hour LC_{50} in rainbow trout is 1.17 mg/L.	EPA 2002
Aq. invert. tox	48-hour LC_{50} in *Daphnia magna* is 4.4 mg/L.	EPA 2002
Aq. amph. tox	The 30-day LC_{50} value for *Rana pipiens* was 105 mg/L.	Eisler 1988

Agency for Toxic Substances and Disease Registry. 1999. Toxicological profile for lead Atlanta, GA. http://www.atsdr.cdc.gov/toxprofiles/tp13.html

ATSDR. See Agency for Toxic Substances and Disease Registry.

Eisler, R. 1988. Lead hazards to fish, wildlife and invertebrates: A synoptic review. Patuxent Wildlife Research Center, U.S. Fish and Wildlife Service. Laurel, MD. http://www.pwrc.usgs.gov/new/chrback.htm

EPA. See U.S. Environmental Protection Agency.

U.S. Environmental Protection Agency. 1993. Integrated risk information system. Office of Research and Development. Cincinnati, OH. http://www.epa.gov/iris/subst/0277.htm

U.S. Environmental Protection Agency. 2002. Ecotox database: Lead. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN. http://www.epa.gov/ecotox/

Agency for Toxic Substances and Disease Registry. 1999. Toxicological profile for lead. Atlanta, GA.

Report summarized by ATSDR in the form of ToxFAQs document; relevant sections follow:

HIGHLIGHTS: Exposure to lead can happen from breathing workplace air or dust, eating contaminated foods, or drinking contaminated water. Children can be exposed from eating lead-based paint chips or playing in contaminated soil. Lead can damage the nervous system, kidneys, and reproductive system.

What happens to lead when it enters the environment? Lead itself does not break down, but lead compounds are changed by sunlight, air, and water. When lead is released to the air, it may travel long distances before settling to the ground. Once lead falls onto soil, it usually sticks to soil particles. Movement of lead from soil into groundwater will depend on the type of lead compound and the characteristics of the soil. Much of the lead in inner-city soils comes from old houses painted with lead-based paint.

How can lead affect my health?
Lead can affect almost every organ and system in your body. The most sensitive is the central nervous system, particularly in children. Lead also damages kidneys and the reproductive system. The effects are the same whether it is breathed or swallowed. At high levels, lead may decrease reaction time, cause weakness in fingers, wrists, or ankles, and possibly affect the memory. Lead may cause anemia, a disorder of the blood. It can also damage the male reproductive system. The connection between these effects and exposure to low levels of lead is uncertain. The Department of Health and Human Services has determined that lead acetate and lead phosphate may reasonably be anticipated to be carcinogens based on studies in animals.
There is inadequate evidence to clearly determine lead's carcinogenicity in people. Small children can be exposed by eating lead-based paint chips, chewing on objects painted with lead-based paint, or swallowing house dust or soil that contains lead. Children are more vulnerable to lead poisoning than adults. A child who swallows large amounts of lead may develop blood anemia, severe stomachache, muscle weakness, and brain damage. A large amount of lead might get into a child's body if the child ate small pieces of old paint that contained large amounts of lead. If a child swallows smaller amounts of lead, much less severe effects on blood and brain function may occur. Even at much lower levels of exposure, lead can affect a child's mental and physical growth. Exposure to lead is more dangerous for young and unborn children. Unborn children can be exposed to lead through their mothers. Harmful effects include premature births, smaller babies, decreased mental ability in the infant, learning difficulties, and reduced growth in young children. These effects are more common if the mother or baby was exposed to high levels of lead.

Eisler, R. 1988. Lead hazards to fish, wildlife and invertebrates: A synoptic review. Patuxent Wildlife Research Center, U.S. Fish and Wildlife Service. Laurel, MD.

SUMMARY

Lead (Pb) and its compounds have been known to man for about 7,000 years, and Pb poisoning has been recognized for at least 2,500 years. All credible evidence indicates that Pb is neither essential nor beneficial to living organisms, and that all measured effects are adverse--including those on survival, growth, reproduction, development, behavior, learning, and metabolism.

Various living resources are at increased risk from Pb: migratory waterfowl that frequent hunted areas and ingest shot; avian predators that eat game wounded by hunters; domestic livestock near smelters, refineries, and Pb battery recycling plants; captive zoo animals and domestic livestock held in enclosures coated with Pb-based paints; wildlife that forage extensively near heavily traveled roads; aquatic life in proximity to mining activities, areas where Pb arsenate pesticides are used, metal finishing industries, organolead industries, and areas of Pb aerosol fallout; and crops and invertebrates growing or living in Pb-contaminated soils.

Adverse effects on aquatic biota reported at waterborne Pb concentrations of 1.0 to 5.1 ug/l included reduced survival, impaired reproduction, reduced growth, and high bioconcentration from the medium. Among sensitive species of birds, survival was reduced at doses of 50 to 75 mg Pb2+/kg body weight (BW) or 28 mg organolead/kg BW, reproduction was impaired at dietary levels of 50 mg Pb /kg, and signs of poisoning were evident at doses as low as 2.8 mg organolead/kg BW. In general, forms of Pb other than shot (or ingestible Pb objects), or routes of administration other than ingestion, are unlikely to cause clinical signs of Pb poisoning in birds. Data for toxic and sublethal effects of Pb on mammalian wildlife are missing. For sensitive species of domestic and laboratory animals, survival was reduced at acute oral Pb doses of 5 mg/kg BW (rat), at chronic oral doses of 5 mg/kg BW (dog), and at dietary levels of 1.7 mg/kg BW (horse). Sublethal effects were documented in monkeys exposed to doses as low as 0.1 mg Pb/kg BW daily (impaired learning at 2 years postadministration) or fed diets containing 0.5 mg Pb/kg (abnormal social behavior). Signs of Pb exposure were recorded in rabbits given 0.005 mg Pb/kg BW and in mice given 0.05 mg Pb/kg BW. Tissue Pb levels were elevated in mice given doses of 0.03 mg Pb/kg BW, and in sheep given 0.05 mg Pb/kg BW. In general, organolead compounds were more toxic than inorganic Pb compounds, food chain biomagnification of Pb vas negligible, and younger organisms were most susceptible. More research seems merited on organolead toxicokinetics (including effects on behavior and learning), and on mammalian wildlife sensitivity to Pb and its compounds.

Recent legislation limiting the content of Pb in paints, reducing the Pb content in gasoline, and eliminating the use of Pb shot nationwide (Pb shot phaseout program/schedule starting in 1986, and fully implemented by 1991) in waterfowl hunting areas will substantially reduce environmental burdens of Pb and may directly benefit sensitive fishery and wildlife resources. Continued nationwide monitoring of Pb in living resources is necessary in order to correlate reduced emission sources with reduced tissue Pb concentrations.

U.S. Environmental Protection Agency. 1993. Integrated risk information system. Office of Research and Development. Cincinnati, OH.

STATUS OF DATA FOR Lead and compounds (inorganic)
File First On-Line 03/01/1988

Category (section)	**Status**	**Last Revised**
Oral RfD Assessment (I.A.)	message	02/01/1991
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	11/01/1993

_I.A. Reference Dose for Chronic Oral Exposure (RfD)
Substance Name -- Lead and compounds (inorganic)
CASRN -- 7439-92-1

A great deal of information on the health effects of lead has been obtained through decades of medical observation and scientific research. This information has been assessed in the development of air and water quality criteria by the Agency's Office of Health and Environmental Assessment (OHEA) in support of regulatory decision-making by the Office of Air Quality Planning and Standards (OAQPS) and by the Office of Drinking Water (ODW). By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. The Agency's RfD Work Group discussed inorganic lead (and lead compounds) at two meetings (07/08/1985 and 07/22/1985) and considered it inappropriate to develop an RfD for inorganic lead. For additional information, interested parties are referred to the 1986 Air Quality Criteria for Lead (EPA-600/8-83/028a-dF) and its 1990 Supplement (EPA/600/8-89/049F).

II.A. Evidence for Human Carcinogenicity
__II.A.1. Weight-of-Evidence Characterization
Classification -- B2; probable human carcinogen
Basis -- Sufficient animal evidence. Ten rat bioassays and one mouse assay have shown statistically significant increases in renal tumors with dietary and subcutaneous exposure to several soluble lead salts. Animal assays provide reproducible results in several laboratories, in multiple rat strains with some evidence of multiple tumor sites. Short term studies show that lead affects gene expression. Human evidence is inadequate.

_II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure
Not available.
Quantifying lead's cancer risk involves many uncertainties, some of which may be unique to lead. Age, health, nutritional state, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk. Thus, the Carcinogen Assessment Group recommends that a numerical estimate not be used.

U.S. Environmental Protection Agency. 2002. Ecotox database: Lead. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN.

The ECOTOXicology database is a source for locating single chemical toxicity data for aquatic life, terrestrial plants and wildlife. ECOTOX integrates three toxicology effects databases: AQUIRE (aquatic life), PHYTOTOX (terrestrial plants), and TERRETOX (terrestrial wildlife). These databases were created by the U.S. EPA, Office of Research and Development (ORD), and the National Health and Environmental Effects Research Laboratory (NHEERL), Mid-Continent Ecology Division.

Scientific name, Common name	End-point	Effect	Trend --------- Effect %	Media Type	Duration ---------- Exp Typ	Conc (ug/L)	Signif ------- Level	Response Site ------- BCF	Ref #
									Test Loc: LAB
CAS #/Chemical: 7439921, Lead									
Daphnia magna Water flea	LC50	MOR	INC --------	FW	48 H ---------- S	T 4400, 3600 - 5300		------	11181
Oncorhynchus mykiss Rainbow trout, donaldson trout	LC50	MOR	INC --------	FW	96 H ---------- F	T 1.17 mg/L		------	14367

Manganese Dioxide, CAS #1313-13-9 (MnO_2)

Data Point	Data Summary	Reference
Water solubility	Insoluble.	ATSDR 2000
K_{oc}	Sorption of manganese is complicated by redox reactions that produce compounds of different oxidation states. Under aerobic conditions, insoluble manganese 3+ and 4+ compounds predominately form.	HSDB 2002
Soil half-life	Insoluble manganese 3+ and 4+ compounds in sediments may be reduced by manganese-reducing bacteria to soluble manganese 2+ compounds.	HSDB 2002
BCF	A BCF of 100 to 600 was estimated for fish.	ATSDR 2000
Ingestion toxicity	The mean manganese intake in the United States from foodstuffs for a 2-year-old child is estimated to be about 1.5 mg/child/day. The mean manganese intake in the United States from foodstuffs for 25- to 30-year-old man and woman are estimated to be about 2.1 and 2.7 mg/person/day, respectively. ATSDR adopted the National Research Council's upper range of the estimated safe and adequate daily dietary intake of 5 mg/day as a provisional guidance value for oral exposure to manganese; this is equivalent to 0.07 mg/kg/day. EPA has set an oral reference dose of 0.14 mg/kg/day for manganese intake. An oral LD_{50} of 11,250 mg/kg was identified for manganese in rats, equivalent to 17,803 mg MnO_2/kg.	HSDB 2002 ATSDR 2000 EPA 1996 ATSDR 2000
Carcinogenicity	Not classifiable as to carcinogenicity in humans.	EPA 1996
Fish toxicity	96-hour LC_{50} for manganese in rainbow trout was 4.83 mg/L, equivalent to 7.64 mg MnO_2/L.	Reimer 1988
Aq. invert. tox	48-hour LC_{50} for manganese in *Daphnia magna* was 4.7 to 56.1 mg/L, equivalent to 7.4 to 89 mg MnO_2/L.	Reimer 1988
Aq. amph. tox	No data.	

Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese. Atlanta, GA. http://www.atsdr.cdc.gov/toxprofiles/tp151.html

ATSDR. See Agency for Toxic Substances and Disease Registry.

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

Reimer, P. 1988. Environmental effects of manganese and proposed freshwater guidelines to protect aquatic life in British Columbia. Department of Chemical and Bio-Resource Engineering. University of British Columbia.

U.S. Environmental Protection Agency. 1996. Integrated risk information system. Office of Research and Development. Cincinnati, OH. http://www.epa.gov/iris/subst/0373.htm

Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese. Atlanta, GA.

Report summarized by ATSDR in the form of ToxFAQs document; relevant sections follow:

Manganese is an essential trace element and is necessary for good health. Manganese can be found in several food items, including grains and cereals, and is found in high amounts in other foods, such as tea.

What happens to manganese when it enters the environment? Manganese can enter the air from iron, steel, and power plants, coke ovens, and from dust from mining operations. It can enter the water and soil from natural deposits, disposal of wastes, or deposits from airborne sources. Manganese exists naturally in rivers, lakes, and underground water. Plants in the water can take up some of the manganese from water and concentrate it.

How can manganese affect my health? Some individuals exposed to very high levels of manganese for long periods of time in their work developed mental and emotional disturbances and slow and clumsy body movements. This combination of symptoms is a disease called "manganism." Workers usually do not develop symptoms of manganism unless they have been exposed to manganese for many months or years. Manganism occurs because too much manganese injures a part of the brain that helps control body movements. Exposure to high levels of airborne manganese, such as in a manganese foundry or battery plant, can affect motor skills such as holding one's hand steady, performing fast hand movements, and maintaining balance. Exposure to high levels of the metal may also cause respiratory problems and sexual dysfunction. There are no human cancer data available for manganese. Exposure to high levels of manganese in food resulted in a slightly increased incidence of pancreatic tumors in male rats and thyroid tumors in male and female mice. The EPA has determined that manganese is not classifiable as to human carcinogenicity.

Daily intake of small amounts of manganese is needed for growth and good health in children. Manganese is constantly present in the mother and is available to the developing fetus during pregnancy. Manganese is also transferred from a nursing mother to her infant in breast milk at levels that are appropriate for proper development. Children, as well as adults, who lose the ability to remove excess manganese from their bodies develop nervous system problems. Because at certain ages children take in more than adults, there is concern that children may be more susceptible to the toxic effects of excess manganese. Animal studies indicate that exposure to high levels of manganese can cause birth defects in the unborn. There is no information on whether mothers exposed to excess levels of manganese can transfer the excess to their developing fetus during pregnancy or to their nursing infant in breast milk.

The EPA has set a non-enforceable guideline for the level of manganese in drinking water at 0.05 milligrams per liter (0.05 mg/L). The National Research Council has recommended safe and adequate daily intake levels for manganese that range from 0.3 to 1 mg/day for children up to 1 year, 1 to 2 mg/day for children up to age 10, and 2 to 5 mg/day for children 10 and older.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD.

HSDB is a toxicology data file on the National Library of Medicine's (NLM) Toxicology Data Network (TOXNET®). It focuses on the toxicology of potentially hazardous chemicals. It is enhanced with information on human exposure, industrial hygiene, emergency handling procedures, environmental fate, regulatory requirements, and related areas. All data are referenced and derived from a core set of books, government documents, technical reports and selected primary journal literature. HSDB is peer-reviewed by the Scientific Review Panel (SRP), a committee of experts in the major subject areas within the data bank's scope. HSDB is organized into individual chemical records, and contains over 4500 such records.

The following is the HSDB summary of human health and environmental fate information:

HUMAN HEALTH EFFECTS:

HUMAN TOXICITY EXCERPTS:

DIVALENT MANGANESE(2+) IS ABOUT 2.5 TO 3 TIMES MORE TOXIC THAN IS MANGANESE(3+) ... THE ANION OF A MANGANESE SALT INFLUENCES THE OVERALL MANGANESE TOXICITY. INHALATION OF MANGANESE CMPD IN AEROSOLS OR FINE DUSTS PRODUCES "METAL FUME FEVER". /MANGANESE AND MANGANESE SALTS/ [Venugopal, B. and T.D. Luckey. Metal Toxicity in Mammals, 2. New York: Plenum Press, 1978. 265]**PEER REVIEWED**

THE USUAL FORM OF CHRONIC MANGANESE POISONING PRIMARILY INVOLVES CNS. EARLY SYMPTOMS INCL LANGUOR, SLEEPINESS, & WEAKNESS IN LEGS. A STOLID MASKLIKE APPEARANCE OF FACE, EMOTIONAL DISTURBANCES SUCH AS UNCONTROLLABLE LAUGHTER, & SPASTIC GAIT WITH TENDENCY TO FALL IN WALKING ARE FINDINGS IN MORE ADVANCED CASES. [American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH:American Conference of Governmental Industrial Hygienists, 1986. 354]**PEER REVIEWED**

ONSET /OF CHRONIC POISONING/ IS INSIDIOUS, WITH APATHY, ANOREXIA, & ASTHENIA. MANGANESE PSYCHOSIS ... HAS CERTAIN DEFINITIVE FEATURES: UNACCOUNTABLE LAUGHTER, EUPHORIA, IMPULSIVENESS, & INSOMNIA, FOLLOWED BY OVERPOWERING SOMNOLENCE. HEADACHE ... LEG CRAMPS; SEXUAL EXCITEMENT, FOLLOWED BY IMPOTENCE /ARE OFTEN PRESENT/. [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 1762]**PEER REVIEWED**

FOLLOWING OR CONCOMITANTLY WITH ... /MANIFESTATIONS OF MANGANESE PSYCHOSIS/ ARE SPEECH DISTURBANCES WITH SLOW & DIFFICULT ARTICULATION, INCOHERENCE, EVEN COMPLETE MUTENESS. MASK-LIKE FACIES SETS IN ... WITH GENERAL CLUMSINESS OF MOVEMENT, NOTICEABLE IN ALTERED GAIT & BALANCE ... . [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 1762]**PEER REVIEWED**

ABSOLUTE DETACHMENT, BROKEN BY SPORADIC & SPASMODIC LAUGHTER, ENSUES & AS IN EXTRAPYRAMIDAL AFFECTIONS, SALIVATION & EXCESSIVE SWEATING

OCCUR. DESPITE SEVERE INCAPACITATIONS ... PT SURVIVES, ALTHOUGH PERMANENTLY DISABLED UNLESS TREATED; CHRONIC MANGANESE POISONING IS NOT A FATAL DISEASE. [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 1762]**PEER REVIEWED**

STEVEDORES REGULARLY EMPLOYED IN HANDLING MANGANESE ORES DEVELOPED PNEUMONIA FROM WHICH 31% DIED, & ... /CASES OF/ PNEUMONIA IN NORWEGIAN WORKERS FOLLOWING INTRODUCTION OF AN ELECTRIC FURNACE FOR MANGANESE ORE, /&/ ... PNEUMONIAS FROM DRILLING & BLASTING IN UNDERGROUND MOROCCAN MINES ... /ARE REPORTED/. [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 1762]**PEER REVIEWED**

... /MANGANESE PSYCHOSIS/ IS NOTABLY ABSENT FROM REPORTS OF MANGANESE POISONING IN STEEL FOUNDRIES & ORE-CRUSHING PLANTS IN UNITED STATES. /MANGANESE/ [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 1755]**PEER REVIEWED**

THERE IS CURRENTLY NO EVIDENCE THAT HUMAN EXPOSURE TO MANGANESE AT LEVELS
COMMONLY OBSERVED IN AMBIENT ATMOSPHERE RESULTS IN ADVERSE HEALTH EFFECTS.
ONLY ... HEALTH EFFECTS ATTRIBUTABLE TO MANGANESE IN AMBIENT AIR WERE FOUND IN PERSONS LIVING IN IMMEDIATE VICINITY OF 2 MAJOR POINT SOURCES IN NORWAY & ITALY. [National Research Council. Drinking Water & Health Volume 1. Washington, DC: National Academy Press, 1977. 268]**PEER REVIEWED**

... /THE HOMEOSTATIC SYSTEM/ REGULATING MECHANISM, PLUS TENDENCY FOR EXTREMELY LARGE DOSE OF MANGANESE SALTS TO CAUSE GI IRRITATION, ACCOUNTS FOR LACK OF SYSTEMIC TOXICITY FOLLOWING ORAL ADMIN OR DERMAL APPLICATION. [Doull, J., C.D. Klaassen, and M. D. Amdur (eds.). Casarett and Doull's Toxicology. 2nd ed. New York: Macmillan Publishing Co., 1980. 450]**PEER REVIEWED**

... MANGANESE TOXICITY IN MAN ARISING FROM EXCESSIVE INTAKES IN FOODS & BEVERAGES HAS NEVER BEEN REPORTED & IS DIFFICULT TO VISUALIZE EVER ARISING, EXCEPT WHERE INDUSTRIAL CONTAMINATION OCCURS. [National Research Council. Drinking Water and Health. Volume 3. Washington, DC: National Academy Press, 1980. 336]**PEER REVIEWED**

... WHILE HIGH LEVELS OF MANGANESE MAY INCREASE ANEMIA BY INTERFERING WITH IRON ABSORPTION, IRON DEFICIENCY MAY INCREASE AN INDIVIDUAL'S SUSCEPTIBILITY TO MANGANESE POISONING. [National Research Council. Drinking Water and Health. Volume 3. Washington, DC: National Academy Press, 1980. 336]**PEER REVIEWED**

MOST CONSPICUOUS INVOLVEMENT OF EYES IS IN DECR MOVEMENT OF EYELIDS & EYES. IT IS SAID, HOWEVER, THAT NEITHER PARESIS OF EYE MUSCLES NOR NYSTAGMUS OCCURS, & THAT MANGANESE POISONING DIFFERS FROM POSTENCEPHALITIC PARKINSONISM IN HAVING NO ACCOMPANYING OCULOGYRIC CRISIS OR LOSS OF BELL'S PHENOMENON. [Grant, W.M. Toxicology of the Eye. 3rd ed. Springfield, IL: Charles C. Thomas Publisher, 1986. 575]**PEER REVIEWED**

CHRONIC MANGANESE TOXICITY ... FOLLOWING CHRONIC EXPOSURE TO MANGANESE THROUGH INHALATION ... FOR PERIODS OF FROM 6 MO TO 2 YR RESULTS IN "MANGANISM", A DISEASE OF CNS INVOLVING PSYCHIC & NEUROLOGICAL DISORDERS. ... /IT/ IS REVERSIBLE IF RECOGNIZED EARLY & ... EXPOSURE ... ELIMINATED. [Venugopal, B. and T.D. Luckey. Metal Toxicity in Mammals, 2. New York: Plenum Press, 1978. 267]**PEER REVIEWED**

... Marked differences in individual susceptibility to inhaled manganese ... may have been caused by alcoholism, syphilis, carbon monoxide, lesions of the excretory system, or the physiological or pathological condition of the respiratory tract. ... [USEPA; Health Assessment Document: Manganese p.9-5 (1984) EPA-600/8-83-013F]**PEER REVIEWED**

Individual susceptibility to the adverse effects of manganese varies considerably. The minimum dose that produces effects on the central nervous system is not known and, with few exceptions, such effects have been observed only in occupationally exposed individuals. Only one epidemiological report is available on adverse effects from drinking water contaminated with manganese. Sixteen cases of manganese poisoning, three of which were fatal (including one suicide), in a small Japanese community, have been described. About 400 dry cell batteries were found buried within 2 m of a well used as a water supply. The manganese content of the water was about 14 mg/l and concentrations of about 8 and 11 mg/l were found in two other wells. The subjects exhibited psychological and neurological disorders associated with manganese poisoning, and high manganese and zinc levels were found in organs at autopsy. [WHO; Environ Health Criteria: Manganese-Executive Summary p.4 (1981)]**PEER REVIEWED**

Acute systemic intoxication rarely occurs after oral administration. ... Aside from parenteral routes, systemic poisoning may result from chronic inhalation or chronic ingestion; chronic exposure to low concentrations may lead to the accumulation of toxic concentrations in critical organs. The brain appears to sustain permanent cellular damage at exposure levels which do not otherwise affect a person. The characteristic pathological lesion in man is destruction of the ganglion cells of the basal ganglia, although symptoms appear before damage becomes discernible. Symptoms of workers exposed to manganese dusts include masklike facial expression, spastic gait, tremors, slurred speech, sometimes dystonia, fatiguability, anorexia, asthenia, apathy, and inability to concentrate. /Manganese salts/ [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. II-144]**PEER REVIEWED**

Manganese poisoning is clinically characterized by the central nervous system involvement including psychiatric symptoms, extrapyramidal signs, and other neurological manifestations. The onset of symptoms is usually insidious and progressive ... . The initial manifestations are usually vague complaints of asthenia, anorexia, apathy, insomnia or drowsiness, and a slowing down in performing motor acts. ... Other frequent symptoms of the early and established phases of the poisoning are malaise, somnolence, imbalance while walking or on arising, slurred speech, difficulty with line movements (handwriting), limb stiffness, diminished libido or impotence. Sometimes, mental languor and lack of energy are prominent symptoms at the onset. Tremor, paresthesia, muscle cramps, memory loss, swallowing difficulty, urinary urgency or incontinence,

lumbosacral pain, metallic taste, anorexia, and nervousness are less frequent ... . /Manganese/ [Chang, L.W. (ed.). Toxicology of Metals. Boca Raton, FL: Lewis Publishers, 1996 416]**PEER REVIEWED**

Psychiatric symptoms are well described in the manganese miners and include sleep disturbance, disorientation, emotional liability, compulsive acts, hallucinations, illusions, and delusions. A marked somnolence is also observed, most often to be replaced later by stubborn insomnia. Most of the cases show emotional incontinence, particularly forced laughing. ... Patients may abruptly burst into laughter or (more rarely) into tears without any apparent reason. Frequent irritability and nervousness resulted in arguments and friction among miners, occasionally approaching violence. /Manganese/ [Chang, L.W. (ed.). Toxicology of Metals. Boca Raton, FL: Lewis Publishers, 1996 416]**PEER REVIEWED**

... A population of Australian aborigines that exhibited signs of motor neuron disease similar, but not identical, to manganism seen among manganese miners /was identified/. These aborigines resided on Groote Eylandt in the Gulf of Carpenteria in northern Australia. Groote Eylandt has unusually rich deposits of manganese ore on or near the surface, and these have been commercially exploited. In a detailed survey of these natives, the authors determined that signs of intoxication were not simply related to potential exposure either in their home life or occupationally. Rather, the appearance of signs and symptoms was the result of a complex interplay of synergistic factors including genetics; inborn errors of trace element metabolism; life styles; dietary deficiencies of dopamine oxidation inhibitors, thiamine, and ascorbic acid; calcium deficiency; and possibly, smoking and excessive alcohol intake. /Manganese/ [Clayton, G.D., F.E. Clayton (eds.) Patty's Industrial Hygiene and Toxicology. Volumes 2A, 2B, 2C, 2D, 2E, 2F: Toxicology. 4th ed. New York, NY: John Wiley & Sons Inc., 1993-1994. 2118]**PEER REVIEWED**

The results of a study of 30 workers exposed to manganese in two different steel mills in Sweden have been described in two publications. The exposed group comprised the 15 "most exposed" workers from each plant. These were compared to a control group of 60 workers from a steel mill in which there was no manganese exposure. Exposed workers were matched 1 to 2 with control workers on the basis of age, geographic area of residence, and type of work. The concentration of manganese in the breathing zone of the exposed group was measured at the time of the study, and the mean concentration ranged from 0.19 to 0.45 mg/cu m. Peak exposures as high as 1.62 mg/ml were reported. ... There had been no change in exposure levels for the past 17 or 18 years. The duration of exposure ranged from 1 year to 35 years or more. ... There were no differences between the exposed and control subjects with respect to general health status and medical history or in any of the psychiatric examinations. Two of the neurological examinations, auditory-evoked response and diadochokinesometry, revealed slight differences between exposed and control workers. /Manganese/ [Clayton, G.D., F.E. Clayton (eds.) Patty's Industrial Hygiene and Toxicology. Volumes 2A, 2B, 2C, 2D, 2E, 2F: Toxicology. 4th ed. New York, NY: John Wiley & Sons Inc., 1993-1994. 2115]**PEER REVIEWED**

The 132 employees of the same factory were divided into four groups, based on the nature of their job assignments and their probable exposure to manganese. The 17 workers in groups 0 were office workers and probably experienced no exposure to manganese, whereas the 24 men in group 3 and

the 8 men in group 4 were furnace workers in the smelting department. Workers in group 4, which included the six cases of manganese intoxication ... experienced the greatest exposure because they operated electrodes at the furnaces and for about 30 min every day were exposed to airborne concentrations of manganese that were estimated to be as high as 28.8 mg/cu m. Measurements made at the time of the study indicated that workers in exposure group 1 were exposed to average concentrations of manganese in air of 0.1 mg/cu m and those in group 2 to 0.5 to 1.5 mg/cu m. Parkinsonism was not diagnosed in any workers other than the six in group 4. However, some symptoms of neurological impairment (e.g., muscle weakness, muscle cramps, loss of libido) were increased in groups 1 and 2 compared to group 0. Blood samples were collected from all workers; the mean blood concentrations in the four exposure groups were 1.49 + 0.92, 2.52 + 0.86, 3.13 + 1.56, and 14.6 + 15.5 ug/100 ml, showing a clear correlation between the level of exposure and the concentration of manganese in the blood. However, ... no correlation between duration of exposure and concentrations of manganese in the blood and ... blood levels were indicators of current exposure, but not of chronic exposure. /Manganese/ [Clayton, G.D., F.E. Clayton (eds.) Patty's Industrial Hygiene and Toxicology. Volumes 2A, 2B, 2C, 2D, 2E, 2F: Toxicology. 4th ed. New York, NY: John Wiley & Sons Inc., 1993-1994. 2115]**PEER REVIEWED**

Clinical examination of the six workers revealed bradykinesia, masklike facial features, clumsiness, impaired dexterity, and abnormal gait. Three of the workers had mild tremor and micrographia. ... Measurement of manganese concentrations in blood, scalp hair, and pubic hair from these workers revealed values that were from 3 to 300 times normal concentrations. /Manganese/ [Clayton, G.D., F.E. Clayton (eds.) Patty's Industrial Hygiene and Toxicology. Volumes 2A, 2B, 2C, 2D, 2E, 2F: Toxicology. 4th ed. New York, NY: John Wiley & Sons Inc., 1993-1994. 2114]**PEER REVIEWED**

Studies of neurologic and psychologic symptoms in workers exposed to manganese suggest that exposure to airborne dust below (5 mg/cu m) for 1 year or more may still lead to clinical signs of intoxication, especially respiratory symptoms, changes in lung ventilatory parameters, alteration of neurofunctional performances, & hypercalcemia. /Manganese/ [Ellenhorn, M.J., S. Schonwald, G. Ordog, J. Wasserberger. Ellenhorn's Medical Toxicology: Diagnosis and Treatment of Human Poisoning. 2nd ed. Baltimore, MD: Williams and Wilkins, 1997. 1587]**PEER REVIEWED**

"Locura manganica" or "manganese madness" is the insidious onset of psychiatric symptoms, including apathy, insomia, confusion, bizarre behavior, visual hallucinations, emotional lability, decr libido, impotence, & anxiety. Neurologic manifestations include nystagmus, disequilibrium, paresthesia, memory impairment, a vocal pattern described as "whispering speech", problems with fine motor movement, lumbosacral pain, urgency, & incontinence. The neurologic syndrome is similar to Parkinson's disease with tremor, ataxia, loss of memory, flat affect, muscle rigidity, & gait disturbances. Unlike Parkinson's, however pathologic lesions are found in the globus pallidus & the striatum rather than the globus pallidus & the substantia nigra. /Manganese/ [Ellenhorn, M.J., S. Schonwald, G. Ordog, J. Wasserberger. Ellenhorn's Medical Toxicology: Diagnosis and Treatment of Human Poisoning. 2nd ed. Baltimore, MD: Williams and Wilkins, 1997. 1587]**PEER REVIEWED**

The most common respiratory symptom is dyspnea. Because of its low solubility in water, airborne manganese does not cause oral or dermal problems. Instead, it penetrates the lower respiratory tract toward the alveolar membrane, leading to the development of manifestations of pneumonitis, pneumonia, & bronchitis. /Manganese/ [Ellenhorn, M.J., S. Schonwald, G. Ordog, J. Wasserberger. Ellenhorn's Medical Toxicology: Diagnosis and Treatment of Human Poisoning. 2nd ed. Baltimore, MD: Williams and Wilkins, 1997. 1587]**PEER REVIEWED**

The usual form of chronic manganese poisoning primarily involves the central nervous system (CNS). Early symptoms include languor, sleepiness, and weakness in the legs. A stolid, mask like appearance of the face emotional disturbances such as uncontrollable laughter, and spastic gait with a tendency to fall when walking are findings in more advanced cases. In addition, a high incidence of pneumonia has been found in workers exposed to the dust or fume of some manganese compounds. [American Conference of Governmental Industrial Hygienists, Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. Volumes I,II, III. Cincinnati, OH: ACGIH, 1991. 876]**PEER REVIEWED**

... Manganese concentrations up to 170 Mg/cu m and averaging 47 Mg/cu m /were reported/ in a mill where 11 of 34 employees were found to suffer from manganese poisoning. No cases occurred among workers exposed at less than 30 Mg/cu m. However, studies in another ore mill with dusty operations, where workers performed similar tasks with more modern equipment and local exhaust ventilation, revealed manganese concentrations averaging 2.3 Mg/cu m (from two air samples), with 6 Mg/cu m at the dustiest operation. [American Conference of Governmental Industrial Hygienists, Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. Volumes I,II, III. Cincinnati, OH: ACGIH, 1991. 876]**PEER REVIEWED**

... 5 Cases /were described/ showing signs indicative of chronic manganese in a study of 71 employees working in a steel mill in Pennsylvania from 1957 to 1965. Of the three workers exposed to manganese fume, one worked as a pourer and had an average exposure of 13.3 mg/cu m ; a second was a "hot blastman" with an average exposure of 0.33 mg/cu m. The third was a general laborer in the blast furnace area. From the data presented, it can be estimated that the third worker's average exposure was about 0.8 Mg/cu m. The two employees exposed to manganese dust had worked in the plant since 1943. Starting in 1957, they worked in a newly installed crushing and screening unit. In 1958, breathing zone manganese concentrations were estimated to average 35 mg/cu m . Between 1958 and 1966, average dust exposures at the original unit and at one installed in 1962 varied from 0.7 to 30 mg/cu m manganese with a monthly average of about 20 mg/cu m. [American Conference of Governmental Industrial Hygienists, Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. Volumes I,II, III. Cincinnati, OH: ACGIH, 1991. 877]**PEER REVIEWED**

... 7 cases and 15 borderline cases of manganese /were recorded/ in 75 Pennsylvania plants where 144 workers were found exposed to manganese dust or fume concentrations exceeding 5 Mg/cu m. Of the seven cases, four resulted from exposure to manganese dust and three from manganese fumes. No cases were reported in 48 workers exposed at air concentrations of fume or dust of less than 5 Mg/cu m. Because the only results reported were

based on the criterion in use, i.e., whether or not the exposure of the affected workers exceeded 5 mg/cu m, the study is of little value in pinpointing the relative degree of hazard between manganese fume and dust. /Manganese dust and fume/ [American Conference of Governmental Industrial Hygienists, Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. Volumes I,II, III. Cincinnati, OH: ACGIH, 1991. 877]**PEER REVIEWED**

In a study on 72 Chilean miners exposed to manganese concn in air of 62.5-250 mg/cu m, 12 (16.5%) were found to have neurological disorders. The avg exposure time was 178 days, with a range of 49-480 days. A further study on 370 miners exposed to manganese concn in air of 0.5-46 mg/cu m showed that 15 workers (4%) had contracted typical manganese intoxication. ... the average time of exposure was 8 years, 2 months, with a range of 9 months- 16 years. [WHO; Environ Health Criteria 17: Manganese p.68 (1981)]**PEER REVIEWED**

16 cases of manganese poisoning /from drinking water/, 3 of which were fatal (including 1 suicide), in a small Japanese community /were studied/. About 400 dry-cell batteries were found buried within 2 m of a well used as a water supply. The manganese content of the water was about 14 mg/liter & concn of 8 & 11 mg/liter were found in two other wells. All 16 intoxicated subjects drank water from these wells. The subjects exhibited psychological & neurological disorders assoc with manganese poisoning, & high manganese & zinc levels were found in organs at autopsy. [WHO; Environ Health Criteria 17: Manganese p.72 (1981)]**PEER REVIEWED**

The primary target organs of managanese toxicity are the brain and the lungs. The toxicity to the brain is manifested as a chronic disorder of the central nervous system resembling Parkinsonism. Toxicity to the lungs is manifested as increased susceptibility to bronchitis or, in more serious cases, manganic pneumonia. [Zenz, C., O.B. Dickerson, E.P. Horvath. Occupational Medicine. 3rd ed. St. Louis, MO., 1994 543]**PEER REVIEWED**

ENVIRONMENTAL FATE/EXPOSURE SUMMARY:

Manganese compounds are found in the earth's crust in the form of numerous minerals such as pyrolusite, romanechite, manganite, hausmannite. Manganese compounds enter the atmosphere and aqueous environment from the weathering of rocks and windblown soil. Manganese compounds and ions may also be released by anthropogenic sources into the environment through their use as antiknock agents (methylcyclopentadienyl manganese tricarbonyl), antiseptics (potassium permanganate), catalysts (manganous acetate), dietary supplements (manganese chloride), dry cells (manganese chloride), feed additives (manganese sulfate, manganese carbonate), fertilizers (manganese sulfate), pesticides (potassium permanganate), and pigments (manganese sulfate). Manganese is multi-valent and can exist in the 2+, 3+, 4+, 6+, and 7+ oxidation states, with 2+, 3+, and 4+ being the dominant oxidation states in the environment. Manganese 2+ is the most stable oxidation state in water while manganese 3+ and 4+ compounds are immobile solids. Organic matter may reduce manganese 3+ and 4+ compounds, resulting in the formation of soluble manganese 2+ compounds. Soluble manganese 2+ compounds do not strongly complex to soil and organic matter. Thus manganese 2+ compounds are relatively mobile and may potentially leach into surface and groundwater. As ions or insoluble solids, most

manganese compounds are not expected to volatilize from water and moist soil surfaces. Manganese compounds, released into the ambient atmosphere are expected to exist in the particulate phase. In the particulate phase, manganese compounds may be removed from the air by wet and dry deposition. Manganese compounds do not bioconcentrate in humans and animals. However, manganese is an essential nutrient for most plants and animals. Dietary intake is the primary source of exposure to manganese compounds for humans. Occupational exposure to elevated levels of manganese compounds may occur through inhalation in the workplace where manganese compounds are produced or used. (SRC) **PEER REVIEWED**

Reimer, P. 1988. Environmental effects of manganese and proposed freshwater guidelines to protect aquatic life in British Columbia. Department of Chemical and Bio-Resource Engineering. University of British Columbia.

Manganese is a naturally occurring substances that is present in surface waters and biota. Aquatic organisms have exhibited toxic responses to manganese in surface waters and regulatory bodies in some jurisdictions have established guidelines for levels of manganese in surface water to protect aquatic life. In British Columbia, a guideline of 0.1 mg/L was established by the Ministry of Environment, Lands and Parks, although it was recognized that the scientific data on which this guideline was based were weak. Toxicity tests applicable to aquatic life in BC waters were commissioned to strengthen the relevant data base and to apply the British Columbia procedures for deriving water quality criteria in an effort to establish more defensible guidelines for the protection of aquatic life in BC. Acute and chronic toxicity tests were conducted on fish, invertebrates and freshwater algae. Acute tests included 48 and 96 hour LC50's, while chronic tests included reproduction, growth and survival endpoints. A range of organisms was chosen in order to evaluate the range of sensitivities to manganese. The possible relationship between water hardness and toxicity to manganese was also investigated at water hardnesses of 25, 100 and 250 mg/L CaCO3.

Data were also gathered from literature sources in support of the new toxicity information. Both acute and chronic studies were identified for fish species resident in BC fresh waters. The collective data were evaluated for suitability with respect to the BC water quality guideline derivation process. Toxicity test data that met the requirements for use in guideline derivation were screened for sensitivity in order to fulfill the objective of developing a guideline protective of the most sensitive aquatic organisms.

A pattern emerged whereby the concentrations of manganese at which adverse effects were observed increased with increasing water hardness. This pattern was identified in both the literature data and in all but one of the new toxicity tests commissioned by the Ministry of Environment, Lands and Parks. Acute and chronic regression equations were developed using the most sensitive data for various (in both cases six) water hardness values. The acute equation was $Y = 0.0441X + 1.81$ and the chronic equation was $Y = 0.0176 + 2.42$, where X = water hardness in mg/L CaCO3 and Y = Mn concentration in mg/L. The equations were used to predict manganese concentrations at water hardness increments of 25 mg/L CaCO3 over the hardness range of 25-325 mg/L CaCO3, a range that encompasses the vast majority of BC surface waters. A factor of safety of 0.25 was applied to the predicted concentrations to account for uncertainty and was based on scientific judgement and the strength of the data set used in the derivation process. The resulting acute manganese concentrations ranged from 0.6 to 3.8 mg/L and are proposed as guidelines for exposure of less than 96 hours. The resulting chronic manganese concentrations ranged from 0.6 to 1.9 mg/L and are proposed as guidelines for exposure exceeding 96 hours. While BC and other surface water data indicate that manganese rarely exceeds concentrations of 1 mg/L, it is recognized that natural events may result in periodic increases. The application of guidelines intended to protect aquatic life from anthropogenic sources of manganese should reflect this in the sampling methodology requirements.

U.S. Environmental Protection Agency. 1996. Integrated risk information system: Manganese. Office of Research and Development. Cincinnati, OH.

_I.A.1. Oral RfD Summary

Critical Effect	Experimental Doses*	UF	MF	RfD
CNS effects Human Chronic Ingestion Data NRC, 1989; Freeland-Graves et al., 1987; WHO, 1973;	NOAEL (food): 0.14 mg/kg-day LOAEL: None	1	1	1.4E-1 mg/kg-day

*Conversion Factors and Assumptions -- The NOAEL of 10 mg/day (0.14 mg/kg-day for 70 kg adult) for chronic human consumption of manganese in the diet is based on a composite of data from several studies.

II.A. Evidence for Human Carcinogenicity

__II.A.1. Weight-of-Evidence Characterization

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Existing studies are inadequate to assess the carcinogenicity of manganese.

__II.A.2. Human Carcinogenicity Data

None.

__II.A.3. Animal Carcinogenicity Data

Inadequate.

MTBE, CAS #1634-04-4 (methyl *tert*-butyl ether)

Data Point	Data Summary	Reference
Water solubility	48,000 mg/L	ATSDR 1996
K_{oc}	Log K_{oc} estimated as 1.05 and calculated as 2.89 (K_{oc}s = 11.2 and 776, respectively) Log K_{oc}s reported as 1.091, 1.035, 1.049 (K_{oc}s = 12.3,10.8, and 11.2, respectively)	ATSDR 1996 Malcolm Pirnie 1999
Soil half-life	Rapid volatilization from surface soils, little degradation in subsurface.	ATSDR 1996
BCF	Insignificant (BCF = 1.5 to 3, with levels rapidly declining after exposure ends). Log BCF was 0.18 in Japanese carp (BCF = 1.5).	ATSDR 1996 EFDB 2002
Ingestion toxicity	ATSDR derived an intermediate-duration minimal risk level of 0.3 mg/kg/day. An oral rat LD_{50} of 4.0 mL/kg was identified; this is equal to 2,962 mg/kg.	ATSDR 1996 HSDB 2002
Carcinogenicity	Possible human carcinogen at high doses. Cancer slope factor = 0.004 per mg/kg/day.	EPA 1997
Fish toxicity	Rainbow trout LC_{50} is 880 to 1,240 mg/L	Johnson 1998
Aq. invert. tox	*Ceriodaphnia dubia* LC_{50} is 340 to 680 mg/L	Johnson 1998
Aq. amph. tox	100 mg/L led to increased weight, stimulated metamorphosis; <2,000 mg/L had no lethal effect on European common frog tadpoles	Pauli et al. 2000

Agency for Toxic Substances and Disease Registry. 1996. Toxicological profile for methyl *tert*-butyl ether. Atlanta, GA. http://www.atsdr.cdc.gov/toxprofiles/tp91.html

ATSDR. See Agency for Toxic Substances and Disease Registry.

EFDB. See Environmental Fate Database.

Environmental Fate Database. 2002. On-line database. Syracuse Research Corporation. http://esc.syrres.com/efdb.htm

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

Johnson, M.L. 1998. Ecological risk of MTBE in surface waters. John Muir Institute of the Environment, University of California. Davis, CA.

Malcolm Pirnie, Inc. 1999. Technical memorandum: Evaluation of fate and transport of methyl tertiary butyl ether (MTBE) in gasoline following a small spill. Prepared for Oxygenated Fuels Association, Inc. Oakland, CA.

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters, Hull, Québec, Canada. http://www.cws-scf.ec.gc.ca/nwrc/ratl/about_e.htm

U.S. Environmental Protection Agency. 1997. Drinking water advisory: Consumer acceptability advice and health effects analysis on methyl tertiary-butyl ether (MtBE). EPA-822-F-97-009. Office of Water. Washington, DC.

Agency for Toxic Substances and Disease Registry. 1996. Toxicological profile for methyl *tert*-butyl ether. Atlanta, GA.

Report summarized by ATSDR in the form of ToxFAQs document; relevant sections follow:

ToxFAQs™ for Methyl *tert*-Butyl Ether, CAS# 1634-04-4, September 1997

What happens to methyl tert-butyl ether (MTBE) when it enters the environment? MTBE quickly evaporates from open containers and surface water, so it is commonly found as a vapor in the air. Small amounts of MTBE may dissolve in water and get into underground water. It remains in underground water for a long time. MTBE may stick to particles in water, which will cause it to eventually settle to the bottom sediment. MTBE may be broken down quickly in the air by sunlight. MTBE does not build up significantly in plants and animals.

How can methyl tert-butyl ether (MTBE) affect my health? Breathing small amounts of MTBE for short periods may cause nose and throat irritation. Some people exposed to MTBE while pumping gasoline, driving their cars, or working in gas stations have reported having headaches, nausea, dizziness, and mental confusion. However, the actual levels of exposure in these cases are unknown. In addition, these symptoms may have been caused by exposure to other chemicals. There are no data on the effects in people of drinking MTBE. Studies with rats and mice suggest that drinking MTBE may cause gastrointestinal irritation, liver and kidney damage, and nervous system effects. There is no evidence that MTBE causes cancer in humans. One study with rats found that breathing high levels of MTBE for long periods may cause kidney cancer. Another study with mice found that breathing high levels of MTBE for long periods may cause liver cancer. The Department of Health and Human Services (DHHS), the International Agency for Research on Cancer (IARC), and the EPA have not classified MTBE as to its carcinogenicity.

Has the federal government made recommendations to protect human health? The EPA has issued guidelines recommending that, to protect children, drinking water levels of MTBE not exceed 4 milligrams per liter of water (4 mg/L) for an exposure of 1-10 days, and 3 mg/L for longer-term exposures. The American Conference of Governmental Industrial Hygienists (ACGIH) has recommended an exposure limit of 40 parts of MTBE per million parts of air (40 ppm) for an 8-hour workday, 40-hour workweek.

Environmental Fate Database. 2002. On-line database. Syracuse Research Corporation. http://esc.syrres.com/efdb.htm

CHEMFATE search results: Methyl tert-butyl ether.

BIOC METHYL TERT-BUTYL ETHER CAS# 1634-04-4
Log Bioc. Fact.: 0.18
Species Name : JAPANESE CARP
Remarks : SRC SUGGESTED VALUE
Abbrev. Ref. : FUJIWARA,Y ET AL. (1984)

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD.

HSDB is a toxicology data file on the National Library of Medicine's (NLM) Toxicology Data Network (TOXNET®). It focuses on the toxicology of potentially hazardous chemicals. It is enhanced with information on human exposure, industrial hygiene, emergency handling procedures, environmental fate, regulatory requirements, and related areas. All data are referenced and derived from a core set of books, government documents, technical reports and selected primary journal literature. HSDB is peer-reviewed by the Scientific Review Panel (SRP), a committee of experts in the major subject areas within the data bank's scope. HSDB is organized into individual chemical records, and contains over 4500 such records.

The following is the HSDB summary of human health and environmental fate information:

HUMAN HEALTH EFFECTS:

EVIDENCE FOR CARCINOGENICITY:
A3. A3= Animal Carcinogen [American Conference of Governmental Industrial Hygienists. TLVs and BEIs. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Cincinnati, OH. 2000. 47]**QC REVIEWED**

Evaluation: There is limited evidence in humans for the carcinogenicity of methyl tert-butyl ether. There is limited evidence in experimental animals for the carcinogenicity of methyl tert-butyl ether. Overall evaluation: Methyl tert-butyl ether is not classifiable as to it carcinogenicity to humans (Group 3). [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. 73 375 (1999)]**QC REVIEWED**

HUMAN TOXICITY EXCERPTS:
A case of acute renal failure is reported in one of 8 patients (aged 37-75 yr) with a history of biliary colic and radiolucent gallstones who were given continuous methyl tert-butyl ether (MTBE I) infusion through a catheter, 5-10 ml for 7 hr. Hemolysis due to extravasation of MTBE after leakage alongside the catheter was suspected as the cause of the renal failure. Dialysis over 18 days was required before renal function recovered completely. [Ponchon T et al; Lancet 2 (July 30): 276-277 (1988)]**PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE:
NIOSH (NOES Survey 1981-1983) has statistically estimated that 3,522 workers (971 of these are female) are potentially exposed to methyl t-butyl ether in the US(1). Occupational exposure to methyl t-butyl ether may occur during its production or subsequent use, particularly in gasoline, via inhalation or dermal contact. The general population may be exposed to methyl t-butyl ether via inhalation of ambient air especially during refueling operations and from ingestion of ambient and drinking water(SRC). [(1) NIOSH; National Occupational Exposure Survey (NOES) (1983)]**PEER REVIEWED**

Methyl t-butyl ether arithmetic mean concentrations (ug/cu m) in air were

1,500 for manufacturing workers, 5,000 for blending workers, 14,000 transportation workers, 2,600 for distribution workers, 5,200 for gasoline station workers, 660 for mechanics, 61 for professional drivers, 61 for commuters, 30 for other drivers, 390 for gasoline station customers, 4 for manufacturing and blending neighbors, 66 for gasoline station neighbors, and 2.6 for the general public(1). Time-weighted personal-breathing-zone samples among mechanics who repaired motor vehicles ranged from less than 108 ug/cu m to 43,464 ug/cu m(2). A methyl t-butyl ether concentration of 412 ug/cu m was detected in a breathing zone grab sample collected during refueling; ambient air grab samples collected at 2 and 16 minutes post refueling contained methyl t-butyl ether at concentrations of 16.8 and 23.4 ug/cu m, respectively(3). Exposure of Finnish tanker drivers to methyl t-butyl ether during loading and delivery was between 13 and 91 mg/cu m(4). Mean exposure of service station attendants to methyl t-butyl ether was 0.3 ppm (range 0.04 to 3.88 ppm) in 41 personal breathing zone air samples collected in the Phoenix, AZ area and 0.14 ppm (range 0.02 to 0.73 ppm) in 48 personal breathing zone air samples collected in the Los Angeles, CA area(5). [(1) Brown SL; Regul Toxicol Pharmacol. 25: 256-76 (1997) (2) White MC et al; An Investigation of Exposure to Methyl Tertiary Butyl Ether Among Motorists and Exposed Workers in Stamford, Connecticut. USEPA-600-R95-134. Proc Conf MTBE and Other Oxygenates, 1993 D42-D64 (1995) (3) Lindstrom AB, Pleil JD; J Air Waste Manage Assoc 46: 676-82 (1996) (4) Hakkola M, Saarinen L; Ann Occup Hyg 40: 1-10 (1996) (5) Hartle R; Environ Health Perspect 101 (Supp 6): 23-6 (1994)]**PEER REVIEWED**

Occupational exposure to methyl t-butyl ether via short-term exposure, less than 30 minutes (TWA, between 6 and 9 hours) was 11.0 (0.24) ppm for transporting neat methyl t-butyl ether, 5.1 (0.58) ppm for blending neat methyl t-butyl ether, 4.7 (0.77) ppm for service station attendants, 3.3 (0.13) ppm for transporting a methyl t-butyl ether/fuel mix, 1.0 (0.14) ppm for manufacturing-maintenance, 0.85 (0.13) ppm for distributing methyl t-butyl ether, 0.84 (0.06) ppm for manufacturing-routine, and 0.58 (0.10) ppm for blending a methyl t-butyl ether/fuel mix(1). Long-term (93 to 570 minutes) methyl t-butyl ether exposure concentrations for refueling attendants were 0.5 ppm or less; winter and summer geometric mean exposures were 0.2 ppm and 0.08 ppm, respectively(2). Winter and summer mechanic geometric mean exposures to methyl t-butyl ether were 0.12 ppm and 0.03 ppm, respectively; only four individual methyl t-butyl ether samples exceeded 0.5 ppm, these four samples (0.63, 0.86, 1.3, and 2.6 ppm) were taken during shift where mechanics duties included fuel line servicing(2). Short-term (8 to 35 minutes) methyl t-butyl ether exposure for refueling attendants was less than 0.21 ppm, with winter and summer geometric mean exposures of 0.6 and 0.31 ppm, respectively(2). Individual mechanic short-term methyl t-butyl ether exposures were less than 0.91 ppm, with winter and summer geometric mean exposures of 1.04 and 0.42 ppm, respectively(2). [(1) American Petroleum Institute; Petroleum Industry Data Characterizing Occupational Exposures to Methyl Tertiary Butyl Ether (MTBE) 1983-1993. Washington,DC: Amer Petrol Instit, API Publ Noo 4622. Order No. I46220 (1995) (2) American Petroleum Institute; Service Station Personnel Exposures to Oxygenated Fuel Components - 1994. Washington,DC: Amer Petrol Instit, API Publ No 4625 . Oder No I46250 (1995)]**PEER REVIEWED**

ENVIRONMENTAL FATE/EXPOSURE SUMMARY:

Methyl t-butyl ether's production and use as an octane booster in gasoline and in the manufacture of isobutene may result in its release to the

environment through various waste streams. If released to air, a vapor pressure of 250 mm Hg at 25 deg C indicates methyl t-butyl ether will exist solely as a vapor in the ambient atmosphere. Vapor-phase methyl t-butyl ether will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals and nitrate radicals; half-lives for these reactions in air are estimated to be 5.5 and 50 days, respectively. Direct photolysis is not expected to be an important removal process since aliphatic ethers do not absorb light in the environmental spectrum. If released to soil, methyl t-butyl ether is expected to have very high mobility based upon a Koc of 6 calculated from a soil/water partition coefficient of 0.0925. Volatilization from moist soil surfaces is expected to be an important fate process based upon a Henry's Law constant of 5.87X10-4 atm-cu m/mole. Methyl t-butyl ether may potentially volatilize from dry soil surfaces based upon its vapor pressure. If released into water, methyl t-butyl ether is not expected to adsorb to suspended solids and sediment in the water column based upon the Koc. Volatilization from water surfaces is expected to be an important fate process based upon this compound's Henry's Law constant. Estimated volatilization half-lives for a model river and model lake are 4.1 hours and 4.1 days, respectively. A BCF of 1.5 in Japanese carp suggests bioconcentration in aquatic organisms is low. Methyl t-butyl ether is not expected to undergo hydrolysis in the environment due to the lack of hydrolyzable functional groups. In general, most studies have indicated that methyl t-butyl ether is difficult to biodegrade. t-Butyl alcohol was identified as a metabolite of methyl t-butyl ether in a study using an enrichment culture capable of degrading methyl t-butyl ether. Occupational exposure to methyl t-butyl ether may occur during its production or subsequent use, particularly in gasoline, via inhalation or dermal contact. The general population may be exposed to methyl t-butyl ether via inhalation of ambient air especially during refueling operations and from ingestion of ambient and drinking water. (SRC) **PEER REVIEWED**

Johnson, M.L. 1998. Ecological risk of MTBE in surface waters. John Muir Institute of the Environment, University of California. Davis, CA.

Conclusions

- MTBE is present in California's surface waters and aquatic organisms are exposed.
- There is little toxicity of MTBE to aquatic organisms, with the most sensitive taxonomic group tested being green algae.
- One experimental study indicates that fish accumulate MTBE to about 1.5 times the concentration of MTBE in the water column.
- The most conservative toxicity reference value calculated for rainbow trout is 7,000ppb.
- The most conservative hazard quotients for rainbow trout exposed to MTBE in two selected surface waters range from 1 x 10 -3 to 6 x 10 -3 , well below the level that indicates potential adverse ecological effects.
- Adverse effects on rainbow trout are not expected until concentrations of MTBE in the water column reach 4,600 ppb to 4,700 ppb. These levels are much greater than the human health standards for MTBE in drinking water supplies.

Malcolm Pirnie, Inc. 1999. Technical memorandum: Evaluation of fate and transport of methyl tertiary butyl ether (MTBE) in gasoline following a small spill. Prepared for Oxygenated Fuels Association, Inc. Oakland, CA.

Recently it has been suggested that small discrete spills of gasoline containing the fuel oxygenate, MTBE onto the ground surface will result in significant groundwater contamination from MTBE. The purpose of this screening level analysis is to determine whether several hypothetical small spill scenarios, defined as spills less than 4 gallons of gasoline, could result in substantial groundwater contamination by MTBE in comparison to the known contamination caused by releases of gasoline from underground storage tanks. For this analysis, we have divided a small gasoline spill into four stages: 1) spill occurrence; 2) surface evaporation; 3) infiltration; and 4) vadose zone transport to the water table. The literature devoted to analyzing the evaporation of individual gasoline components during the time between the initial spill (Stage 1) and the introduction to groundwater (Stage 4) is extensive. Specifically, researchers have concluded that in most situations, more than 99% of volatile gasoline components will evaporate within a few hours of the spill prior to transport through the vadose zone.

In this analysis, we show that under conservative geologic scenarios, approximately 0.2% of the initial mass of MTBE in a 4 gallon spill on concrete and approximately 0.7% of the initial mass of MTBE in a 1 gallon spill on soil will reach groundwater. Using a conservative mixing cell calculation, we show that this mass of MTBE entering groundwater will result in maximum concentrations of less than 11 ug/l within the immediate vicinity of the spill. For spills less than 1 gallon, we show that negligible amounts of MTBE are expected to reach the groundwater. Based on these highly conservative gasoline spill scenarios, MTBE impacts to groundwater from small spills will be significantly smaller than impacts caused by releases from leaking underground storage tanks (e.g., 200 ug/l). Thus, small spills are not expected to represent a significant source of groundwater contamination relative to other sources of MTBE groundwater contamination. Nonetheless, in limited circumstances, small spills will impact shallow aquifers, and thus, warrant the support of outreach programs on proper handling of gasoline, and instruction of the public on appropriate procedures to minimize the occurrence of small gasoline spills.

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters, Hull, Québec, Canada.

The RATL (Reptile and Amphibian Toxicology Literature) database contains data extracted from the primary literature for amphibian and reptile ecotoxicology studies published up to and including 1997; there are some data from studies published in 1998 and 1999. As of September, 2000, there was approximately 2000 references in the database. Citations were gathered through searches of various literature databases, but these searches concentrated on the environmental pollution literature with the result that the bibliography cannot be considered exhaustive.

U.S. Environmental Protection Agency. 1997. Drinking water advisory: Consumer acceptability advice and health effects analysis on methyl tertiary-butyl ether (MtBE). EPA-822-F-97-009. Office of Water. Washington, DC.

The EPA Office of Water is issuing this Advisory to provide guidance for communities that may be exposed to drinking water contaminated with MtBE. The Advisory provides an analysis of current health hazard information and an evaluation of currently available data on taste and odor problems associated with MtBE contamination of water, as the latter affect consumer acceptance of the water resource. This Advisory does not recommend either a low-dose oral cancer risk number or a reference dose (RfD) due to certain limitations of available data for quantifying risk. Guidance is given on the concentrations at which taste and odor problems likely would be averted, and how far these are from MtBE concentrations at which toxic effects have been seen in test animals. (The measure used is called a "margin of exposure" or MoE. For instance, if a measured concentration is 100,000 times less than the range of observation of effects in test animals, the margin of exposure is 100,000.

Conclusion and Recommendation

This Advisory recommends that keeping levels of contamination in the range of 20 to 40 µg/L or below to protect consumer acceptance of the water resource would also provide a large margin of exposure (safety) from toxic effects. Taste and odor values are presented as a range, since human responses vary depending upon the sensitivities of the particular individual and the site-specific water quality conditions. These values are provided as guidance recognizing that water suppliers determine the level of treatment required for aesthetics based upon the customers they serve and the particular site-specific water quality conditions. There are over four to five orders of magnitude between the 20 to 40 µg/L range and concentrations associated with observed cancer and noncancer effects in animals. There is little likelihood that an MtBE concentration of 20 to 40 g/L in drinking water would cause adverse health effects in humans, recognizing that some people may detect the chemical below this range. It can be noted that at this range of concentrations, the margins of exposure are about 10 to 100 times greater than would be provided by an EPA reference dose (RfD) for noncancer effects. Additionally, they are in the range of margins of exposure typically provided by National Primary Drinking Water Standards under the Federal Safe Drinking Water Act to protect people from potential carcinogenic effects.
When adequate data become available, the Office of Water will publish another Advisory that includes quantitative estimates for health risks. This Advisory gives practical guidelines for addressing contamination problems and supersedes previous draft advisories. An Advisory does not mandate a standard for action.

Polystyrene, CAS #9003-53-6

Data Point	Data Summary	Reference
Water solubility	ND	
K_{oc}	ND	
Soil half-life	ND	
BCF	ND	
Ingestion toxicity	Not absorbed when administered orally to laboratory rats.	Monte 1983
Carcinogenicity	Subcutaneous implantation of polystyrene discs, rods, spheres or powder in rats induced local sarcomas, the incidences of which varied with the size and form of the implant.	IARC 1979
Fish toxicity	ND	
Aq. invert. tox	ND	
Aq. amph. tox	ND	

International Agency for Research on Cancer. 1979. Styrene, polystyrene, and styrene-butadiene compounds. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 19:231. http://193.51.164.11/htdocs/monographs/vol19/styrene%26polymers.html

Monte, W. 1983. Lack of gut absorption of solubilized polystyrene by the rat (abstract). Journal of Agricultural and Food Chemistry 31(1):174-175.

International Agency for Research on Cancer. 1979. Styrene, polystyrene, and styrene-butadiene compounds. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 19:231.

Evaluation

Although no information is available on carcinogenicity in humans attributable to styrene, its wide use and the facility with which it can be absorbed by inhalation indicate that it may be possible to carry out studies measuring both dose and cancer incidence in exposed workers. The finding of chromosomal aberrations in workers exposed to styrene further supports the need for epidemiological investigations.

Results from polystyrene implant studies in animals point to the need for further investigations with regard to the polymer. Recent epidemiological information on styrene-butadiene copolymer workers, which indicates lymphato-haematopoeietic malignancies, clearly requires elucidation by further studies.

Monte, W. 1983. Lack of gut absorption of solubilized polystyrene by the rat (abstract). Journal of Agricultural and Food Chemistry 31(1):174-175.

The absorption of solubilized polystyrene (9003536) was studied in rats. Carbon-14 labeled polystyrene, with a molecular weight range similar to that of commercial grades of expanded polystyrene, was dissolved in lemon oil. Male Long-Evans-rats were weighed and administered intragastrically 2 microCuries carbon-14 labeled polystyrene in 100 milliliter of lemon oil. Urine and feces were collected at 8 hour intervals. Weights, food eaten, and appearance of animals were recorded. Animals were killed 120 hours after polystyrene feeding. Samples of blood, skin, subcutaneous tissue, lungs and bronchi, trachea, bone marrow, spleen, lymph nodes, heart, liver, pancreas, stomach, large and small intestines, kidney, urinary bladder, testes, and brain were tested for carbon-14 activity. Urine and fecal material were tested by scintillation determination for carbon-14 activity. Rats appeared normal. None of the tissue samples showed any activity above background. Two urine samples, contaminated with fecal matter, showed slight activity. Within the bounds of experimental error, all the carbon-14 was found in the fecal samples and 99 percent was excreted within 48 hours after intubation. The author concludes that polystyrene solubilized in an absorbable solvent does not pass through the intestinal barrier of rats.

Potassium Chloride, CAS # 7447-40-7 (KCl), and Potassium Hydroxide, CAS # 1310-58-3 (KOH)

Data Point	Data Summary	Reference
Water solubility	281,000 mg/L (KCl) and 970,000 mg/L (KOH)	HSDB 2002
K_{oc}	No data.	
Soil half-life	No data.	
BCF	No data.	
Ingestion toxicity	Maximal nontoxic oral dose of KCl in man varies from 200 to 1,000 mg/kg/day, depending on efficiency of individual renal excretory mechanism. KOH is one of the strongest alkalies--it is extremely corrosive. Swallowing caustic alkalies causes immediate burning pain in the mouth, throat, and stomach, and the lining membranes become swollen and detached.	HSDB 2002
Carcinogenicity	No data.	
Fish toxicity	EPA has set an ambient water quality criteria level of 230 mg/L for chloride for the protection of freshwater aquatic life.	EPA 1999
Aq. invert. tox		
Aq. amph. tox		

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

U.S. Environmental Protection Agency. 1999. National recommended water quality criteria--Correction. EPA 822-A-99-01. Office of Water. Washington, DC.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD.

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The following is the HSDB summary of human health and environmental fate information:

HUMAN HEALTH EFFECTS:

HUMAN TOXICITY EXCERPTS:

LARGE DOSES BY MOUTH CAN CAUSE GI IRRITATION, PURGING, WEAKNESS AND CIRCULATORY DISTURBANCES. [The Merck Index. 9th ed. Rahway, New Jersey: Merck & Co., Inc., 1976. 990]**PEER REVIEWED**

AN 84-YR-OLD WOMAN WITH...MANY EPISODES OF CONGESTIVE FAILURE WAS IN... CONTROLLED CARDIAC STATUS WHEN SHE COMMITTED SUICIDE BY INGESTING...LIQ POTASSIUM SUPPLEMENT. EST DOSE...(EQUIV TO ABOUT 40 TO 50 G KCL)... GRAND MAL CONVULSION OCCURRED AFTER 1 HR FOLLOWED BY COMA...BLOOD PRESSURE WAS UNOBTAINABLE. /POTASSIUM SALTS/ [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. II-124]**PEER REVIEWED**

NAUSEA, VOMITING, DIARRHEA, & ABDOMINAL DISCOMFORT COMMONLY OCCUR. OVERDOSES MAY CAUSE PARESTHESIAS, GENERALIZED WEAKNESS, FLACCID PARALYSIS,
LISTLESSNESS, VERTIGO, MENTAL CONFUSION, HYPOTENSION, CARDIAC ARRHYTHMIAS, & HEART BLOCK. DEATH MAY ENSUE. [Osol, A. and J.E. Hoover, et al. (eds.). Remington's Pharmaceutical Sciences. 15th ed. Easton, Pennsylvania: Mack Publishing Co., 1975. 771]**PEER REVIEWED**

ACUTE POTASSIUM INTOXICATION BY MOUTH IS RARE BECAUSE LARGE SINGLE DOSES USUALLY INDUCE VOMITING AND BECAUSE IN THE ABSENCE OF PRE-EXISTING KIDNEY DAMAGE POTASSIUM IS RAPIDLY EXCRETED. /POTASSIUM SALTS/ [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. II-124]**PEER REVIEWED**

Potassium chloride in a commercial dietary salt substitute ... has produced a near fatal poisoning in an 8 month old infant. [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. II-124]**PEER REVIEWED**

MAXIMAL NONTOXIC ORAL DOSE OF KCL IN MAN VARIES FROM 0.2 TO 1.0 G K/KG/DAY, DEPENDING UPON EFFICIENCY OF INDIVIDUAL RENAL EXCRETORY MECHANISM; LOWER DOSES SOMETIMES CAUSE IMPAIRMENT OF RENAL FUNCTION AS SHOWN BY REDUCED INULIN & UREA CLEARANCE. ... SERUM K LEVEL OF 40 MG/100 ML IS FATAL IN MAN. [Venugopal, B. and T.D. Luckey. Metal Toxicity

in Mammals, 2. New York: Plenum Press, 1978. 16]**PEER REVIEWED**

ENVIRONMENTAL FATE & EXPOSURE:

NATURAL POLLUTION SOURCES:
A main commercial product is sylvite, KCl [Harben PW, Bates RL; Geology of the Nonmetallics, p.246 (1984)]**PEER REVIEWED**

KCl makes up 4% of the salts in the Great Salt Lake; present in the Bonneville Salt Flats and Searles Lake & the Paradox Basin [Harben PW, Bates RL; Geology of the Nonmetallics, p.246 (1984)]**PEER REVIEWED**

U.S. Environmental Protection Agency. 1999. National recommended water quality criteria--Correction. EPA 822-A-99-01. Office of Water. Washington, DC.

SUMMARY: EPA is publishing a compilation of its national recommended water quality criteria for 157 pollutants, developed pursuant to section 304(a) of the Clean Water Act (CWA or the Act). These recommended criteria provide guidance for States and Tribes in adopting water quality standards under section 303(c) of the CWA. Such standards are used in implementing a number of environmental programs, including setting discharge limits in National Pollutant Discharge Elimination System (NPDES) permits. These water quality criteria are not regulations, and do not impose legally binding requirements on EPA, States, Tribes or the public.

Silicon Dioxide, CAS #7631-86-9 (silica)

Data Point	Data Summary	Reference
Water solubility	Practically insoluble.	HSDB 2002
K_{oc}	Not applicable.	
Soil half-life	Stable (occurs as sand and quartz).	HSDB 2002
BCF	None.	
Ingestion toxicity	When male and female beagle dogs or CD rats were fed 800 mg silicon/kg/day as the dioxide for 1 month ... neither clinical signs of toxicity nor histologic changes were seen in these animals. It is chemically and biologically inert when ingested. It is approved for use in food products at levels up to 2%, and is Generally Recognized as Safe (GRAS).	HSDB 2002, EPA 2002
Carcinogenicity	Crystalline silica is carcinogenic.	HSDB 2002, EPA 1991
Fish toxicity	Chemically unreactive in the environment, occurs naturally in various forms and is practically non-toxic to non-target organisms.	EPA 1991
Aq. invert. tox		
Aq. amph. tox		

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

U.S. Environmental Protection Agency. 1991. Reregistration eligibility document: Silicon dioxide and silica gel. Office of Pesticide Programs. Washington, DC. http://www.epa.gov/oppsrrd1/REDs/old_reds/4081red.pdf

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD.

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The following is the HSDB summary of human health and environmental fate information:

Human Health Effects:

Human Toxicity Excerpts:

The details of toxicity associated with metallurgical silicon are unknown.
[Kirk-Othmer Encyclopedia of Chemical Technology. 3rd ed., Volumes 1-26. New York, NY: John Wiley and Sons, 1978-1984.,p. V20 851 (1982)]**PEER REVIEWED**

Nuisance particulate (accumulation in lungs).
[Cralley, L.J., L.V. Cralley (eds.). Patty's Industrial Hygiene and Toxicology. Volume III: Theory and Rationale of Industrial Hygiene Practice. 2nd ed., 3A:The Work Environment. New York, NY: John Wiley Sons, 1985. 181]**PEER REVIEWED**

... Increased renal silicon (200 ppm dry weight; normal = 14-23 ppm) /was found/in an adult male bricklayer who presented with proteinuria and hypertension, but who had a normal chest roentgenogram. Moderate thickening of the glomerular basement membrane was noted on transmission electron microscopy.
[American Conference of Governmental Industrial Hygienists, Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. Volumes I,II, III. Cincinnati, OH: ACGIH, 1991. 1387]**PEER REVIEWED**

Skin, Eye and Respiratory Irritations:

Unpleasant deposits /of silicon dust/ in eyes, ears & nasal passages & injury to the skin and mucous membranes may be caused by the dust itself or by cleansing procedures used for its removal.
[Sittig, M. Handbook of Toxic and Hazardous Chemicals and Carcinogens, 1985. 2nd ed. Park Ridge, NJ: Noyes Data Corporation, 1985. 787]**PEER REVIEWED**

Environmental Fate & Exposure:

Natural Pollution Sources:

Silicon is not found free in nature, but occurs chiefly as the oxide, & as silicates. Sand, quartz, rock crystal, amethyst, agate, flint, jasper, & opal are some of the /oxide/ forms. Granite, hornblende, asbestos, feldspar, clay, mica ... are but a few of the numerous silicate minerals.
[Lide, D.R. (ed.). CRC Handbook of Chemistry and Physics. 73rd ed. Boca Raton, FL: CRC Press Inc., 1992-1993.,p. 4-26]**PEER REVIEWED**

THREE NATURALLY OCCURRING ISOTOPES: 28 (92.18%); 29 (4.71%); 30 (3.12%) ... FOUND ASSILICA (... SANDSTONE) OR AS SILICATE (... ORTHOCLASE, KAOLINITE, ANORTHITE). CONSTITUTES ABOUT 27.6% OF EARTH'S CRUST; SECOND MOST ABUNDANT ELEMENT ON EARTH ... .
[Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989. 1346]**PEER REVIEWED**

U.S. Environmental Protection Agency. 1991. Reregistration eligibility document: Silicon dioxide and silica gel. Office of Pesticide Programs. Washington, DC.

This Reregistration Eligibility Document addresses both silicon dioxide and silica gel. Silicon dioxide is essentially an inert material that contains approximately 90% silica. It is commonly used as an inert carrier in dry concentrates, dry pesticides, as an anti-caking agent, soil conditioner and turf soil supplement and occasionally used as an active ingredient. Silicon dioxide's most common insecticidal use today is for control of stored grain insects. It is also registered for use to control a variety of insects/mites in and around domestic/commercial dwellings, ornamental gardens, in kennels and on domestic pets. Silica gel is a registered insecticide and acaricide for use to control a variety of insects in and around residences/commercial dwellings, agricultural premises, institutions, warehouses, food plants, livestock, cat, dogs and in granaries. Because of their abrasive characteristics both active ingredients act on insects by removing the oily protective film covering their bodies which normally prevents the loss of water. Thus the mode of action is physical in nature causing
desiccation of the insect. Both active ingredients are usually combined with other pesticides which act as a knockdown agent. All products which contain silicon dioxide and silica gel registered for these uses are eligible for reregistration.

The U. S. Environmental Protection Agency (EPA) conducted a review of the scientific data base and other relevant information supporting the reregistration of silicon dioxide and silica gel and has determined that the data base is sufficient to conduct a reasonable risk assessment. In addition, the Agency has conducted a tolerance reassessment for silicon dioxide and silica gel and its conclusions are discussed in Section IIC. The data available to the EPA support the conclusion that the currently registered uses of silicon dioxide and silica gel will not result in unreasonable public health risks or effects to the environment. No further generic data are required.

Accordingly, the EPA has determined that all products containing silicon dioxide and silica gel as the active ingredients are eligible for reregistration and will be reregistered when appropriate labeling and/or product specific data are submitted and/or cited. Before reregistering each product, the EPA is requiring product specific data to be submitted within eight months of the issuance of this document. After reviewing these data and the revised labels, the EPA will determine whether to reregister a product based on whether or not the conditions of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) Section 3(c)(5) have been met. End use products containing silicon dioxide and silica gel in combination with other active ingredients will not be reregistered until those other active ingredients are determined to be eligible for reregistration. However, product specific data are being called in at this time.

Strontium Oxides and Sulfate, CAS # 1314-11-0 (strontium oxide, SrO), 1314-18-7 (strontium peroxide, SrO_2), 7759-02-6 (strontium sulfate, $SrS0_4$)

Data Point	Data Summary	Reference
Water solubility	SrO forms the hydroxide with evolution of heat in presence of water. SrO_2 is almost insoluble in water, but is gradually decomposed by water with the evolution of oxygen. $SrSO_4$ is soluble in water at about 114 mg/L.	Budavari et al. 1989
K_{oc}	The distribution coefficient, K_d (amount of ion per kg of air dry soil/amount of ion per liter of soil solution), for strontium in a podsol forest soil was determined to be 140 L/kg in the top layer and 44 L/kg in the lower layer.	HSDB 2002
Soil half-life	No data.	
BCF	BCF of strontium was 576 to 1,286 in bluegill sunfish.	HSDB 2002
Ingestion toxicity	The strontium ion has a low order of toxicity. It is chemically and biologically similar to calcium. The oxides are moderately caustic materials. The human daily intake of strontium has been determined to be 2 mg. An oral reference dose of 0.6 mg/kg/day was estimated for stable strontium. An oral rat LD_{50} of 2,750 mg/kg was identified for strontium nitrate Sr(NO3)2. This is equivalent to an LD_{50} of 1,139 mg strontium/kg.	Lewis 1994 HSDB 2002 EPA 1996 Oxford 2002
Carcinogenicity	No data.	
Fish toxicity	A 96-hour LC_{10} of 0.049 mg/L was identified for Sr for newly hatched rainbow trout.	EPA 2002
Aq. invert. tox	No data.	
Aq. amph. tox	7-day LC_{50} for Sr in eastern narrowmouth toad embryo-larvae was 0.16 mg/L	Pauli et al. 2000

Budavari, S., M. O'Neil, A. Smith, and P. Heckelman, eds. 1989. *The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals.* 11th ed. Merck & Co., Inc. Rahway, NJ.

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

Lewis, R. 1994. *Sax's Dangerous Properties of Industrial Materials*. 8th ed. Van Nostrand Reinhold Company. New York.

Oxford University. 2002. Safety data for strontium nitrate. The Physical and Theoretical Chemistry Laboratory. http://physchem.ox.ac.uk/MSDS/ST/strontium_nitrate.html

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters, Hull, Québec, Canada. http://www.cws-scf.ec.gc.ca/nwrc/ratl/about_e.htm

U.S. Environmental Protection Agency. 1996. Integrated risk information system. Office of Research and Development. Cincinnati, OH. http://www.epa.gov/iris/subst/0550.htm

U.S. Environmental Protection Agency. 2002. Ecotox database: Lead. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN. http://www.epa.gov/ecotox/

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD.

HSDB is a toxicology data file on the National Library of Medicine's (NLM) Toxicology Data Network (TOXNET®). It focuses on the toxicology of potentially hazardous chemicals. It is enhanced with information on human exposure, industrial hygiene, emergency handling procedures, environmental fate, regulatory requirements, and related areas. All data are referenced and derived from a core set of books, government documents, technical reports and selected primary journal literature. HSDB is peer-reviewed by the Scientific Review Panel (SRP), a committee of experts in the major subject areas within the data bank's scope. HSDB is organized into individual chemical records, and contains over 4500 such records.

The following is the HSDB summary of human health and environmental fate information:

HUMAN HEALTH EFFECTS:

HUMAN TOXICITY EXCERPTS:

The toxicity of strontium compounds depends on the anion. /Strontium/ [Seiler, H.G., H. Sigel and A. Sigel (eds.). Handbook on the Toxicity of Inorganic Compounds. New York, NY: Marcel Dekker, Inc. 1988. 633]**PEER REVIEWED**

ACCIDENTAL INGESTION MAY CAUSE GASTROINTESTINAL DISORDERS, PAINFUL CONTRACTIONS IN LIMBS ... /STRONTIUM AND COMPOUNDS/ [International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983. 2111]**PEER REVIEWED**

The hazard of (90)Sr is primarily that of internal contamination. In the body it is deposited mainly in the bones & due to its long biological half-life, it may result in beta-ray induced hemopoietic tissue lesions & malignant bone growth. /(90)Sr/ [International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983. 2112]**PEER REVIEWED**

This isotope /(90)Sr/ ... has been implicated as a causative agent in ... leukemia. /(90)Sr/ [National Research Council. Drinking Water & Health, Volume 4. Washington, DC: National Academy Press, 1981. 189]**PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE:

NIOSH (NOES Survey 1981-1989) has statistically estimated that 2,991 workers (35 of these are female) are potentially exposed to strontium in the US(1). Occupational exposure to strontium may occur through inhalation of this compound at workplaces where strontium is produced or used(SRC). The general population may be exposed to strontium via inhalation of ambient air and ingestion of drinking water and milk containing strontium(SRC). [(1) NIOSH; National Occupational Exposure Survey (NOES) (1983)]**PEER REVIEWED**

ENVIRONMENTAL FATE/EXPOSURE SUMMARY:

Strontium forms 0.02-0.03% of the earth's crust and is present in igneous rocks in amounts averaging 375 ppm. Of the naturally occurring strontium

compounds, only the minerals strontianite (strontium carbonate) and celestite (strontium sulfate) are of economic importance. Of the two, celestite occurs much more frequently in sedimentary deposits of sufficient size to make development of mining facilities attractive. Strontium is the fifth most abundant metallic ion in seawater, occurring in quantities of approximately 14 grams per metric ton. More than 80% of all strontium consumed in 1995 was used in ceramic and glass manufacture, primarily in televison faceplate glass (strontium carbonate/oxide) and ceramic ferrite magnets (strontium ferrite). Because of its brilliant red flame, strontium (in particular, strontium nitrate) is used in pyrotechnic devices for the military (tracer ammunition, military flares, marine distress signals) as well as non-military applications including warning devices and fireworks. In addition, strontium (strontium carbonate) is used to remove lead impurities during the electrolytic production of zinc, as an additive to corrosion resistant paint (strontium chromate), in toothpaste for temperature-sensitive teeth (strontium chloride), and in the manufacture of fluorescent lights (strontium phosphate). In addition, strontium-90 has been distributed worldwide by the fallout of nuclear explosions during the 1960's and the fallout of the Chernobyl, U.S.S.R., accident in 1986; most of the radioactive strontium was sorbed in top soil layers. The concn of strontium sorbed in 21 natural sediment-groundwater systems was determined to range from 9.2X10-7 to 1.04X10-4 mole/cu-dm; the main parameters governing strontium sorption were cation-ion exchange capacity of the sediment and ionic strength of the groundwater. The distribution coefficient, Kd, for strontium in a podsol forest soil was determined to be 140 l/kg in the top layer and 44 l/kg in the lower layer. Volatilization from soil surfaces will not be an important fate process. Strontium compounds are expected to exist primarily in the particulate-phase in the ambient atmosphere. Particulate-phase strontium may be physically removed from the air by wet or dry deposition. Volatilization of the ionic form of strontium from water surfaces will not occur. Bioconcentration of strontium in bluegill fish (Lepomis macrochirus) was determined to range from 1.4-1286.0 in fish collected from the Merced River and Salt Slough, CA. Occupational exposure to strontium may occur through inhalation of this compound at workplaces where strontium is produced or used. The general population may be exposed to strontium via inhalation of ambient air and ingestion of drinking water and milk containing strontium. (SRC) **PEER REVIEWED**

Lewis, R. 1994. *Sax's Dangerous Properties of Industrial Materials*. 8th ed. Van Nostrand Reinhold Company. New York.

STRONTIUM COMPOUNDS
DPIM: SMH500 Hazard Rating: 1
SAFETY PROFILE:
The strontium ion has a low order of toxicity. It is chemically and biologically similar to calcium. Strontium salicylate is the most toxic compound. The oxides and hydroxides are moderately caustic materials. Symptoms of acute toxicity are excessive salivation, vomiting, colic, and diarrhea, and possibly respiratory failure. The gastrointestinal absorption of soluble strontium ranges from 5 to 25%. Workers in strontium salt plants have reduced activity of choline esterase and acetylcholine. Drinking water with 13 mg Sr/L caused impaired tooth development in 1-year-old children. As with other compounds, the toxicity of a given compound may be a function of the anion. Compounds are highly dangerous if they contain the radioactive isotope 90Sr.
Updated: 08/27/90

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters, Hull, Québec, Canada.

The RATL (Reptile and Amphibian Toxicology Literature) database contains data extracted from the primary literature for amphibian and reptile ecotoxicology studies published up to and including 1997; there are some data from studies published in 1998 and 1999. As of September, 2000, there was approximately 2000 references in the database. Citations were gathered through searches of various literature databases, but these searches concentrated on the environmental pollution literature with the result that the bibliography cannot be considered exhaustive.

U.S. Environmental Protection Agency. 1996. Integrated risk information system. Office of Research and Development. Cincinnati, OH.

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RFD):

:

Substance Name -- Strontium
CASRN -- 7440-24-6

Last Revised -- 12/01/1996

I.A.1. ORAL RFD SUMMARY:

Critical Effect	Experimental Doses*	UF	MF	RfD
Rachitic bone	NOAEL: 0.19% Sr (as SrCO3) (190 mg Sr/kg/day)	300	1	6E-1 mg/kg/day

U.S. Environmental Protection Agency. 2002. Ecotox database: Lead. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN.

The ECOTOXicology database is a source for locating single chemical toxicity data for aquatic life, terrestrial plants and wildlife. ECOTOX integrates three toxicology effects databases: AQUIRE (aquatic life), PHYTOTOX (terrestrial plants), and TERRETOX (terrestrial wildlife). These databases were created by the U.S. EPA, Office of Research and Development (ORD), and the National Health and Environmental Effects Research Laboratory (NHEERL), Mid-Continent Ecology Division.

Scientific name, Common name	Endpoint	Effect	Trend -------- - Effect %	Media Type	Duration ---------- Exp Typ	Conc (ug/L)	Signif ------- Level	Response Site ------- BCF	Ref #
Oncorhynchus mykiss Rainbow trout, donaldson trout	LC10	MOR	INC --------	FW	4 dph ----------	T 49.0		------	14527

HHS Public Access

Author manuscript

Epidemiology. Author manuscript; available in PMC 2018 January 01.

Published in final edited form as:

Epidemiology. 2017 January ; 28(1): 77–85. doi:10.1097/EDE.0000000000000556.

Wildfire-specific Fine Particulate Matter and Risk of Hospital Admissions in Urban and Rural Counties

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Abstract

Background—The health impacts of wildfire smoke, including fine particles ($PM_{2.5}$), are not well understood and may differ from those of $PM_{2.5}$ from other sources due to differences in concentrations and chemical composition.

Methods—First, for the entire Western US (561 counties) for 2004–2009, we estimated daily $PM_{2.5}$ concentrations directly attributable to wildfires (wildfires-specific $PM_{2.5}$), using a global chemical transport model. Second, we defined *smoke wave* as ≥2 consecutive days with daily wildfire-specific $PM_{2.5}$>20μg/m^3, with sensitivity analysis considering 23μg/m^3, 28μg/m^3, and 37μg/m^3. Third, we estimated the risk of cardiovascular and respiratory hospital admissions associated with smoke waves for Medicare enrollees. We used a generalized linear mixed model to estimate the relative risk of hospital admissions on smoke wave days compared to matched comparison days without wildfire smoke.

Results—We estimated that about 46 million people of all ages were exposed to at least one smoke wave during 2004 to 2009 in the Western US. Of these, 5 million are Medicare enrollees (≥65y). We found a 7.2% (95% confidence interval: 0.25%, 15%) increase in risk of respiratory admissions during smoke wave days with high wildfire-specific $PM_{2.5}$ (>37μg/m^3) compared to matched non-smoke-wave days. We did not observe an association between smoke wave days with wildfire-$PM_{2.5}$≤37μg/m^3 and respiratory or cardiovascular admissions. Respiratory effects of wildfire-specific $PM_{2.5}$ may be stronger than that of $PM_{2.5}$ from other sources.

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Conflicts of interest: none to declare

Conclusion—Short-term exposure to wildfire-specific $PM_{2.5}$ was associated with risk of respiratory diseases in the elderly population in the Western US during severe smoke days.

Introduction

Wildfires are a growing concern, as climate change is anticipated to increase their frequency, intensity, and spreading speed[1]. Wildfires are known to cause substantial ecologic and economic burden, and the economic costs may be underestimated because they do not account for the potentially severe impact of air pollution from wildfires on human health[2]. Understanding the public health impact of wildfire smoke can inform intervention-focused policies to protect population health and promote more accurate estimates of the consequences of wildfires[3].

The Western US historically suffers from wildfires[4] due to large areas of forests, vegetation, and relatively arid weather. The burning of biomass can dramatically increase levels of toxic air pollutants, such as fine particles ($PM_{2.5}$)[5]. Numerous studies have demonstrated links between all-source particulate matter (PM) measured as total mass and health outcomes, especially for respiratory and cardiovascular diseases[3]. Many studies have indicated that $PM_{2.5}$ raises more human health concerns than coarse PM because the smaller particles penetrate the respiratory system more deeply[6].

The health effects of wildfire-emitted fine particles are not well understood. Wildfire smoke can increase ambient PM levels several times higher than that on days with no wildfire sources[3]. The size of fire-generated PM tends to be small, such as fine particles ($PM_{2.5}$)[7]. The composition of wildfire-generated $PM_{2.5}$ may be different from $PM_{2.5}$ from other sources, which in turn can affect toxicity[8,9]. Wildfires are episodic, making it especially challenging to link wildfire-specific air pollution with health.

We previously performed a literature review of the small number of studies on health impact of wildfire smoke on community populations. We found that the results on the effects of wildfires on hospital admissions were inconsistent, especially for cardiovascular diseases, in the Western US[3]. To date, most of the literature focused on a single fire episode and small population[e.g.10,11,12]. It is unknown whether the health impacts of wildfire-emitted $PM_{2.5}$ differ from those of $PM_{2.5}$ from other sources. As a result, research that investigates health impact from wildfires on a large geographical area and over a long time is needed.

The understanding of the health impact of wildfire-related air pollution is hindered by the challenge of estimating exposure to air pollution that can be specifically attributable to wildfires. Ambient monitors measure $PM_{2.5}$ concentration but cannot distinguish the proportion directly attributable to fires versus other sources. The majority of current wildfire-health studies used air monitoring data, which are limited in spatial (no monitors available in rural areas) and temporal resolution (generally measure every 3–6 days) and cannot isolate wildfire-specific pollution[3].

Our study aimed to address many of these challenges described above. Using a chemical transport model, we could fill in the spatial and temporal gaps of monitoring data and make source attributions of the modeled $PM_{2.5}$. We estimated daily 2004–2009 $PM_{2.5}$

concentrations specifically from wildfires for 561 Western US counties and linked them to daily numbers of Medicare admissions for respiratory and cardiovascular diseases. We applied statistical methods that have not been previously used in wildfire-health studies and estimated health impacts of wildfire-specific $PM_{2.5}$, incorporating rural populations into statistical analysis.

Methods

Study domain

The study domain is the Western US (lat: 31–49, lon: −101 to −125) (eFigure 1), where wildfires occur frequently[13]. The study region consists of 561 counties in 16 states.

Wildfire modeling

We employed wildfire simulations from the GEOS-Chem chemical transport model (v9-01-03) to generate daily wildfire-specific $PM_{2.5}$ levels for six years (2004–2009). GEOS-Chem is a global 3D atmospheric chemistry model driven by meteorology[14]. It has been used to understand the pollution impact of present-day fires[15,16] and to predict future wildfire-specific aerosols[1,17]. The modeling integrates meteorological data from Goddard Earth Observing System (GEOS-5) of the NASA Modeling and Assimilation Office and observed wildfire area burned based on the Global Fire Emissions Database (GFED3). GFED3 combines satellite observations of fire counts, area burned, and fuel load to produce gridded, daily maps of wildfire emissions[18,19].

The GEOS-Chem simulation model outputs used in this study are daily (24-hour-average), gridded surface $PM_{2.5}$ concentrations for fire seasons (May 1-Oct. 31) 2004–2009. The grid size is 0.5x0.67 degrees (approximately 50x75km) latitude-by-longitude. We generated estimates under two simulations: 1) the "all-source $PM_{2.5}$": total $PM_{2.5}$ levels from all sources including wildfires; and 2) "no-fire $PM_{2.5}$": $PM_{2.5}$ from all sources except the contribution from wildfires, by performing model simulations without wildfire emissions. Non-fire sources for $PM_{2.5}$ in the West include fossil fuel combustion from transportation, industry, and power plants[20,21]. The difference between outputs from these two simulations provides an estimate of the wildfire-specific $PM_{2.5}$ for each day and gridcell. We defined exposure based on daily wildfire-specific $PM_{2.5}$ estimates, which may differ from the actual locations of wildfires as smoke can travel large distances[22]. This model provided exposure estimates for all study subjects in the spatial domain, including those far from monitors. The results of GEOS-Chem simulations on particulate matter have been validated against observations[16,23]. We use ground-based or aircraft measurements, not satellite data, to validate the GEOS-Chem surface $PM_{2.5}$, including wildfire $PM_{2.5}$ (eAppendix Methods 2).

The modeled estimates of $PM_{2.5}$ from wildfires were spatially misaligned with health and weather data, with GEOS-Chem exposure data in a gridded form, health data at the county level, and weather data at the point level (i.e., monitor location). We converted daily grid-level wildfire-specific $PM_{2.5}$ and all-source $PM_{2.5}$ into daily county-level values using area-weighted averaging[24] (eAppendix Methods 3). We assumed that all persons residing in a given county have the same exposure to wildfire-specific $PM_{2.5}$ on a given day.

Hospital admissions data

The hospital admission data are based on billing records 2004–2009 from the Medicare Cohort Air Pollution Study (MCAPS)[25]. Ethical review was not required for this study. We included county-level data for all Medicare beneficiaries (US residents≥65y) enrolled in fee-for-service plan (70.0% of all Medicare beneficiaries) in 561 counties including rural and sparsely populated counties (eFigure 1). The Medicare data contain daily counts of cause-specific hospital admissions by county along with detailed information on date of admission, age category, sex, race, and daily total numbers of Medicare enrollees, representing the population at risk, in each combination of age category, sex and race. The hospital admissions counts can include repeated admissions.

We selected emergency hospital admissions for cardiovascular (CVD) and respiratory diseases as health outcomes. A visit coded as an emergency admission might not be admitted from an emergency room/department directly but the admission was emergency (admission type is emergency not elective). Previous studies connected these disease categories with total mass $PM_{2.5}$[e.g.25,26,27]. The ICD-9 codes of diagnoses are in eAppendix Methods 1.

Air monitoring data and weather data

Daily total $PM_{2.5}$ measurements from the monitoring data, reflecting real-world $PM_{2.5}$ from all sources, were used to calibrate the total GEOS-Chem $PM_{2.5}$ results ("all-source" $PM_{2.5}$). The air monitoring data were acquired from EPA AirData (http://aqsdr1.epa.gov/aqsweb/aqstmp/airdata/download_files.html#Daily). When a county had measurements from multiple monitoring sites on a given day, we averaged all monitor measurements to estimate the county's total $PM_{2.5}$ level on that day.

Weather information was used in statistical analysis since temperature may confound health impact of air pollution[28]. Daily county-level weather data, including temperature and dew point temperature, were obtained from the National Centers for Environmental Information of National Oceanic and Atmospheric Administration.

Calibration

As in other chemical transport models, the GEOS-Chem $PM_{2.5}$ estimates were biased low during extreme events, reflecting the challenge in capturing smoke plumes on fine spatial scales[e.g.23]. To address this bias, we calibrated the daily, county-level 2004–2009 modeled total $PM_{2.5}$ estimates ("all-source" $PM_{2.5}$) in all 561 counties) with the county-level total $PM_{2.5}$ data from air monitors, by matching the quantile functions of the two datasets. This approach scales the distribution of modeled $PM_{2.5}$ data to more closely resemble the distribution of the monitored data[29]. This method maintains the ordering of $PM_{2.5}$ in the original (modeled) data (e.g., any day above the 98^{th} percentile of $PM_{2.5}$ in the original modeled data is above the 98^{th} percentile in the calibrated data). This calibration process results in empirical cumulative distribution functions for the simulated total $PM_{2.5}$ that matches that of the observed $PM_{2.5}$. Hence the overall proportion of $PM_{2.5}$ that comes from wildfire smoke is identical in the original and calibrated data. We calibrated the daily *total* modeled $PM_{2.5}$ using county-average monitoring data, calculated the proportions of total

modeled $PM_{2.5}$ contributed by modeled wildfire-specific $PM_{2.5}$ on each day, and then multiplied the calibrated total modeled $PM_{2.5}$ with the proportions to obtain the calibrated wildfire-specific $PM_{2.5}$. Results from the calibration process are shown in eTable 1 and eFigure 2.

Definition of a Smoke Wave

Traditionally, the short-term effects of $PM_{2.5}$ have been investigated by estimating the association between day-to-day variations in pollutant levels with the day-to-day variation in health outcome rates. For example, some researchers applied time-series analysis to associate daily ambient air pollution exposures with daily hospital admission rates in large multi-city studies[25–27]. However, the frequency distribution of wildfire-specific $PM_{2.5}$ data differs from that of traditional ambient levels of total $PM_{2.5}$. Absent a wildfire smoke event, the wildfire-specific $PM_{2.5}$ level is near zero. Among all the days with an estimated wildfire-specific $PM_{2.5}$ levels, only 28.1% have values >1μg/m^3 but levels can reach >200μg/m^3 during the wildfire days. To estimate health effects associated with rare but extreme episodes of wildfire-specific $PM_{2.5}$ we introduced a new modeling approach that to our knowledge has not previously been used in the wildfire–health literature.

Specifically, we first introduce the concept of “smoke wave”. The concept of smoke wave allows us to capture periods with high concentration, sporadic, and short-lived characteristics of wildfire $PM_{2.5}$. We define a smoke wave as at least two consecutive days with daily calibrated wildfire-specific $PM_{2.5}$>20μg/m^3 (near the 98th percentile of all county-days across all 561 counties). This definition is based on daily wildfire-specific $PM_{2.5}$ levels above a designated threshold and the daily levels in all days in a smoke wave must exceed the threshold. We conducted sensitivity analyses that varied the definition of smoke wave with respect to duration and intensity; for example, we also defined smoke wave as at least *one* day with daily calibrated wildfire-specific $PM_{2.5}$>20μg/m^3 (“single-day smoke-waves”). Among all smoke-wave days, we investigated whether health impact differs on smoke wave days with different intensity and considered intensity thresholds of 23μg/m^3, 28μg/m^3, and 37μg/m^3 corresponding to the 98.5th, 99th, and 99.5th quantile of all county-days across all 561 counties, respectively. We investigated whether timing within smoke waves (during the first 2 days, 3rd to 7th day, and 8th or later day of a smoke wave) affects health risks, i.e. whether the health risks on an earlier day in a smoke wave differed from those for a later day in a smoke wave. We also conducted sensitivity analysis on counties with fee-for-service enrollment ≥75% among Medicare beneficiaries.

Statistical modeling

We conducted a matched analysis to compare the hospital admission rates (number of admissions/number of Medicare fee-for-service enrollees) on smoke-wave days (exposure) and matched non-smoke-wave days (no-exposure to high wildfire-specific $PM_{2.5}$). We chose to conduct matched analysis because the wildfire-specific $PM_{2.5}$ exposure is episodic and occurs infrequently (1.63% days were smoke wave days among all county-days). Each smoke-wave day was matched with up to three non-smoke-wave days in the same county. Smoke-wave days in counties with many smoke-wave days may be matched with fewer than three non-smoke-wave days when we were not able to find three suitable no-smoke-wave

days. Among the total 10080 smoke-wave days in all counties in 6 years, 9184 were each matched with 3 non-smoke-wave days, 697 with 2 non-smoke-wave days, and 199 with 1 non-smoke-wave days. We considered non-smoke-wave days to be eligible match days if they are: 1) within the window of 7 calendar days before or 7 days after the smoke-wave day but primarily in a different year (before or after the year of the smoke-wave day) and 2) are separated from any other smoke-wave day by at least 2 days. Among all eligible days meeting the matching criteria for a non-smoke-wave day, we selected the matched non-smoke-wave days at random. By matching based on a 15-day period primarily in a different year, we accounted for larger seasonal trends such as the greater propensity for wildfires to occur during the hotter and drier months. We assessed the difference in daily temperature, daily dew point temperature, and non-fire $PM_{2.5}$ for exposure (smoke-wave) days and no-exposure (non-smoke-wave) days. All statistical analyses were conducted in R v2.15.0.

Matching reduces the effects of confounding such as from seasonal trend[30]. We controlled for seasonal factors by 1) including a fixed effect of study year; 2) controlling for daily temperature; and 3) using a matched approach to ensure the same seasonality of smoke-wave days and matched non-smoke-wave days. The matching approach guarantees that the smoke wave and non-smoke-wave days have the same distribution across season (eTable 2), and hence controls by design for confounding by seasonal trends. We also conducted sensitivity analysis with the statistical model not adjusting for modeled non-fire $PM_{2.5}$ levels.

We investigated the Relative Risk (RR) of hospital admissions on the same day as a smoke wave (lag 0). We fitted a log-linear (Poisson) mixed effects regression model separately for each disease (cardiovascular or respiratory diseases) for smoke wave days and matched non-smoke-wave days across all 561 counties (details in eAppendix Methods 4). Similar statistical models have been applied in previous epidemiologic studies[31].

Results

Wildfire $PM_{2.5}$ characteristics

The frequency distribution of $PM_{2.5}$ levels from wildfire sources (calibrated) differs from that of $PM_{2.5}$ from non-fire sources. Levels of wildfire-specific $PM_{2.5}$ are highly skewed, with about 72% of daily county-level calibrated wildfire-specific $PM_{2.5}<1\mu g/m^3$. Wildfire-specific $PM_{2.5}$ has lower mean and median, but higher extremes, compared with $PM_{2.5}$ from non-fire sources (Table 1). The time-series pattern of wildfire-specific $PM_{2.5}$ is mostly zero with occasional high peaks for short periods.

Smoke wave characteristics

Based on our definition of a smoke wave (at least two consecutive days with wildfire-$PM_{2.5}>20\mu g/m^3$), about 66% of Western US counties (369 of 561) experienced at least one smoke wave during the 6-year period. Among the 369 counties with at least one smoke wave, on average a county had 4.6 smoke-wave days/year (Table 2). We found that the dates and locations of smoke wave days generally matched well with MODIS records of large wildfires (eFigure 4).

The number of smoke-wave days experienced by counties is spatially heterogeneous. Coastal California and central Idaho had the highest frequency of smoke-wave days (>10 smoke wave days/year) (Figure 1). The average wildfire-$PM_{2.5}$ concentration during each smoke wave day was lower during the first two days of smoke waves and gradually increased over time during a smoke wave (eFigure 3). The median length of a smoke wave was 3 days (ranged 2 to 58). Temperatures during smoke wave days did not differ largely based on the smoke wave day's intensity (eTable 3(a)) or smoke wave length (eTable 3(b)).

Hospital admission summary statistics

The study population for the 561 counties during the study timeframe (2004–2009) includes on average about 5 million Medicare enrollees per day. This population had a total of 832,244 cardiovascular admissions and 245,926 respiratory admissions during the study timeframe. Within the study timeframe, 369 counties had at least one smoke wave. For these counties, there were 648,789 cardiovascular admissions and 191,095 respiratory admissions. Counties that experienced a smoke wave had, on average, lower rates of hospital admissions than counties with no smoke wave (Table 3). There are 3,844,414 people exposed to at least one smoke wave, and 1,114,513 with no exposure to smoke waves.

Association between wildfire $PM_{2.5}$ and hospital admissions

Overall, smoke waves were not associated with increased rates of cardiovascular hospital admissions. The overall association with cardiovascular admissions on a smoke-wave day compared to a non-smoke-wave day was −0.74% (95% CI: −3.1%, 1.65%) (RR=0.99). The overall association with respiratory hospital admissions on a smoke wave day compared to a non-smoke-wave day was 2.3% (95% CI: −2.2%, 7.0%) (RR=1.0).

Smoke wave days with different intensity (level of wildfire $PM_{2.5}$) and the various days within the smoke waves exhibited indication of trends of different health effects. Central estimates for respiratory admissions showed an increasing trend as smoke wave day intensity increases (Figure 2 (b)). Smoke wave days with intensity >37μg/m^3 (99.5^{th} quantile) were associated with a 7.2% increase in respiratory admissions by 7.2% (95% CI: 0.25%, 15%) compared to non-smoke-wave days. Therefore, more intense smoke wave days are estimated to have higher health impacts on respiratory diseases for the study population. This association is robust to no inclusion of a variable for non-fire $PM_{2.5}$ levels in the model (results not shown). Results on single-day smoke waves and counties with fee-for-service enrollment>75% are summarized in eAppendix Results 1 and 2.

Central estimates for CVD admissions tend to be highest during the first two days of a smoke wave, and decreasing over the later days within a smoke wave (Figure 3(a)). Respiratory admissions exhibit an opposite trend, with higher estimate estimate in later days of the smoke wave (Figure 3(b)). For each types of admission, effect estimates based on timing within a smoke wave were imprecise.

Discussion

Our systematic assessment indicates an association between respiratory admissions and intense smoke wave days, with daily wildfire-specific $PM_{2.5}$ levels >37μg/m^3. Single-day

smoke waves have a potentially more certain positive association with respiratory admission rates, possibly due to larger sample sizes and the acute response of respiratory diseases.

To our knowledge this is the first study to use wildfire-specific data to analyze the health impact of wildfire-specific $PM_{2.5}$ over multiple years at a large geographical scale. Key contributions of this study include: 1) estimation of exposure to $PM_{2.5}$ specifically from wildfires; 2) ability to estimate exposure to wildfire $PM_{2.5}$ every county with and without air monitors, therefore expanding the study populations to include persons that live far from $PM_{2.5}$ monitoring stations; and 3) application of statistical models that estimate percent increases in hospital admission by matching smoke wave days to non-smoke-wave days.

Although previous literature on the association between wildfire smoke and health is limited, several studies have made important contributions. The majority of such studies used air monitor measurements, which cannot identify pollution specifically from wildfires with current technology, and studied a single wildfire episode and one or a small number of communities[3]. A few studies compared air pollution exposure (from all sources) during wildfires to the periods or locations with no fire[e.g.11,32,33]. Our study results for respiratory diseases are consistent with those found in most of the previous literature[e.g.34,35], in that wildfire smoke was found to be associated with respiratory diseases. Association between wildfire smoke and cardiovascular morbidities was found in five US studies that each examined a single local wildfire episode[3], but our multi-state, multi-year study did not provide evidence for such association.

Previous studies have demonstrated that the chemical composition of $PM_{2.5}$, which is related to source, can result in different effect estimates for human health[9,36,37]. Thus, estimates from wildfire $PM_{2.5}$ may differ from those from $PM_{2.5}$ from other sources, such as transportation or industry. Earlier studies examined the association between risk of hospital admission and levels of $PM_{2.5}$ from all sources (i.e., $PM_{2.5}$ total mass) (e.g., change of risk of hospital admission for Medicare enrollees per 10μg/m^3 increase in $PM_{2.5}$ in the Western US[25,26,38]). As we compared the health risk among smoke wave days with that of non-smoke-wave days, rather than by a specific increment of $PM_{2.5}$, direct comparisons of results is challenging. Further, these studies focused on urban counties with high populations, whereas our study included rural populations in the analysis as well. Still, a general comparison can give some indication of whether $PM_{2.5}$ from wildfire smoke is more or less harmful than $PM_{2.5}$ total mass.

For Medicare cardiovascular admissions, one study estimated an increased risk of 0.53% (95% posterior interval: 0.00%, 1.05%) per 10μg/m^3 $PM_{2.5}$ total mass (from all sources) for 25 urban counties in the Southwest US , and 0.74% (-1.74, 3.29%) for 9 urban counties in the Northwest[26]. Our results did not indicate an association between wildfire $PM_{2.5}$ and risk of cardiovascular admissions.

For respiratory hospital admissions, we estimated an increase of 7.2% (0.25%, 15%) comparing smoke-wave days with wildfire-$PM_{2.5}$>37μg/m^3 to non-smoke-wave days with wildfire-specific $PM_{2.5}$<20μg/m^3, which corresponds to an average difference of 29.6μg/m^3 in those two groups of days. The earlier study identified associations between $PM_{2.5}$ total

mass and respiratory admissions for the Medicare population in the Southwest at lag 2 days at 0.94% (0.22%, 1.67%) per 10 μg/m^3 [26], which corresponds to an increased risk of 2.8% (0.64, 5.0%) per 29.6μg/m^3. Therefore, our estimates of respiratory admissions risks indicate that wildfire-specific $PM_{2.5}$ from intense smoke waves are associated with more harm than $PM_{2.5}$ from other sources for the elderly in the Western US. Further research is needed to investigate the relative toxicity of $PM_{2.5}$ from wildfire smoke with that of other sources.

Our approaches for assessing pollutant exposure and estimating health risks address key challenges in studying the health impact of wildfire-specific pollutant. The GEOS-Chem model provided a new approach to distinguish wildfire-specific $PM_{2.5}$ from $PM_{2.5}$ from other sources. The fire scheme in the simulation can explain up to 60% of the observed variance of area burned in the Western US, and is ecosystem dependent[17]. This method also improves the spatial and temporal resolution of exposure estimates for air pollution. Unlike air monitoring data that generally measure $PM_{2.5}$ concentrations every 3–6 days in urban areas, GEOS-Chem estimates concentrations for every day and covers the entire study area. Our smoke-wave methods provide an approach suitable for the study of highly-skewed air pollution data and enable identification and investigation of pollution episodes with high source-specific pollutant concentrations. Matched analysis can reduce the confounding effect of seasonality and county-specific effects. These methods can be applied to future studies investigating other pollution events and populations.

Limitations of our study include potential spatial misalignment between the exposure estimates (gridded estimates) and health data (county). Our study population was restricted to Medicare fee-for-service enrollees, a sample of elderly persons. Our smoke wave approach does not fully capture the dose–response relationship, cause-specific health outcomes, etc. that could be investigated in future studies. The GFED emissions applied to GEOS-Chem contribute uncertainty to the modeled estimates of wildfires-specific $PM_{2.5}$. The GFED3 data may underestimate fire contributions to background $PM_{2.5}$ because of the omission of small fires[39] and the biases in the modeled fuel consumption. GFED3 relies on satellite observations of active fire counts and area burned, and may have difficulty discerning such phenomena, especially on cloudy days[40]. Another limitation arises as EPA monitors generally measure $PM_{2.5}$ values every 3–6 days and are located in populated areas. Given a large number of days with monitoring measurements for calibration, we assumed that the systematic sampling of EPA monitors generate measurements with mean and standard deviation representing the full time-series of real-world $PM_{2.5}$. While it would be ideal to have the full continuous measure we believe that calibration using this discrete sample of the continuous measure is the best possible alternative in using the available data. While our exposure estimates are advances over methods that do not isolate the air pollution from wildfires specifically, additional work could address these limitations. We choose not to a priori identify lags in this study as little is known about how wildfire-specific $PM_{2.5}$ affects human health. Most of the wildfire-health literature to date has investigated effects of lag 0 or short lags (<5 days)[3]. Future studies can explore the lagged effect of wildfire-specific air pollution.

Our findings indicate that wildfires are associated with increased risk of admissions for respiratory diseases for the elderly population during severe wildfire episodes. As climate

change is anticipated to increase the frequency and intensity of wildfires[1], the health burden from wildfire-specific pollutants may increase in the future. With improvement of atmospheric modeling, future studies can estimate daily wildfire-specific $PM_{2.5}$ at a finer spatial resolution. Future studies can also investigate vulnerability to wildfire smoke, health impact of different species of wildfire-specific $PM_{2.5}$, the economic consequence of the health burden from wildfire smoke, combined effect of wildfire smoke and other air pollutants, and estimated health burden in the future under climate change.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Sources of financial support:

NIH/NIEHS R21 ES022585-01

NIH R01 ES019560

NIH R01 ES024332

EPA RD 83479801

NIH R21 ES021427

NIEHS R21 ES020152

Yale Institute of Biospheric Studies Doctoral Dissertation Improvement Grant

This publication was developed under Assistance Agreement No. 83587101, awarded by the U.S. Environmental Protection Agency to Yale University. It has not been formally reviewed by EPA. The views expressed in this document are solely those of the authors and do not necessarily reflect those of the Agency. EPA does not endorse any products or commercial services mentioned in this publication.

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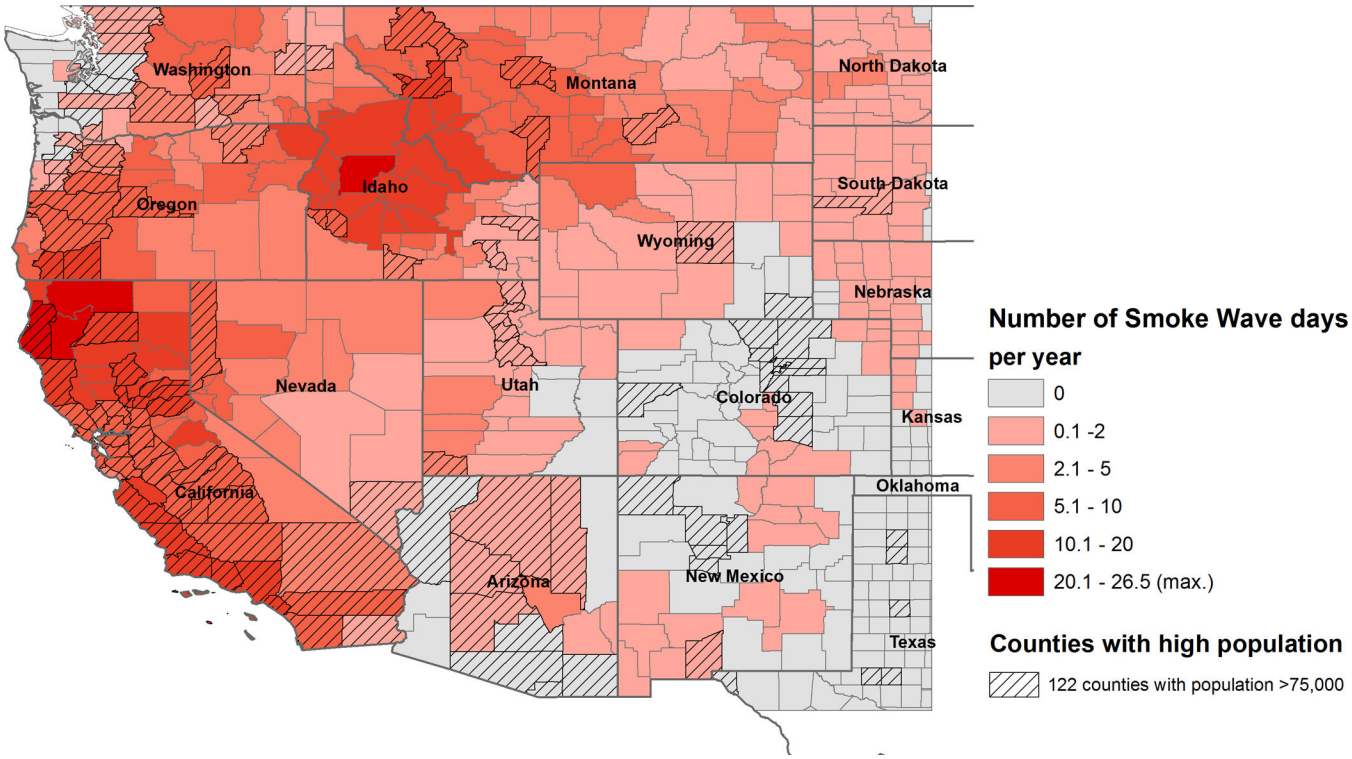

Figure 1.

Average number of Smoke Wave days/year for 561 Western US counties during 2004–2009. Hashed counties have population >75,000 in the 2010 Census.

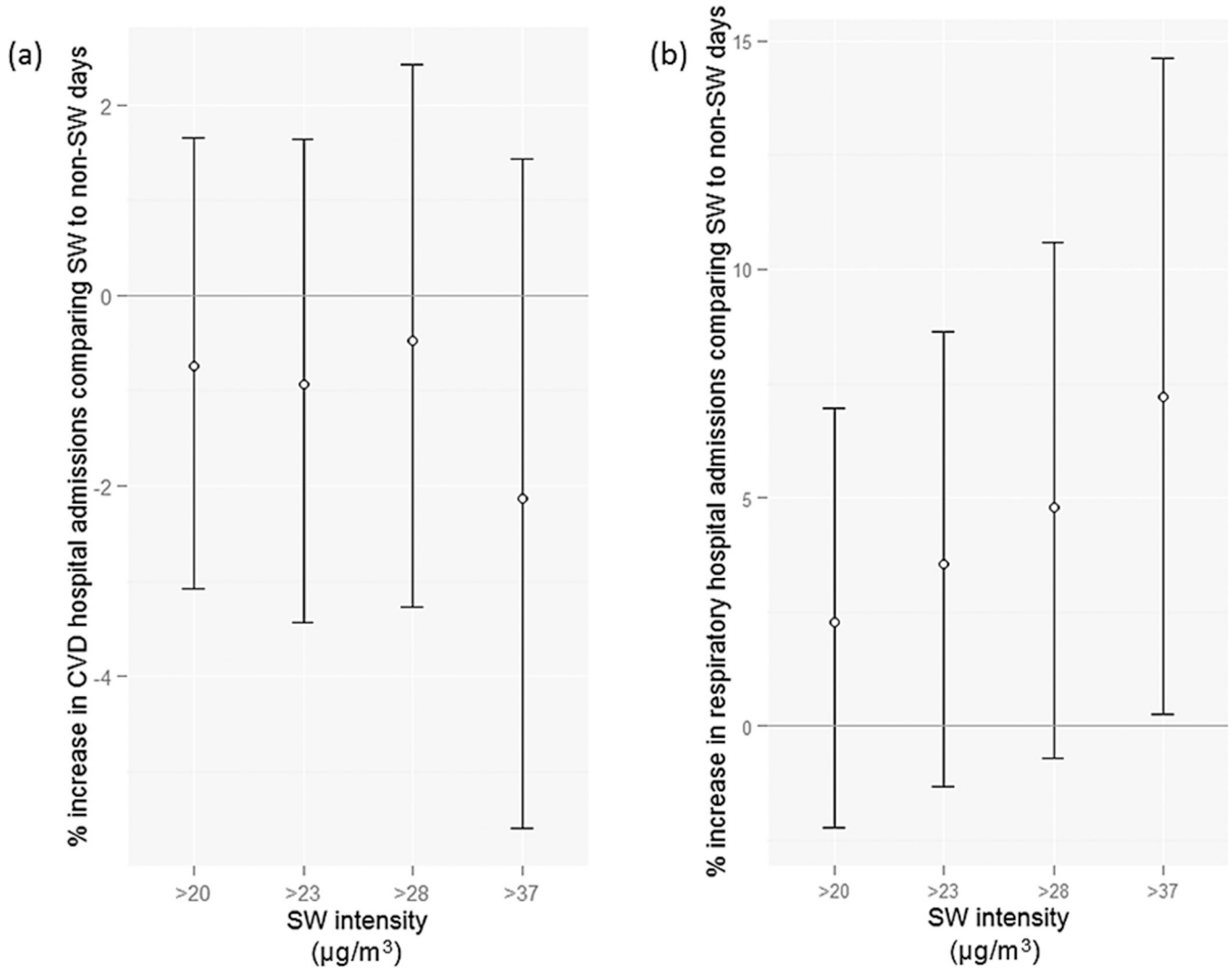

Figure 2.

Associations between hospital admissions and exposure to smoke-wave (SW) days (compared to non-smoke-wave days) for (a) cardiovascular disease and (b) respiratory disease, by different intensity (level of wildfire-specific $PM_{2.5}$) definitions of a smoke wave.

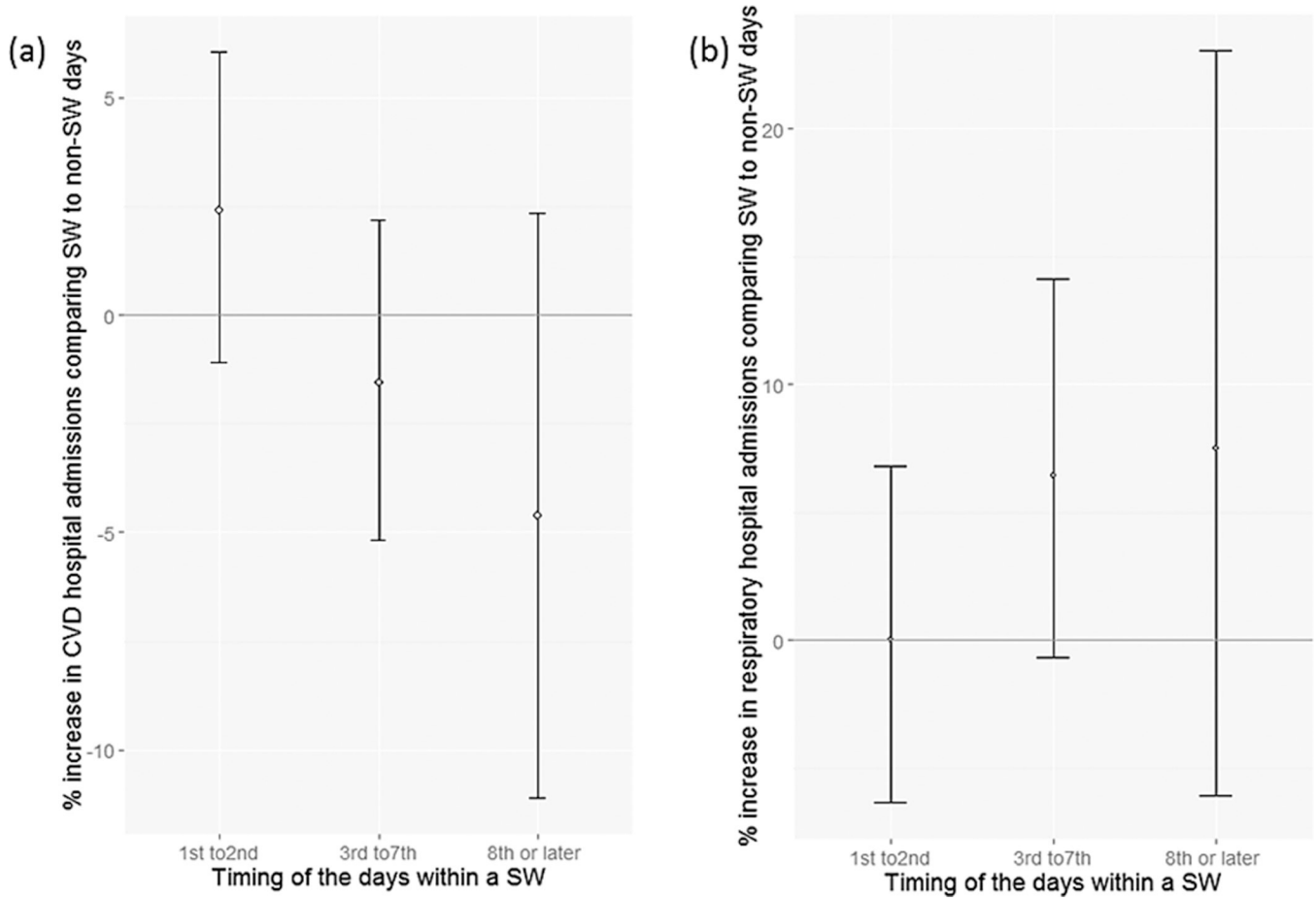

Figure 3.

Associations between hospital admissions and exposure to smoke-wave (SW) days (compared to non-smoke-wave days) for (a) cardiovascular disease and (b) respiratory disease, by timing of the days within a smoke wave.

Table 1

Summary statistics for daily GEOS-Chem $PM_{2.5}$ concentrations (calibrated) from wildfire sources and non-fire sources in 561 western US counties ($\mu g/m^3$) during the wildfire season (May 1– Oct. 31), 2004–2009.

	Minimum	25th Percentile	Median	Mean	75th Percentile	Maximum
$PM_{2.5}$ from wildfires	0	0.09	0.3	2.0	1.2	242
$PM_{2.5}$ from non-fire sources	0	4.4	6.2	7.0	8.7	45.1

Epidemiology. Author manuscript; available in PMC 2018 January 01.

Table 2

Summary statistics for smoke waves (SW, defined as at least two consecutive days with wildfire-specific $PM_{2.5}$ >20μg/m^3) for the 369 Western US counties that experienced smoke waves during 2004–2009.

SW characteristics	Average (Standard Deviation)	Median	Minimum	Maximum
No. SW days /year [a]	4.6 (4.9)	2.5	0.33	26.5
No. SW events / year [a]	1.0 (0.8)	0.83	0.17	3.8
SW intensity (μg/m^3) [b]	29.3 (6.4)	28.1	20.1	70.0
SW length (days) [b]	4.4 (4.7)	3	2	58

[a] Statistics based on the 369 county-average values.

[b] Statistics based on all SW-level values across all SWs in the 369 counties.

Table 3

County-level hospital admission per 100,000 Medicare enrollees per day (2004–2009)

		Minimum	25th percentile	Median	Mean	75th percentile	Maximum
561 counties	Cardiovascular disease	1.59	8.18	11.5	12.2	15.0	43.7
	Respiratory	0	1.81	3.33	3.59	4.87	17.1
369 counties with smoke waves	Cardiovascular disease	1.59	7.87	10.7	11.2	13.7	39.7
	Respiratory	0	1.63	3.07	3.25	4.52	11.7
192 counties with no smoke waves	Cardiovascular disease	4.88	9.03	13.5	14.1	17.4	43.7
	Respiratory	0	2.43	3.91	4.25	5.74	17.8

American Journal of Epidemiology

Vol. 186, No. 6
DOI: 10.1093/aje/kwx141
Advance Access publication:
August 10, 2017

Original Contribution

Who Among the Elderly Is Most Vulnerable to Exposure to and Health Risks of Fine Particulate Matter From Wildfire Smoke?

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Initially submitted May 10, 2016; accepted for publication November 7, 2016.

Wildfires burn more than 7 million acres in the United States annually, according to the US Forest Service. Little is known about which subpopulations are more vulnerable to health risks from wildfire smoke, including those associated with fine particulate matter. We estimated exposure to fine particles specifically from wildfires, as well as the associations between the presence of wildfire-specific fine particles and the amount of hospital admissions for respiratory causes among subpopulations older than 65 years of age in the western United States (2004–2009). Compared with other populations, higher fractions of persons who were black, lived in urban counties, and lived in California were exposed to more than 1 smoke wave (high-pollution episodes from wildfire smoke). The risks of respiratory admissions on smoke-wave days compared with non–smoke-wave days increased 10.4% (95% confidence interval: 1.9, 19.6) for women and 21.7% (95% confidence interval: 0.4, 47.3) for blacks. Our findings suggest that increased risks of respiratory admissions from wildfire smoke was significantly higher for women than for men (10.4% vs. 3.7%), blacks than whites (21.7% vs. 6.9%), and, although associations were not statistically different, people in lower-education counties than higher-educated counties (12.7% vs. 6.1%). Our study raised important environmental justice issues that can inform public health programs and wildfire management. As climate change increases the frequency and intensity of wildfires, evidence on vulnerable subpopulations can inform disaster preparedness and the understanding of climate change consequences.

air pollution; health; $PM_{2.5}$; respiratory outcomes; vulnerability; wildfire smoke

Abbreviations: $PM_{2.5}$, fine particulate mater with aerodynamic diameter greater than 2.5 μm; SES, socioeconomic status.

Climate change is anticipated to increase the frequency, intensity, and spreading speed of wildfires. In addition to property damage and expenditures on fire suppression and recovery, wildfire smoke dramatically worsens air pollution, especially by increasing levels of fine particulate mater with aerodynamic diameter greater than 2.5 μm ($PM_{2.5}$) (1, 2). Wildfire smoke can increase $PM_{2.5}$ levels to several times those seen during nonwildfire periods (3). Because wildfire-specific $PM_{2.5}$ might have chemical compositions and/or uniquely high concentrations of specific chemicals that differ from those of $PM_{2.5}$ from other sources, it could impose a different health-response function on exposed populations.

Some subpopulations may be particularly vulnerable to health risks from wildfire smoke because of biophysical and/or socioeconomic conditions (4). Older persons may have degraded immune systems (3, 5–9). Socioeconomic status (SES) or other demographic characteristics can be associated with exposure or the ability to adapt to environmental exposure (4, 10). Sex or race may be associated with occupation or activity patterns that lead to different environmental exposures (11). There have been few studies in which investigators have assessed vulnerability from wildfire pollution, and the results were inconsistent (3).

METHODS

We estimated $PM_{2.5}$ concentrations specifically from wildfires and from nonfire sources (2004–2009) in 561 counties

in the western United States (Web Figure 1, available at https://academic.oup.com/aje) by using the GEOS-Chem, version v9-01-03 (http://acmg.seas.harvard.edu/geos/) global chemical transport model and utilizing reports of daily emissions from the Global Fire Emissions Database (12). Details on GEOS-Chem modeling and validation can be found elsewhere (13). We converted GEOS-Chem's gridded estimation (resolution ≈0.5° latitude × 0.67° longitude) to county-level estimates by using area-weighted averaging. Wildfire-specific $PM_{2.5}$ estimates were calibrated with monitoring data (13, 14).

In our previous study, we found that hospital admissions for respiratory problems among persons older than 65 years of age were 7.2% (95% confidence interval: 0.25, 14.6) higher on smoke-wave days than on non–smoke-wave days, when a smoke wave was defined as a period with more than 2 consecutive days with daily calibrated wildfire-specific $PM_{2.5}$ concentrations greater than 37 $\mu g/m^3$ (13). Using Medicare claims data, we calculated total respiratory admissions as the sum of admissions for primary disease discharge codes of chronic obstructive pulmonary disease and respiratory tract infections (*International Classification of Diseases, Ninth Revision*, codes 490–492, 464–466, 480–487).

We classified each day in each county as a smoke-wave or non–smoke-wave day. Each smoke-wave day was matched with up to 3 non–smoke-wave days in the same county that occurred within the 7-day window before and after the smoke-wave day primarily in a different year and were separated from any other smoke-wave day by more than 2 days. We selected control days at random from among eligible control days for a given smoke-wave day to avoid a systematic pattern as to whether the matched days occurred before or after the smoke-wave day. When 3 eligible control days were not available, we used 1 or 2 days.

We categorized subpopulations by the following: 1) individual characteristics, including age (65–74, 75–84, or >85 years), sex, and race (black, white, or other); and 2) county characteristics, including education (<20% of elderly with bachelor's degree vs. ≥20% of elderly with bachelor's degree) (15), poverty rate (<10%, 10%–15%, or >15%) (16), urbanicity, and region (Web Appendix 1, Web Figure 1).

We assessed vulnerability for each subpopulation by the following: 1) exposure to smoke waves (2004–2009): proportion of exposed to more than 1 smoke wave, average number of smoke-wave days, and average intensity of smoke waves (Web Appendix 2); and 2) health risks from smoke waves: increase in respiratory admissions associated with smoke waves.

To estimate the associations of smoke-wave days with hospital admissions for respiratory conditions stratified by individual-level characteristics, we fitted a log-linear Poisson mixed-effects regression model for respiratory admissions across all 561 counties, with a term for the interaction between an indicator for smoke-wave day and an indicator for the specific subpopulation that controlled for nonfire $PM_{2.5}$ concentration, temperature, age, sex, race, and study year (Web Appendix 3). To estimate associations of smoke-wave exposure with hospital admissions for respiratory causes categorized by community-level characteristics, we stratified counties by community characteristics and fitted separate models for each stratum (Web Appendix 4). We then compared the associations in different subpopulations (17).

RESULTS

The total number of Medicare enrollees in the western United States from 2004 to 2009 was approximately 5 million (Table 1). Admission rates for respiratory illness were highest among persons in the oldest age group, among blacks, in counties with a poverty rate above 15%, and in counties in which

Table 1. Categorization of Subpopulation Based on Individual Characteristics, Population in These Groups, and Population Exposed to at Least 1 Smoke Wave in Each Subpopulation in Western US Counties, 2004–2009

Individual Characteristic	Average Population		Average No. Exposed to >1 Smoke Wave	% Subpopulation Exposed to >1 Smoke Wave[b]	Average No. of Smoke-Wave Days per Year	Average Smoke Wave Intensity, $\mu g/m^3$
	No.[a]	% of Total Population				
Age, years						
65–74	2,700,367	54.5	1,604,366	59.4	1.51	44.06
75–84	1,643,695	33.1	966,542	58.8	1.56	44.10
>84	614,865	12.4	391,847	63.7	1.62	44.13
Sex						
Female	2,743,008	55.3	1,641,338	59.8	1.55	44.11
Male	2,215,919	44.7	1,321,416	59.6	1.52	44.06
Race						
Black	157,934	3.2	115,933	73.4	1.77	43.95
White	4,110,641	82.9	2,302,364	56.0	1.47	44.01
Other	690,352	13.9	544,457	78.9	1.91	44.45

[a] The average Medicare population in each subpopulation during the study period. The Medicare population changes over time; values here represent the population size on an average day during the study period.

[b] Interpretation example: Of people 65–74 years of age, 59.4% were exposed to smoke waves during the study period compared with 58.8% of people 75–84 years of age.

less than 20% of the population had bachelor's degrees (Web Table 1). Some SES characteristics were correlated (Web Table 2). For example, counties with high fractions of black persons were more likely to be urban.

Smoke-wave exposure differed by subpopulations (Table 1). Approximately 73.4% of blacks were exposed to more than 1 smoke wave, compared with 56.0% of whites. Nearly all participants in California (99.2%) were exposed to more than 1 smoke wave, compared with 7.49% in the southwestern United States. Larger proportions of participants in urban counties (64.8% vs. 47.1% of participants in less-urban/rural counties) and more educated counties (63.3% vs. 49.8% for participants in less-educated counties) were exposed to more than 1 smoke wave (Web Table 3). The proportion exposed decreased as poverty decreased: The proportions were 61.5%, 56.2%, and 55.9% for persons living in counties with more than 15%, 10%–15%, and less than 10% of the population in poverty, respectively.

California had 4.08 smoke-wave days per year, the highest among the 4 regions (Web Table 3). The poorest counties (>15% people living in poverty) had the highest number of smoke-wave days/year (2.70 days per year on average compared with 1.28 days per year for counties with <10% people living in poverty).

We also assessed the intensity of smoke waves by measuring the average wildfire-specific $PM_{2.5}$ levels on smoke-wave days. Smoke waves in the Northern Rocky Mountains were the most intense (mean $PM_{2.5}$ concentration = 47.83 $\mu g/m^3$) compared with those elsewhere (in the Southwest, mean = 40.60 $\mu g/m^3$; Web Table 3). Although a smaller fraction of people in less-urban/rural counties was exposed to more than 1 smoke wave (47.1%) than in urban counties (64.8%), smoke-wave intensity was higher in less-urban/rural counties (mean = 47.01 $\mu g/m^3$) than in urban counties (mean = 43.85 $\mu g/m^3$). Smoke-wave intensity did not differ much by individual-level characteristics.

Results provided suggestive evidence that women (compared with men) and blacks (compared with whites or persons of other races) had higher risks of hospital admissions for respiratory illness associated with exposure to smoke waves (Table 2). The central estimate of relative risk of respiratory admissions on smoke-wave days compared with non–smoke-wave days was higher for people living in less-educated counties (for counties in which <20% of the elderly had a bachelor's degree, relative risk = 1.13, 95% confidence interval: 0.97, 1.31) than that of people living in more-educated counties (for counties in which ≥20% of the elderly had a bachelor's degree, relative = 1.06, 95% confidence interval: 0.98, 1.14). No subpopulation had a health risk that was significantly different from those of its counterparts in the respective characteristic categories.

DISCUSSION

Our study filled in important scientific gaps on wildfire and population health and addressed many challenges in characterizing vulnerability to wildfire smoke, such as the typically small sample sizes of subpopulations, difficulty in determining exposure to wildfire smoke, and the low frequency and geographical coverage of monitor measurements. In most previous studies on vulnerability to wildfire smoke, researchers investigated small numbers of communities exposed to single fire episodes. In our multiyear, multistate study, we considered a population of approximately 5 million and incorporated both urban and rural counties, which allowed us to estimate health risks by subpopulation and region. The present research is the first wildfire vulnerability study in which daily source-specific exposure estimates that distinguish wildfire-specific $PM_{2.5}$ from $PM_{2.5}$ from other sources in all western US counties were used. Instead of using "hot spots" from satellite images or rough start/end days of recorded wildfires, we utilized a new approach to define smoke days by using source-specific $PM_{2.5}$.

The present study has limitations. Although our study is the largest wildfire vulnerability study to date, we only focused on the elderly population. Previous research has indicated that preexisting medical conditions could be related to a vulnerability to the association between air pollution and health (18, 19), which could be investigated in relation to wildfire smoke in future studies. In future work, researchers can also investigate wildfire vulnerability with other ages or individual-level SES data. In addition, the correlation among variables hinders our ability to disentangle their associations with respect to variability.

Wildfire-related pollution is potentially an environmental justice issue because of the disparities in wildfire-smoke exposures and health responses, as well as the options to adapt (e.g., via accessing medical care and making lifestyle changes). In our study, we demonstrated important policy implications of this environmental justice issue. Public health would be improved by raising awareness of wildfire smoke exposure for high-risk subpopulations. Other efforts, such as prescribed fires, can reduce "the intensity, size, and damage of wildfires" (20, p. 117), which may benefit high-risk communities.

The patterns of subpopulations with higher exposure to smoke waves relate to the patterns of wildfire smoke and the interacting patterns of race, poverty, urbanicity, and region. Persons who are ethnic minorities are more likely to be socioeconomically disadvantaged (21). Poverty and education have been used as a indicators of SES in previous studies in which the associations between air pollution and health were investigated (22–25). Our findings suggest that counties with low SES might be more likely than others to experience intense smoke waves. These findings are generally consistent with conclusions from previous studies on SES and air pollution (26–28).

Although our results relate to the ambient levels of $PM_{2.5}$ due to wildfires, personal exposures may also differ by subpopulation. Persons with disadvantaged SES might be less aware of the potential health risks caused by wildfire smoke (29) or less likely to quickly respond to extreme wildfire smoke by moving or staying indoors (30). They are also more likely to live in low-cost neighborhoods that lack community support in response to adverse environmental conditions (28). All of these could result in higher exposures to wildfire smoke.

Some subpopulations might be more vulnerable in health response to wildfire-specific $PM_{2.5}$ because of biological and/or social factors. Results from the literature have suggested that females might be more vulnerable than males, possibly because of differences in lung function and dermal absorption (11). In the present study, the central estimate of the smoke-wave association was higher for women than for men, but those for

Table 2. Percent Change in Rate of Respiratory Hospital Admissions on Smoke-Wave Days Compared With Non–Smoke-Wave Days, by Subpopulation, Western US Counties, 2004–2009

Subpopulation	Relative Risk of Hospital Admission[a]		Difference in % Change in Rates of Admission[b]	
	Central Estimate	95% CI	Central Estimate	95% CI
Age, years[c]				
65–74	1.07	0.97, 1.18	0.0	Referent
75–84	1.08	0.99, 1.18	0.9	−10.0, 13.2
>85	1.06	0.97, 1.17	−0.5	−11.8, 12.2
Sex[c]				
Male	1.04	0.95, 1.13	0.0	Referent
Female	1.10	1.02, 1.20[d]	6.5	−3.2, 17.1
Race[c]				
White	1.07	1.00[e], 1.15	0.0	Referent
Black	1.22	1.00, 1.47[d]	13.8	−6.0, 37.9
Other race	1.04	0.90, 1.18	−3.2	−15.5, 10.9
Urbanicity[f]				
Urban	1.07	0.99, 1.15	0.0	Referent
Less urban and rural	1.12	0.96, 1.29	4.8	−11.2, 23.5
Region[f]				
California	1.04	0.96, 1.12	0.0	Referent
Northwest	1.28	0.98, 1.67	23.6	−6.3, 63.0
Southwest	1.09	0.51, 2.38	5.5	−51.7, 130.5
Rocky Mountains	1.04	0.73, 1.47	0.5	−29.4, 43.0
Poverty, %[f]				
<10	1.23	0.86, 1.76	0.0	Referent
10–15	1.06	0.93, 1.20	−14.4	−41.5, 25.3
>15	1.06	0.98, 1.15	−14.0	−40.4, 24.1
Educational level[f]				
≥20% with bachelor's degree	1.06	0.98, 1.14	0.0	Referent
<20% with bachelor's degree	1.13	0.97, 1.31	6.3	−10.2, 25.8

Abbreviation: CI, confidence interval.

[a] Relative risk of hospital admission for respiratory causes on smoke-wave days compared with non–smoke-wave days within subpopulation.

[b] Difference in percent change in rates of admission on smoke-wave days compared with non–smoke-wave days when comparing rates in subpopulation with rates in the reference population.

[c] Associations for individual-level characteristics were estimated using interaction models.

[d] Statistically significant difference between the subpopulation and reference subpopulation ($P < 0.05$).

[e] The lower confidence interval is 0.996 and was rounded to 1.00.

[f] Associations for community-level characteristics were estimated using stratified models.

the 2 sexes were not statistically different. Results from previous studies also suggested that those with lower SES may have higher health risks because of poorer nutrition, less access to health care (31), and higher baseline health rates (e.g., a higher morbidity rate than other subpopulations) (Web Table 1). We found that central estimates of smoke-wave associations for persons in less-educated counties were higher than those for persons in more-educated counties, but the associations for the 2 educational levels were not statistically different.

More studies are needed to investigate the association between exposure to wildfire smoke and health outcomes, especially among vulnerable populations (3). In some prior work, researchers investigated wildfire vulnerability. Künzli et al. (32) assessed exposure to wildfire smoke by surveying the number of days participants smelled smoke. Their findings suggested higher exposure for elementary school children than for high school children. Some prior studies indicated that, in general, older people (e.g., >65 years of age) (5–7, 9) and small children (0–4 years of age) are the most vulnerable to wildfire smoke (6). Some studies found low SES populations to be more vulnerable than other populations (33–37). Two studies found females to be more vulnerable than males to wildfire smoke (35, 37). In most earlier studies, researchers defined wildfire periods or seasons a priori and assessed health risks in relation

to variations of metrics of total mass air pollutant levels potentially elevated by wildfire smoke, whereas we assessed exposure to $PM_{2.5}$ specifically from wildfire smoke.

The subpopulations that experience higher exposure to wildfire-specific $PM_{2.5}$ may be similar to those subpopulations with higher exposures to all-source $PM_{2.5}$ (i.e., total mass $PM_{2.5}$). In the United States, persons who are black, have a low educational level (less than a high school diploma), and live in poverty have higher exposure to ambient $PM_{2.5}$ (26).

The subpopulations that are vulnerable to potential health effects from wildfire-specific $PM_{2.5}$ may differ from those who are vulnerable to all-source $PM_{2.5}$. The association between total mass $PM_{2.5}$ concentrations and hospital admissions for respiratory conditions among Medicare patients (≥65 years of age) was higher in northern California and the Rocky Mountain regions than the rest of the western United States (38). In comparison, we found that the central estimate of smoke waves for persons older than 65 years in the northwestern region of the United States was higher than that for persons from other western regions of the United States, but risks were not significantly different across regions. Bell et al. (39) found that women were more vulnerable than were men to hospital admissions associated with all-source $PM_{2.5}$ concentrations. People older than 75 years of age had higher risks for admissions due to chronic obstructive pulmonary disease when exposed to total mass $PM_{2.5}$ than did people aged 65–74 years (40), but we did not find differences in association estimates by age for wildfire-specific $PM_{2.5}$.

In the present study, we assessed vulnerability based on a large spatial domain and 6-year period with numerous wildfire smoke episodes. Our results provide suggestive evidence that sex, sociodemographic characteristics, and region may play a role in vulnerability to wildfire smoke. More research is needed to estimate vulnerability to wildfire smoke by incorporating future wildfire patterns and changes in demographic characteristics.

ACKNOWLEDGMENTS

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This work was supported by grants R01ES019560, R21ES020152, R21ES021427, R21ES022585, and T32 ES007142 from the National Institute of Environmental Health Sciences, National Institutes of Health, and grants 83479801 and 83587501 from the US Environmental Protection Agency (EPA). This publication was developed under assistance agreement 83587101, which awarded by the EPA to Yale University.

This publication has not been formally reviewed by the EPA. The views expressed in this document are solely those of the authors and do not necessarily reflect those of the Agency. The EPA does not endorse any products or commercial services mentioned in this publication.

Conflict of interest: none declared.

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Generation of Viable Bacterial and Fungal Aerosols during Biomass Combustion

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Received: 20 February 2020; Accepted: 22 March 2020; Published: 24 March 2020

Abstract: Biomass combustion is known to be one of the main contributors to air pollution. However, the influence of biomass burning on the distribution of viable bacterial and fungal aerosols is uncertain. This study aimed to examine survivability of bacteria and fungi in the post-combustion products, and to investigate the aerosolization of viable cells during combustion of different types of organic materials. Laboratory experiments included a small-scale combustion of organic materials contaminated with microorganisms in order to determine the survivability of microbes in the combustion products and the potential aerosolization of viable cells during combustion. Field experiments were completed during intentional and prescribed biomass burning events in order to investigate the aerosolization mechanisms that are not available at the laboratory scale. Laboratory experiments did not demonstrate aerosolization of microorganisms during biomass combustion. However, the relatively high survival rate of bacteria in the combustion products ought to be accounted for, as the surviving microorganisms can potentially be aerosolized by high velocity natural air flows. Field investigations demonstrated significant increase in the bioaerosol concentration above natural background during and after biomass combustion.

Keywords: bioaerosol generation; biomass combustion; combustion bioaerosols; high temperature bioaerosols; prescribed burning

1. Introduction

Bioaerosols are known to be highly sensitive to high temperatures. It was shown that common bacterial species such as *Escherichia coli* and *Bacillus subtilis* become more than 99.9% inactive when exposed to the temperature of 160 °C and 350 °C, respectively, for about 0.3 s [1]. However, a number of industrial high temperature processes can be responsible for microorganisms' aerosolization. An exothermic process of lime slaking associated with a significant amount of heat causes aerosolization of bacteria at rates similar to other intensive mechanical processes, such as diffused aeration of wastewater [2]. In our previous work [3] we experimentally proved a possibility of aerosolization of viable microorganisms as a result of interaction of biologically contaminated liquid with hot surfaces that may occur in the processes of industrial cooling.

Biomass burning is a major source of aerosols which contributes up to 67% more carbon particle emissions compared to the combustion of fossil fuels [4]. At the same time, biomass burning is a process of interest as a renewable source for power and heat generation. Biomass-fueled electric generating facilities are characterized by a high level of particulate matter (PM), especially in boiler rooms and biomass storage rooms [5]. However, it was not confirmed that the combustion process itself represents a mechanism of bioaerosol generation, as the elevated levels of bioaerosols and biogenic organics are commonly related to the pre-combustion processes [6,7] that include storage or mechanical disturbance of biomass.

Some potential generation of biological aerosols can be related to biomass combustion in open fire at residential and industrial dwellings. Semple et al. [8] measured endotoxin levels within the

living area of 69 houses while burning different biomass fuels in Malawi and Nepal. The results showed that median concentrations of total inhalable endotoxin were orders of magnitude higher than the level of 0.49 EU/m^3 (geometric mean) which is linked to respiratory illnesses in children [9]. A very recent investigation reported some increase in airborne fungal concentration in the vicinity to forest fires at Madeira island [10]. It was suggested that the fire-induced convections are capable of promoting the release of fungal spores from their natural habitat.

Recent investigations of bacterial and fungal communities were conducted in China during summer harvest and biomass burning season [11]. The study showed that the total bacterial and fungal concentrations during biomass burning events are higher than the non-biomass burning events. However, the difference between the concentrations was not confirmed and the influence of harvesting activities was not investigated. It was suggested that bacterial cells and fungal spores can be carried: (a) by the turbulent air caused by combustion; or (b) attached to particulates released during combustion.

The aims of this study were: (1) to examine survivability of bacteria and fungi during combustion of different types of contaminated organic materials under controlled laboratory conditions; and (2) investigate microbial aerosolization during and post biomass combustion processes occurring in controlled and natural environments. Special attention will be given to comparison of microbial behavior in combustion processes of dramatically different scales in the laboratory and in the field.

2. Experiments

2.1. Laboratory Investigation-Microorganisms' Survivability during Combustion

2.1.1. Bacterial Strains and Cultivation

Similar to our previous work [3], two common environmental bacterial strains, namely Gram-positive *Bacillus subtilis (B. subtilis)* American Type Culture Collection (ATCC) 6633 and Gram-negative *Escherichia coli (E. coli)* ATCC 27325 were obtained from Southern Biological (Nunawading, VIC, Australia) and used for the laboratory section of experiments. *E. coli* bacterial cells are known to be sensitive to pasteurization if present in a liquid material.

Stock cultures of *B. subtilis* and *E. coli* were grown in 1.3 g/100 mL of dry nutrient broth (OXOID Ltd., Basingstoke, Hampshire, England) in deionized and sterilized water for 18 h in an incubator shaker at a constant temperature of 37 °C. The microbial suspension was used directly after incubation in order to avoid sporulation of *B. subtilis* cells. The aliquots of the two microbial suspensions were mixed in a ratio of 1:1 (w/w) and the mixture was used for all of the experiments.

2.1.2. Samples Preparation

The entire experimental program was undertaken inside a 1200 mm wide Class II Biohazard Cabinet. The biohazard cabinet was used to meet the biosafety requirements, to prevent any escape of microbial materials to the laboratory air space and to ensure zero aerosol concentration eliminating chance for any potential alien microbial particles reaching experimental zone and interfere with the results.

The bacterial suspension was prepared as per the above description and spread undiluted over 60 mm diameter filter paper samples. The filters were then placed in the biohazard cabinet and air-dried overnight. The weight of each filter was measured with analytical scale ensuring even distribution of biological materials across all filters used. Then, the microbial materials from three filters were washed down with 50 mL of sterile deionized water and the aliquots were analyzed for culturable bacteria by commonly used plating technique as follows. An aliquot of 0.1 mL of an appropriate 10-fold dilution of the fluid was spread on the surface of the nutrient agar (NA) plates. The culture plates with bacteria were incubated at 37 °C for 1 day. Colony forming units (CFU) were counted after incubation with a colony counter (Biolab, Clayton, VIC, Australia), and the

corresponding viable bacterial concentration in the liquid was determined. The results were expressed in CFU per g of paper.

Dry leaves were randomly collected from the soil organic horizon at the university surroundings at Nathan campus, mixed together, and delivered to the laboratory. Then, 1 g of the dry leaf matter was washed with 40 mL of sterile deionized water and the concentration of bacteria in the aliquot was analyzed according to the procedure described above for the paper filters and expressed in CFU/g of leaves.

2.1.3. Laboratory Set-Up

The laboratory setup consisted of a metal tray (40 cm × 30 cm), plastic funnel (3 0 cm diameter), and bioaerosol sampling equipment. The plastic funnel was strategically placed above the tray to ensure minimal escape of generated bioaerosols achieving maximum possible microbial collection by the bioaerosol sampling equipment during combustion experiments.

A personal bioaerosol sampler with the operational principle based on the passing of air through a porous medium submerged into a liquid [12] was used in the experiments. According to the previously described procedure [13], the personal sampler was sterilized before the experiments, drained, and filled with 40 mL of sterile distilled water. For all the experiments the air temperature (T) and the relative humidity (RH) were controlled at T = 24–25 °C and RH = 24–26%. The sampler was connected to the air sampling pump (Model PCXR8, SKC Inc., PA USA) and operated at a flowrate of 4 L/min over 60 s for all the experiments in order to cover entire duration of combustion process ensuring complete collection of released bioaerosols.

To analyze the collection fluid after sampling, the sampler was drained, and the porous media removed from the device and placed into container filled with 20 mL of distilled sterilized water. Then, the container was sonicated in an ultrasonic bath for 5 min to remove any microorganisms possibly remaining inside the porous medium and, on completion of the procedure mixed with the original collection fluid. The concentration was determined according to the previously described procedure [12]. The total volume of liquid samples was divided into three equal parts followed by filtration through a Nalgene cellulose nitrate membrane filter with 0.2 μm pore size (Nalge Co., Rochester, NY, USA). Then the filters were placed onto triplicate nutrient agar (NA) and malt extract agar (MEA) plates for bacteria and fungi respectively.

Then the NA plates were labelled and incubated at 37 °C for 2 days and the MEA plates were kept at room temperature until colonies had developed (all plates were observed daily). After incubation, the number of colonies was counted with the colony counter and the results were represented in CFU/mL for the bacterial and fungal concentration.

2.1.4. Combustion and Bioaerosol Collection

100 g of contaminated paper filters were placed in the metal tray and combusted. Bioaerosols were collected during the entire period of combustion. The materials remaining in the tray after combustion were collected, weighed, and diluted with 40 mL of sterilized deionized water. The concentration of bacteria was analyzed as described in the previous sections and expressed in CFU per g of ash. 100 g of the dry leaf matter were combusted in a similar way. The concentration of viable bacteria was analyzed following the same procedure.

Bacteria recovery was calculated using the following formula:

$$RR = C_0/C_{ASH} \quad (1)$$

where C_0 is the bacterial concentration on the contaminated paper filters or leaf matter in CFU/g and C_{ASH} is the bacterial concentration in the ash remaining after combustion in CFU/g.

2.1.5. Ash Aerosolization

Dry leaves was randomly collected from the ground at the university surroundings. Undiluted bacterial suspension was applied on the leaves. The leaves were air-dried in a biohazard cabinet overnight.

The dried leaves were ignited with a flexible gas lighter and combusted outdoors inside a metal cylinder with an open top. Bioaerosols were collected with a personal bioaerosol sampler over the entire period of combustion.

The remaining ash was left in the metal cylinder and covered with a metal lid with a hole in the middle utilized for placement of the bioaerosol sampler and bioaerosol collection. The set-up allows for minimization of the influence of background concentrations on the results of experiment.

Air flow, created with a manual pump, was passing from the bottom of the cylinder in order to aerosolize the ash. Bioaerosols were collected for two minutes and incubated at 37 °C over 48 h.

2.2. Field Investigation–Generation of Bioaerosols during Biomass Combustion

Four separate series of experiments were conducted in three different locations in order to use various burning strategies and sampling techniques.

2.2.1. 5 August 2018, Cainbable, Queensland

In order to characterize the bioaerosol from an open biomass burning, an experiment was conducted in a field near a farm at Cainbable. Branches from common Australian tree species including Angophora, Lophostemon, Syzygium, and Eucalyptus genera were collected and piled up on the field to dry the biomass for three months. The length of the branches was in the range from 30 cm up to 2 m. The pile was 1.7 m high with an estimated volume of 11 m^3. There was no mechanical disturbance of the biomass pile at least two days before the experiment. Weather conditions remained consistent during all stages of the experiment; temperature was 23 °C, RH was 25%, the wind was below 10 km/h in the NE direction, and there was full sun.

All bioaerosol were collected with a personal bioaerosol sampler in a height of 1.5 m and distance of 2 m downwind from the pile. No closer sampling point location was possible due to very high air temperature and significant possibility of sampling equipment destruction in closer vicinity to the fire. The first series of samples had been collected before the pile was ignited to obtain natural bioaerosol background.

The pile was ignited with a lighter and combusted. The total combustion period lasted about 30 min. A number of bioaerosol samples was collected at the same point during the entire period of biomass flaming.

A series of bioaerosol samples was collected 30 min past combustion after biomass smoldering had been completed. There was no mechanical disturbance of the ash and remaining biomass prior to the bioaerosol sampling.

The bioaerosol samples were treated and incubated as per the procedure described above. The concentrations of viable microorganisms in the samples were analyzed and expressed in CFU/m^3.

Three identically sized piles were burned to ensure statistically reliable and reproducible results.

2.2.2. 28 June 2019, Dirranbandi, Outback Queensland

A second series of experiments was completed in a remote arid area of Queensland in order to eliminate the influence of surrounding vegetation, utilize a different sampling technique, and analyze whether the microbe abundance differs with increasing distance from the fire. As in the first series of experiments, a variety of dry branches of common Australian tree species were collected in a pile and ignited with a lighter for combustion. Petri dishes with nutrient agar (NA) and malt extract agar (MEA) were placed at distances of 1.5, 2.7, 3.9, 5.1, 6.3 and 7.5 m downwind from the burning biomass pile (Figure 1) similar to the method described by Kobziar et al. [14]. Background samples were collected in the area not affected by the fire, however, to ensure similar ambient parameters and the abundance of vegetation. Samples were collected via passive deposition onto agar over 1.5 h. The samples were closed, sealed with parafilm and delivered to the laboratory. NA samples were incubated at 37 °C for 1 day, and MEA samples were stored at room temperature until colonies had developed. After incubation, the number of colonies was counted with the colony counter and the

results were represented in CFU. The temperature during experiments was 21 °C, RH was 43%, wind speed was around 17 km/h in SW direction, and there was full sun.

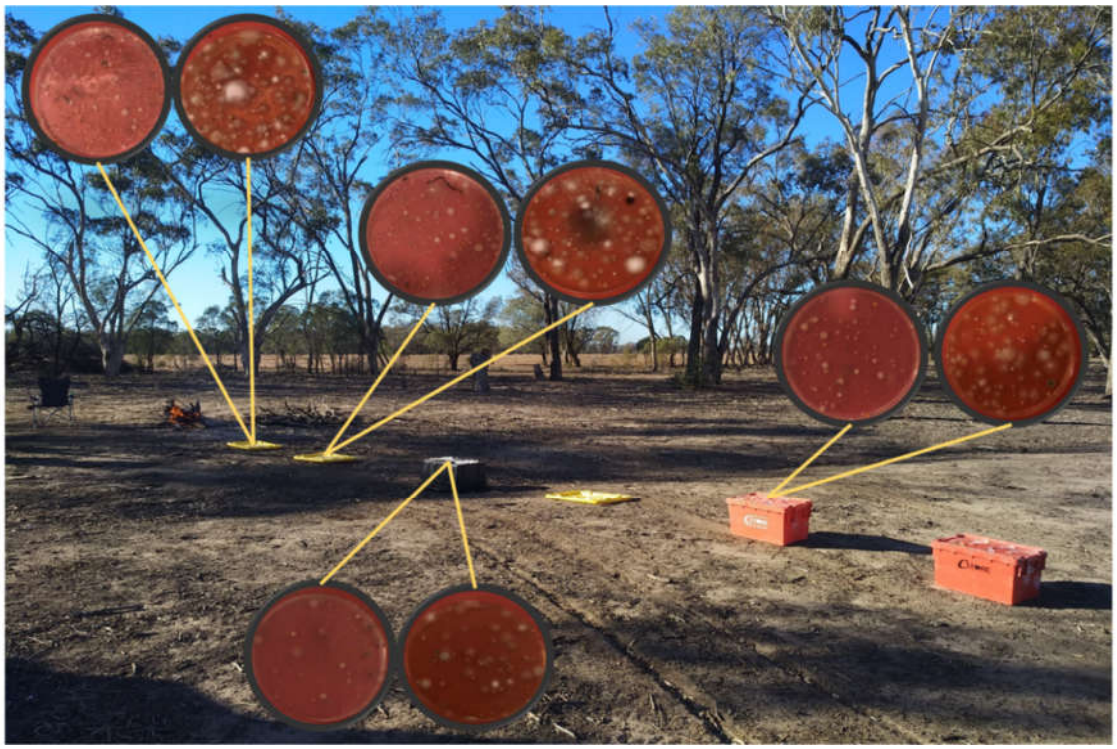

Figure 1. Positioning of Petri dishes in the Dirranbandi series of biomass combustion experiments.

2.2.3. 11 and 12 June 2019, Nathan campus of Griffith University

A third series of experiments was conducted during the hazard reduction burns at Nathan campus of Griffith University on 11 and 12 June 2019 (Figure 2). Fuel loads on the forest floor were assessed medium to high in places and needed to be managed for the safety of students, staff, motorists, and other people in the area. Controlled burning was lit in a mosaic pattern to have minimum impact on fauna in the area. The temperature was 23 °C, RH was 50%, wind speed was 11 km/h in the direction of NE, and it was slightly cloudy.

A number of samples were collected a day before the prescribed burning in order to evaluate the background concentration of microorganisms in the same environment. Twelve Petri dishes were suspended on the trees in six different parts of the forest approximately 2 m above ground. The samples were collected via passive deposition onto nutrient agar (NA) and malt extract agar (MEA) during 6 h. The samples were closed, sealed with parafilm and delivered to the laboratory immediately after the collection had been completed.

The same method was used to collect samples of bioaerosols during the prescribed burning. Petri dishes were suspended at the same locations 30 min before the fire was initiated with a drip torch. The dishes were collected in 6 h and at that time a remaining fire and smoldering were still observed in different parts of the forest. Weather conditions remained consistent during the two days of sampling.

The samples were delivered to the laboratory immediately after collection. NA samples were placed in the incubator at 37 °C for 48 h, and the MEA samples were kept at room temperature in the dark over 5 days. The plates were visually examined for microorganisms daily and CFUs were counted where possible.

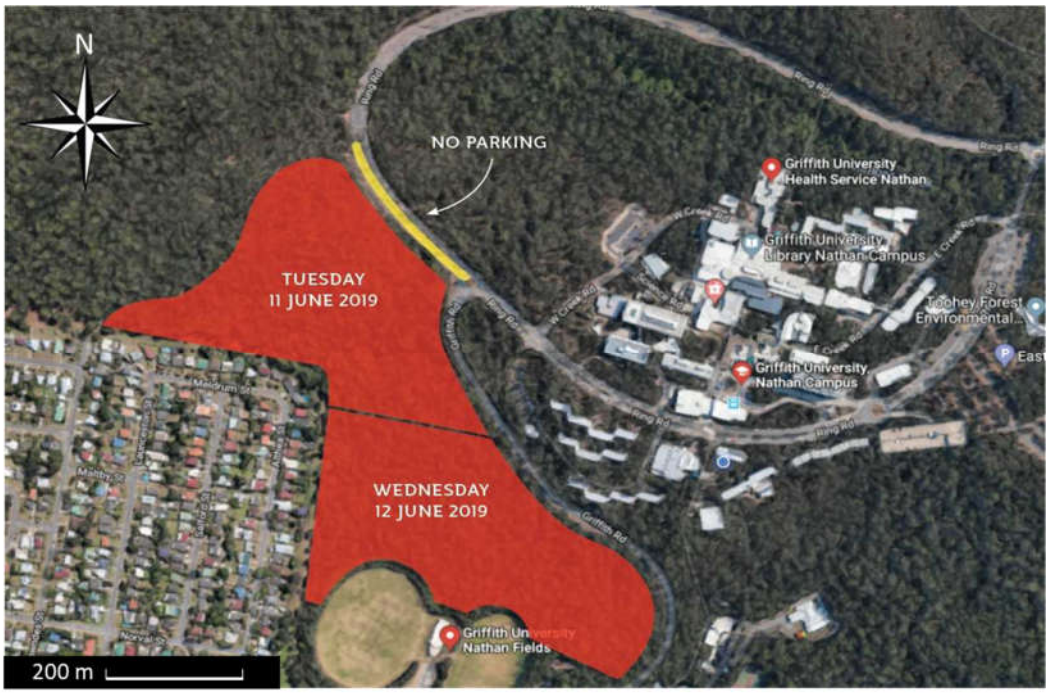

Figure 2. Map of hazard reduction burns carried out at Nathan campus of Griffith University on 11 and 12 June 2019.

2.2.4. 17 and 18 August 2019, Nathan campus of Griffith University

The final series of experiments was conducted during the next period of prescribed burning at Nathan campus of Griffith University (Figure 3) in order to utilize a different sampling strategy and technique. During the experiments, the temperature was 22 °C, RH was 47%, wind speed was 13 km/h in the direction of SW, and there was full sun.

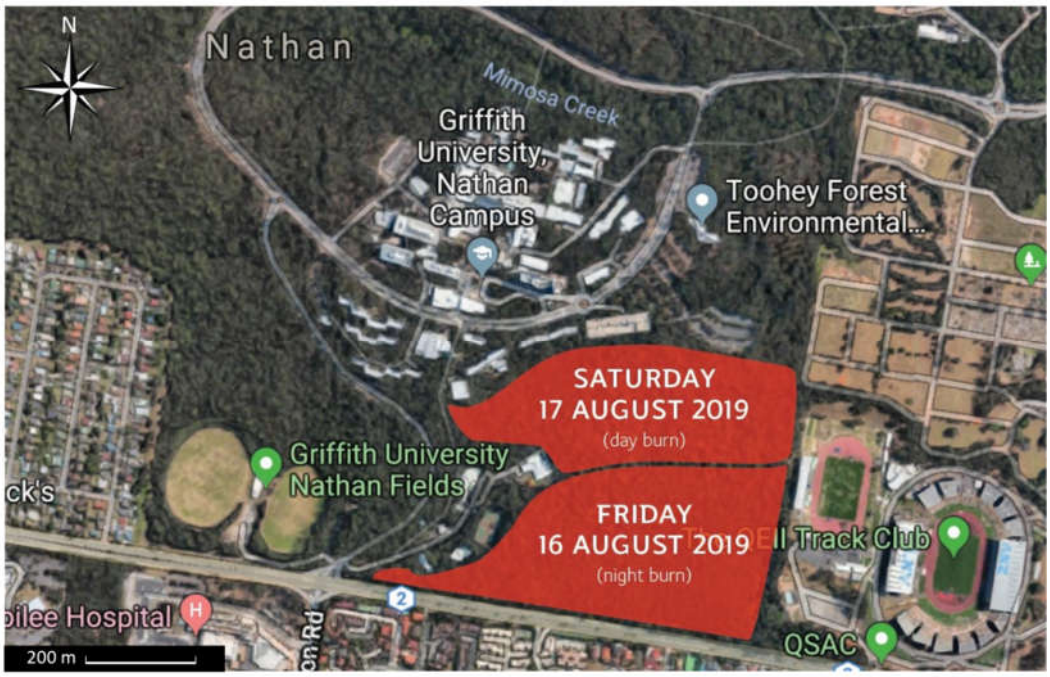

Figure 3. Map of hazard reduction burns carried out at Nathan campus of Griffith University on 17 and 18 August 2019.

Air samples were collected with a personal sampler over 15 min. Prior to the experiment, each device was charged with 40 mL of fresh collecting liquid, sealed in plastic bag and transported to the sampling location. On arrival, the samplers were unpacked, connected to the air sampling pump (Model PCXR8, SKC Inc., Eighty Four, PA, USA), and operated for 15 min at the flow rate of 4 L/min.

A number of samples had been collected: (a) before the prescribed burning started, in order to evaluate the background concentration; (b) in the hot areas of burning characterized by high smoke concentration and elevated temperature, in order to assess the potential of burning process itself to generate bioaerosols; and (c) at a distance of 30 m downwind from the most intensive burning, in order to assess the influence of other possible factors and mechanisms of aerosolization of microorganisms.

On completion, the sampler was disconnected from the pump, placed in the sealed plastic bags and carefully delivered to the laboratory for processing in accordance with the procedure described above.

3. Results

3.1. Contaminated Material Combustion and Bioaerosol Collection

The abundance of culturable bacteria on contaminated paper filters and leaf matter, as well as material remaining post combustion, were analyzed, and the results are summarized in Table 1. Despite significant mass reduction (94%), 28% of culturable bacteria were recovered from the paper filters post combustion. Leaf matter was characterized by lower mass reduction due to higher moisture content and complex nature of the material. However, the post combustion remainders of leaf mater showed a lower bacteria recovery rate (18%).

Table 1. Survivability of microorganisms as a result of incomplete combustion of contaminated organic materials. Bacteria recovery rate is calculated using formula (1).

Parameters	Units	Before Combustion		Post Combustion	
		Paper Filter	Leaf Matter	Paper Filter	Leaf Matter
Mass	g	0.65(±0.01)	1.89(±0.28)	0.04(±0.00)	0.71(±0.11)
Concentration	CFU/g	$2.51(\pm0.38) \times 10^6$	$6.33(\pm0.95) \times 10^3$	$6.90(\pm1.04) \times 10^5$	$1.13(\pm0.17) \times 10^3$
Mass reduction	%	-	-	94%	62%
Bacteria recovery	%	-	-	28%	18%

There were no viable microorganisms detected in the air samples collected during combustion of contaminated paper filters or leaf matter. The results allow for the possibility that the combustion process itself does not promote aerosolization of viable bacteria; however, culturable microorganisms were detected in the post combustion remainders as a result of incomplete combustion. Bioaerosols collected during ash aerosolization with manual pump demonstrated almost five times higher concentration of viable microorganisms (3244 CFU/m^3) than the ambient concentration (650 CFU/m^3).

3.2. Generation of Bioaerosols during Biomass Combustion

Bioaerosols collected during the first series of field investigation at a distance of two meters from the fire demonstrated higher concentrations of bacteria during biomass combustion (2.85×10^3 CFU/m^3) compared to the background concentrations measured prior to ignition (1.33×10^3 CFU/m^3). A significant increase in the total concentration of bacteria in the post combustion environment (1.49×10^3 CFU/m^3) can also be observed in Figure 4.

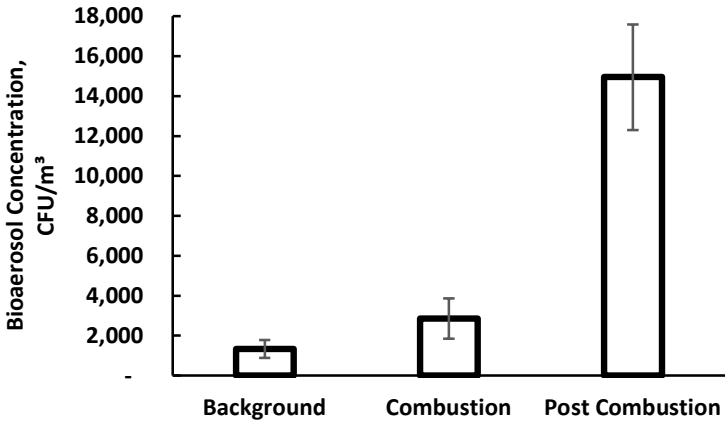

Figure 4. Concentration of culturable bacteria in the ambient air collected with a personal sampler in the Cainbable series of experiments. Error bars represent the standard deviation of three experimental runs.

Bioaerosols collected via passive deposition in the arid area showed that the concentration of both bacteria and fungi in the air was reduced with increased distance from the fire (Figure 5). Visual assessment of the samples showed that the samples collected near the fire were characterized by a larger number of ash particles settling on the plates. The number of ash particles was reduced with increased distance from the fire. The background concentration was acquired by using the same plates placed at the same distances from the fire prior to the commencement of combustion process. To ensure sufficient integrity of the results, three repeats of the background concentration monitoring procedure were undertaken.

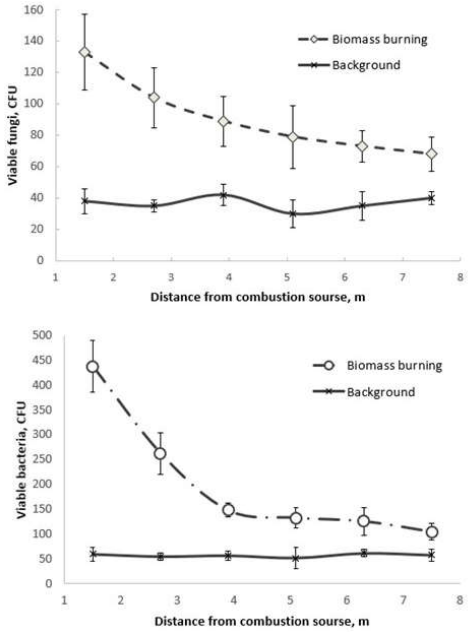

Figure 5. Relationships between the concentration of viable bacteria (**a**) and fungi (**b**), in the Dirranbandi series of experiments, and the distance from the fire. Error bars show the standard deviation of three experimental repeats for both, background and burning related monitoring.

3.3. Prescribed Burning Experiment

Bioaerosol samples collected during prescribed burning via passive deposition on nutrient agar (NA) did not demonstrate any colony growth. The number of fungal colonies was higher in the

background samples than in the samples collected in the hot area affected by the fire (temperature above 40 °C).

The samples collected with a personal sampler in August showed lower concentrations of microorganisms in the proximity and significantly higher concentrations of bioaerosols in the surrounding areas compared to the non-fire concentration (Figure 6). The difference in concentration was quite significant, reaching a concentration of bacterial aerosols almost 7 times higher compared to the fire front areas.

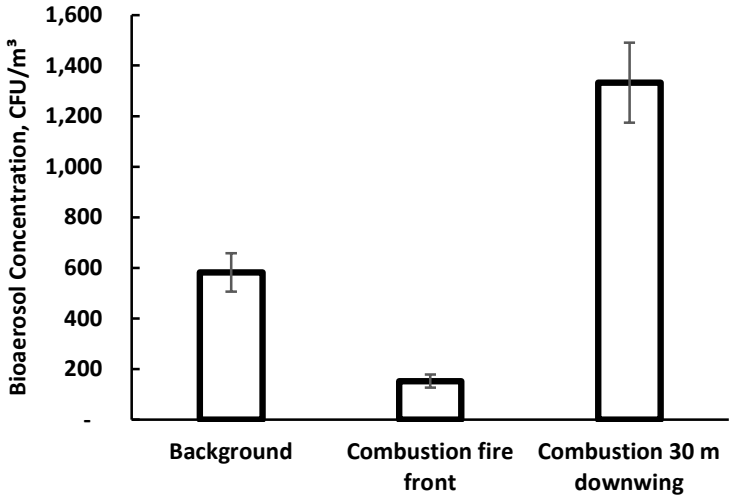

Figure 6. Concentration of viable microorganisms in the ambient air collected with a personal sampler during biomass combustion in the Griffith series of experiments. Error bars show the standard deviation of five experimental repeats.

4. Discussion

Kobziar et al. [14] conducted a number of laboratory and field investigations to provide a foundational understanding of the capability of wildland fire to aerosolize viable microorganisms in smoke. As the investigation collaborated a few different disciplines, a new term, Pyroaerobiology, was introduced to integrate micro- and aerobiology, smoke and atmospheric sciences, and fire behavior and ecology.

The laboratory experiments did not demonstrate any significant differences between the concentration of culturable microorganisms during combustion and the ambient samples [14]. This study was conducted in a biohazard cabinet to eliminate the influence of ambient conditions. Similar to the current study, no culturable microorganisms were detected in the air during the laboratory experiments. This allows for the suggestion that a small-scale fire does not provide a mechanism of microorganism aerosolization. However, such conclusion can only be made for culturable microbes involved in the current investigation.

At the same time, the experiment showed a significant extent of microorganisms' survivability in the post combustion material and possibility of their aerosolization where an additional factor such as wind is present. Survivability of microorganisms in these experiments can be explained by incomplete combustion of contaminated material that would also take place during biomass burning events and wildfires when the soil and vegetation biomass are affected by the fire. Biomass burning is known to have significant short-term effects on the abundance of soil microorganisms. A decrease in the abundance of total bacteria in fire-affected soil was observed three days after biomass burning, with the following restoration in the abundance of total bacteria nine days after burning [15]. Soil ecosystem stability is influenced by the burning frequencies and can sustain prescribed burning occurring with more than four-year intervals [16]. An investigation of wildfire effects on soil bacterial and fungal communities in an extreme fire season in the northwestern Canadian boreal forest showed

that fire occurrence, as well as moisture regime, are among the significant predictors of post combustion soil microbial community composition [17].

Since the soil microorganisms, as well as microorganisms attached to the plants and trees, are capable of surviving during bushfires, they can be aerosolized if a suitable mechanism or conditions occur. There are two possible changes in the ambient conditions caused by the fire and relevant to variations in the bioerosol concentration: elevated level of pollutants in the air and turbulent air flow created by the burning process.

A number of laboratory biomass burning experiments were conducted to quantify emission factors of domestic heaters operating in different conditions and different types of biomass fuels. However, they are hardly capable of reflecting burning conditions similar to those of wide, open fires, due to the sensitivity of the combustion process to the burning conditions [18]. Small scale experiments conducted in remote areas as part of this study demonstrated significant dependence of both fungi and bacteria distribution upon the ash content in the air.

Wildfires and biomass burning events provide a significant contribution to the elevated concentrations of particulate matter with aerodynamic diameter <10 μm (PM10) [19,20]. PM10 is known to be the most significant factor that often shows positive correlation with the concentration of airborne microorganisms [21,22].

A comprehensive one-year study [23] showed a strong positive correlation of Gram-positive and gran-negative bacteria concentrations with organic carbon and biomass burning derived potassium that indicated their association with emissions from biomass burning. Wei et al. [11] have recently confirmed elevated concentrations total bacteria and fungi during the burning period in China. The critical factors for fungal and bacterial communities included organic carbon, magnesium and wind speed.

Elevated concentrations of hydroxy fatty acids were recently observed during biomass burning events in China [24]. Hydroxy fatty acids are potential tracers for soil microbes, plant pathogens, and higher plant waxes. Endotoxin concentrations were also higher than the health-based occupational guidance limit (~90 EU m^{-3}). Emission factors for trace gases and aerosols, as well as their chemical speciation for the open fire burning of pruning residues were investigated in Portugal. Burning prunings emitted substantial amounts of gaseous and particulate pollutants that can have harmful impacts on human health and ecosystems, including polyols which might occur as constituents in bacteria or fungi [25]. Elevated concentrations of arabitol and mannitol, fungal molecular tracers, were detected in fine particle samples collected in during a biomass burning season in China [26].

Total bacteria in atmospheric aerosols depend on a number of factors, including geographical terrain and analytical methods, and may vary from 10^2 to 10^6 cells·m^{-3} [11]. The average concentration of microorganisms in the air before combustion was around 1.33×10^3 CFU/m^3 and 5.82×10^2 CFU/m^3 for biomass combustion ad prescribed burning experiments respectively, which is in line with other studies. The concentration of microorganisms measured during combustion downwind from the fire in both experiments was two times higher than the background concentration and the concentration in the post combustion environment was more than an order of magnitude higher than the background concentration.

Wind speed is considered to be one of the main factors affecting bioaerosol and $PM_{10\text{-}2.5}$ concentrations in the atmosphere. Winds with a mean speed of ≥5.5 m s^{-1} can mechanically resuspend surface dust, thus re-aerosolizing $PM_{10\text{-}2.5}$ as well as bioaerosols settled on the surface or attached to dust particles [27]. Fire plumes are characterized by high velocities. Vertical velocity can reach 13 m/s, with downdrafts of ~8 m/s [28]. It is speculated that this high velocity acts a strong wind and causes a significant disturbance of plants therefore detaching microorganisms from biomass (leaves, trees etc.).

Future investigations are required in order to evaluate the relationship between the composition of microorganisms deposited on different types of biomass (soil layers, grass, trees) in the fire areas and the composition of microorganisms in the surrounding atmosphere. Laboratory investigations may focus on the aerosolization of microorganisms with vertical air flows, in order to imitate the process of biomass burning in the natural environment.

5. Conclusions

This study demonstrates the capability of viable microorganisms to survive in the material remaining after biomass combustion. Viable microorganisms are able to aerosolize if an additional source of aerosolization is present. Large-scale fires such as wildfires or prescribed biomass burnings significantly contribute to air microbial quality. Elevated concentrations of fungi and bacteria were observed during biomass combustion and prescribed burnings, as well as in the post combustion environment. The two possible changes in the ambient conditions caused by the fire and relevant to variations in the bioaerosol concentration include elevated levels of all types of combustion related air pollutants and turbulent air flow created by the burning process. Finally, it ought to be noticed that laboratory scale experiments might not always be representative for modeling bioaerosol release from natural bushfire events; special attention must be given to various simulated parameters, especially plume velocity and direction.

Author Contributions: Conceptualization, E.M. and I.E.A.; Methodology, E.M. and I.E.A.; investigation, E.M. and I.E.A.; writing—original draft preparation, E.M.; writing—review and editing, I.E.A.; visualization, E.M.; supervision, I.E.A. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Conflicts of Interest: The authors declare no conflict of interest.

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Is Exposure to Wildfires Associated with Invasive Fungal Infections?

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Meeting: 2019 American Transplant Congress

Abstract number: A338

Keywords: Risk factors

Topic:

Session Information

Session Name: Poster Session A: Transplant Infectious Diseases

Date: Saturday, June 1, 2019

Session Time: 5:30pm-7:30pm

Presentation Time: 5:30pm-7:30pm

Location: Hall C & D

***Purpose:** In recent decades, climate change and other factors have led to a global increase in wildfires. After a large fire in Northern California in 2017, we anecdotally noted more invasive fungal infections (IFI) at our quarterary care hospital that led us to hypothesize a link between the two. We sought to determine whether exposure to wildfires was associated with an increased risk of IFI.

***Methods:** We performed time series analysis of admissions for infections with invasive mold (all molds, including *Aspergillus*), *Aspergillus* spp, and *Coccidioides* spp during periods with and without fire across twenty-two hospitals in California between October 2014 and May 2018. All hospitals were members of Vizient, Inc and consistently reported into the Clinical Database Resource Manager. Accounting for clustering by hospital and controlling for season, we used quasi-Poisson regression to determine whether there was an increased risk of IFI in months where there was a large fire (> 5000 acres) within a 200-mile radius of the hospital. Invasive *Candida* infections served as controls as these tend to be endogenous and not typically associated with environmental exposures.

***Results:** During the study period, there were a total of 968 months of time across all the hospitals with 400 months (41.3%) experiencing large fires. Average rates of invasive mold, aspergillosis, coccidioidomycosis, and invasive candidal infections were 5.84, 2.89, 0.88, and 1.01 cases per 1000 admissions, respectively. Table 1 shows incidence rate ratios for IFI during wildfire exposure.

***Conclusions:** Months during which large wildfires occurred within 200 miles of a hospital were associated with an increased risk of invasive mold infection, aspergillosis, and coccidioidomycosis but not with candidal infection. Wildfire smoke contains particulate matter, which in turn contains fungal spores. One possible mechanism for the increased risk is that wildfire smoke leads to spread of fungal spores that can be inhaled by at-risk individuals. It is possible that the association between fires and IFI may result from environmental circumstances that predispose to fire rather than to the actual fires; further work needs to be done to incorporate these conditions into the model to elucidate our observation. If borne out in further prospective studies, the association between IFI and wildfire exposure has important implications for prophylaxis and monitoring of at-risk patients.

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Table I: Incidence rate ratios (95% CI) for IFI admissions by wildfire exposure and season

	Large wildfire within 200 miles (compared to months with no fire)	Fall season (compared to Summer)
Invasive mold	1.18 (1.11-1.25)	1.24 (1.15-1.33)
Aspergillosis	1.22 (1.11-1.32)	1.35 (1.21-1.50)
Coccidioidomycosis	1.22 (1.07-1.40)	1.36 (1.14-1.62)
Invasive *Candida*	1.03 (0.90-1.18)	1.01 (0.85-1.20)

To cite this abstract in AMA style:

Mulliken JS, Rappold AG, Fung M, Babik JM, Doernberg SB. Is Exposure to Wildfires Associated with Invasive Fungal Infections? [abstract]. *Am J Transplant*. 2019; 19 (suppl 3). https://atcmeetingabstracts.com/abstract/is-exposure-to-wildfires-associated-with-invasive-fungal-infections/. Accessed October 22, 2021.

Inhalation Toxicology, 19:67–106, 2007

ISSN: 0895-8378 print / 1091-7691 online
DOI: 10.1080/08958370600985875

informa
healthcare

Woodsmoke Health Effects: A Review

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The sentiment that woodsmoke, being a natural substance, must be benign to humans is still sometimes heard. It is now well established, however, that wood-burning stoves and fireplaces as well as wildland and agricultural fires emit significant quantities of known health-damaging pollutants, including several carcinogenic compounds. Two of the principal gaseous pollutants in woodsmoke, CO and NO_x, add to the atmospheric levels of these regulated gases emitted by other combustion sources. Health impacts of exposures to these gases and some of the other woodsmoke constituents (e.g., benzene) are well characterized in thousands of publications. As these gases are indistinguishable no matter where they come from, there is no urgent need to examine their particular health implications in woodsmoke. With this as the backdrop, this review approaches the issue of why woodsmoke may be a special case requiring separate health evaluation through two questions. The first question we address is *whether woodsmoke should be regulated and/or managed separately,* even though some of its separate constituents are already regulated in many jurisdictions. The second question we address is *whether woodsmoke particles pose different levels of risk than other ambient particles of similar size.* To address these two key questions, we examine several topics: the chemical and physical nature of woodsmoke; the exposures and epidemiology of smoke from wildland fires and agricultural burning, and related

Received 22 March 2006; accepted 4 August 2006.

The authors thank the Air Health Effects Division of Health Canada for funding to help prepare the report on which this article is based, but the views expressed are those of the authors and do not necessarily represent the views of Health Canada. The authors also thank the following for their assistance: Jamesine Rogers, MPH, and Zohir Chowdhury, PhD, of the University of California at Berkeley; Diana Ceballos, doctoral candidate at the University of Washington; and Jessica Duffy, MS, doctoral candidate, Shannon Doherty, MS, and Colette Prophete at New York University School of Medicine.

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controlled human laboratory exposures to biomass smoke; the epidemiology of outdoor and indoor woodsmoke exposures from residential woodburning in developed countries; and the toxicology of woodsmoke, based on animal exposures and laboratory tests. In addition, a short summary of the exposures and health effects of biomass smoke in developing countries is provided as an additional line of evidence. In the concluding section, we return to the two key issues above to summarize (1) what is currently known about the health effects of inhaled woodsmoke at exposure levels experienced in developed countries, and (2) whether there exists sufficient reason to believe that woodsmoke particles are sufficiently different to warrant separate treatment from other regulated particles. In addition, we provide recommendations for additional woodsmoke research.

As the ability to control fire is often considered the characteristic distinguishing prehuman and human evolution and wood is the oldest of human fuels, it is literally true that exposure to woodsmoke* is as old as humanity itself. Even today, biomass in the form of wood and agricultural wastes is a significant source of direct human energy consumption worldwide, representing about 10% of the total. Of this, about 90% is used in its traditional forms as household heating and cooking fuels in developing countries, the rest being modern forms such as powerplant fuel, principally in developed countries (United Nations Development Programme [UNDP], 2004). Because household use dominates total fuel demand in many developing countries, particularly in rural areas where half of humanity still lives, it is likely that biomass remains the main source of energy for most of humanity.

Surprisingly, although the percentage of total fuel demand constituted by wood declines with economic development, the absolute amount remains relatively constant. For example, the average use of biomass fuel per capita in the primarily wealthy countries participating in the Organization for Economic Cooperation and Development (OECD) is quite similar to that in Asia, which has the world's largest developing nations (UNDP, 2004). Of course, per capita use varies substantially with local circumstances. Countries with ample wood supplies, such as Finland, Sweden, and Canada, burn more biomass fuel per capita than most other countries, while those with low supplies, such as South Korea and Singapore, burn less (Koopmans, 1999).

Over the past few decades, rising fossil energy costs, the availability of new technologies, and the desire to use renewable sources have led to increases in the use of wood and other biomass fuels in North America. For example, in Canada, such fuels increased at about 2.4% annually during the 1990s, more than half again as fast as overall energy demand (IEA, 2004).

During this same period, the knowledge of, and consequent concern about, the health effects of air pollution have increased dramatically around the world, leading to stricter air pollution regulation and controls. While commercial sources of wood combustion have been subject to some regulation in North America and Europe, there are still important unregulated sources of woodsmoke, including household heating stoves and fireplaces. The latter have been the target of local ordinances in a number of areas where woodsmoke dominates outdoor air pollution during some seasons. To attain standards for such important pollutants as fine particles ($PM_{2.5}$ or particulate matter less than 2.5 μm in diameter), however, additional controls of these household sources in more areas may be needed.

There are also important nonpoint sources of woodsmoke, particularly wildland fires and intentional burning of agricultural waste. The apparent increases of accidental wildfires in some areas may be due to forest management practices, climate change, and the rise in human population density near fire-prone areas. In addition, the practice of clearing forested areas through the use of fire has resulted in several spectacular long-burning conflagrations in Southeast Asia and elsewhere, which have resulted in a growing concern about the potential health impacts of such events.

The sentiment that woodsmoke, being a natural substance, must be benign to humans is still sometimes heard. It is now well established, however, that wood-burning stoves and fireplaces as well as wildland and agricultural fires emit significant quantities of known health-damaging pollutants, including several carcinogenic compounds (e.g., polycyclic aromatic hydrocarbons, benzene, aldehydes, respirable particulate matter, carbon monoxide [CO], nitrogen oxides [NO_x], and other free radicals) (Tuthill, 1984; Koenig & Pierson, 1991; Larson and Koenig, 1994; Leonard et al., 2000; Dubick et al., 2002; Smith, 1987; Traynor et al., 1987). Many of these toxic pollutants present in woodsmoke are listed in Table 1.

Two of the principal gaseous pollutants in woodsmoke, CO and NO_x, add to the atmospheric levels of these regulated gases emitted by other combustion sources. Health impacts of exposures to these gases and some of the other wood smoke constituents (e.g., benzene) are well characterized in thousands of publications. As these gases are indistinguishable no matter where they come from, there is no urgent need to examine their particular health implications in woodsmoke. There are reasons, however, why woodsmoke may be a special case requiring separate health evaluation.

1. At the point of emissions, woodsmoke contains a vast array of solid, liquid, and gaseous constituents that change, sometimes rapidly, with time, temperature, sunlight, and interaction with other pollutants, water vapor, and surfaces. Many constituents are known to be hazardous to human health, but are not specifically regulated or even fully evaluated. Current methods of health-effects assessment do poorly in estimating impacts by summing the effects of separate constituents. The best approach, therefore, is to examine the toxicity of the entire mixture, as has been done with the most well-studied biomass smoke, that from tobacco burning. Although there have been more than 4000 compounds identified

*Here, we use the term "smoke" to refer to the entire mixture of gases, solid particles, and droplets emitted by combustion.

TABLE 1
Major health-damaging pollutants from biomass combustion

Compound	Examples[a]	Source	Notes	Mode of toxicity
Inorganic gases	*Carbon monoxide (CO)*	Incomplete combustion	Transported over distances	Asphyxiant
	Ozone (O_3)	Secondary reaction product of nitrogen dioxide and hydrocarbons	Only present downwind of fire, transported over long distances	Irritant
	Nitrogen dioxide (NO_2)	High-temperature oxidation of nitrogen in air, some contribution from fuel nitrogen	Reactive	Irritant
Hydrocarbons	Many hundreds	Incomplete combustion	Some transport—also react to form organic aerosols. Species vary with biomass and combustion conditions	
	Unsaturated: 40+, e.g., *1,3-butadiene*			Irritant, carcinogenic, mutagenic
	Saturated: 25+, e.g., n-*hexane*			Irritant, neurotoxicity
	Polycyclic aromatic (PAHs): 20+, e.g., *benzo[a] pyrene*			Mutagenic, carcinogenic
	Monoaromatics: 28+, e.g., *benzene, styrene*			Carcinogenic, mutagenic
Oxygenatated organics	Hundreds	Incomplete combustion	Some transport—also react to form organic aerosols. Species vary with biomass and combustion conditions	
	Aldehydes: 20+, e.g., *acrolein*, *formaldehyde*			Irritant, carcinogenic, mutagenic
	Organic alcohols and acids: 25+, e.g., *methanol* acetic acid			Irritant, teratogenic
	Phenols: 33+, e.g., *catechol, cresol (methylphenols)*			Irritant, carcinogenic, mutagenic, teratogenic
	Quinones: *hydroquinone,* fluorenone, anthraquinone			Irritant, allergenic, redox active, oxidative stress and inflammation, possibly carcinogenic
Chlorinated organics	*Methylene chloride*, *methyl chloride*, *dioxin*	Requires chlorine in the biomass		Central nervous system depressant (methylene chloride), possible carcinogens

(*Continued on next page*)

TABLE 1
Major health-damaging pollutants from biomass combustion *(Continued)*

Compound	Examples[a]	Source	Notes	Mode of toxicity
Free radicals	Semiquinone type radicals	Little is known about their formation		Redox active, cause oxidative stress and inflammatory response, possibly carcinogenic
Particulate matter (PM)	*Inhalable particles (PM_{10})*	Condensation of combustion gases; incomplete combustion; entrainment of vegetation and ash fragments	Coarse[b] + fine particles. Coarse particles are not transported far and contain mostly soil and ash	Inflammation and oxidative stress, may be allergenic
	Respirable particles	Condensation of combustion gases; incomplete combustion	For biomass smoke, approximately equal to fine particles	[See below]
	Fine particles ($PM_{2.5}$)	Condensation of combustion gases; incomplete combustion	Transported over long distances; primary and secondary production[c]	Inflammation and oxidative stress, may be allergenic

[a]Compounds in italics either are criteria air pollutants or are included on the list of hazardous air pollutants specified in Section 112 of the U.S. Clean Air Act. At least 26 hazardous air pollutants are known to be present in woodsmoke.

[b]Coarse particles are defined as those between 2.5 and 10 μm in size.

[c]Particles are created directly during the combustion process and also formed later from emitted gases through condensation and atmospheric chemical reactions.

in tobacco smoke, many dozens of which possess toxic properties, there are few well-understood links between individual constituents and many of the health effects known to be caused by exposure to this mixture.

The first question we address, therefore, is *whether separate regulation/management of woodsmoke should be considered*, even though some of its separate constituents are already regulated in many jurisdictions.

2. Fine particles are thought to be the best single indicator of the health impacts of most combustion sources. Although woodsmoke particles are usually within the size range thought to be most damaging to human health, their chemical composition is different from those derived from fossil fuel combustion, on which most health-effects studies have focused. Because their composition differs from those produced by fossil fuel combustion, woodsmoke particles may not produce the same health effects per unit mass as other combustion particles. Currently, however, except for size, national regulations and international guidelines do not distinguish particles by composition.

The second question we address, therefore, is *whether woodsmoke particles pose different levels of risk than other ambient particles of similar size.*

To address these two key questions, we examine several topics:

- The chemical and physical nature of woodsmoke.*
- The exposures and epidemiology of smoke from wildland fires and agricultural burning, and related controlled human laboratory exposures to biomass smoke.
- The epidemiology of outdoor and indoor woodsmoke exposures from residential woodburning in developed countries.
- The toxicology of woodsmoke, based on animal exposures and laboratory tests.

A short summary of the exposures and health effects of biomass smoke in developing countries is provided as an additional line of evidence. At the end we provide recommendations for additional woodsmoke research.

*Although "woodsmoke" is the substance of primary interest in this report, evidence related to smoke from other biomass (agricultural residues, grass, etc.) is also examined where relevant.

Although cancer-related epidemiology and toxicology are discussed, we do not attempt a judgment because the International Agency for Research on Cancer (IARC) has just completed its Monograph #95, which includes an assessment of the carcinogenicity of household biomass fuel combustion. It was categorized as Category 2A, probably carcinogenic in humans, with limited human evidence although supporting animal and mechanistic evidence (Straif et al., 2006).

BRIEF SUMMARY OF METHODS

The authors searched available biomedical and scientific literature databases in English for articles dealing with controlled human exposure, occupational, and epidemiologic health-effects studies, and toxicologic investigations dealing with woodsmoke; biomass smoke; forest, vegetation, and wildland fires; agricultural burning; and related terms under developed-country conditions. Because of the scattered nature of the literature, however, each author also used his or her knowledge of the literature to identify other papers that did not show up in searches and material in the gray literature. We believe that the result is a nearly complete review of the major relevant publications on these subjects and that there was no bias in selecting papers to review, although we were not able to apply specific inclusion/exclusion criteria.

We did not attempt to search for or review all the literature on the physical and chemical nature of woodsmoke, its environmental concentrations and human exposures, or its health effects in developing-country conditions, such as indoor burning for cooking. In these arenas, we only try to summarize major findings by others.

CHEMICAL COMPOSITION OF BIOMASS SMOKE

Wood consists primarily of two polymers: cellulose (50–70% by weight) and lignin (approximately 30% by weight) (Simoneit et al., 1998). Other biomass fuels (e.g., grasses, wheat stubble) also contain these polymers, although their relative proportions differ. In addition, small amounts of low-molecular-weight organic compounds (e.g., resins, waxes, sugars) and inorganic salts are also present in wood. During combustion, pyrolysis occurs and the polymers break apart, producing a variety of smaller molecules. Biomass combustion is typically inefficient, and a multitude of partially oxidized organic chemicals are generated in biomass smoke. Biomass smoke contains a large number of chemicals, many of which have been associated with adverse health impacts. The major health-damaging particulate and gaseous chemicals present in biomass smoke are listed in Table 1, along with some of their main modes of toxic action.

Tables 2 and 3 summarize the major chemical classes detected in woodsmoke; detailed chemical speciation of the several hundred individual compounds that have been detected in smoke samples is reported in the original references (Rogge et al., 1998; Schauer et al., 2001; Fine, et al., 2002; McDonald et al., 2000; Oros & Simoneit, 2001). The studies cited in Tables 2 and 3 by Rogge et al., Schauer et al., Fine et al., and McDonald et al. all attempted to recreate conditions of residential wood combustion. In contrast, the studies by Oros et al. aimed at being more representative of wildfire emissions. More recently, Lee et al. have also described comprehensive chemical composition of smoke from prescribed burns (Lee & Baumann, 2005). Although less well characterized, a similar mixture of chemicals is reported in smoke emissions from other types of biomass, including grasses, rice straw, sugarcane, and ferns (Simoneit et al., 1993, 1998; Rinehart et al., 2002).*

TABLE 2
Fine particle emissions and bulk chemical composition in woodsmoke

Compound class	Concentration	References
Fine particle emissions rate (g/kg of wood burned)	1.6–9.5	(Schauer et al., 2001; Fine et al., 2002; McDonald et al., 2000)
Organic carbon (wt% of fine particle mass)	12–101	(Schauer et al., 2001; Fine et al., 2002; McDonald et al., 2000)
Elemental carbon (wt% of fine particle mass)	0.65–79	(Schauer et al., 2001; Fine et al., 2002; McDonald et al., 2000)
Ionic species (wt% of fine particle mass)	0.014–1.7	(Schauer et al., 2001; Fine et al., 2002; McDonald et al., 2000)
Elemental species (wt% of fine particle mass)[a]	0.01–4.0	(Schauer et al., 2001; Fine et al., 2002; McDonald et al., 2000)

[a]Chloride included as an element.

In general, it is difficult to make quantitative comparisons among emission factors for specific organic compounds reported by different authors. This is because many of the reports are semiquantitative and the analytical methods used were not comprehensively validated for each analyte, authentic standards were frequently not available to calibrate instrument response, variable combustion conditions (fuel type, moisture content, combustion device) were used, and emission factors were reported in a variety of units.

*It should be noted that most studies have used gas chromatography/mass spectrometry (GC/MS) to characterize the chemical content of woodsmoke. GC is a very efficient tool for separating complex mixtures of organic chemicals. Combined with MS, the technique allows for highly sensitive, specific and accurate detection and quantification of a range of organic chemicals in environmental samples. GC/MS fails to detect compounds that are nonvolatile or thermally labile, however. The application of novel methods, such as liquid chromatography–mass spectrometry (LC/MS), that are appropriate for analysis of nonvolatile or thermally labile compounds will further expand the list of chemicals known to be present in biomass smoke.

TABLE 3
Emissions by chemical class for particle and vapor constituents in woodsmoke

Chemical	Particle-phase (mg/kg wood burned)	References	Vapor-phase (mg/kg wood burned)	References
Carbon monoxide	—		130,000	(McDonald et al., 2000)
		Hydrocarbons		
Alkanes (C2–C7)	0.47–570	(Rogge et al., 1998; Fine et al., 2002)	1.01–300	(Schauer et al., 2001; McDonald et al., 2000)
Alkenes (C2–C7)	0.58–280	(Rogge et al., 1998; Fine et al., 2002)	92–1300	(McDonald et al., 2000)
Polycyclic aromatic hydrocarbons (PAHs) and substituted PAHs	5.1–32,000	(Oros & Simoneit, 2001; Fine et al., 2002; Rogge et al., 1998; McDonald et al., 2000)	43.4–355	(Schauer et al., 2001; McDonald et al., 2000)
Methane	—		4100	(Schauer et al., 2001)
Total nonmethane hydrocarbons C2–C7	[Included in vapor phase]		390–4000	(Schauer et al., 2001; McDonald et al., 2000)
Unresolved complex mixture (UCM)	300–1,130,000	(Oros & Simoneit, 2001; Fine et al., 2002)		
		Oxygenated organics		
Alkanols	0.24–5400	(Oros & Simoneit, 2001; Fine et al., 2002)	120–9200	(McDonald et al., 2000)
Carboxylic acids	6200–755,000	(Oros & Simoneit, 2001; Fine et al., 2002; Rogge et al., 1998)	2.4	(Schauer et al., 2001)
Aldehydes and ketones	[Included in vapor phase]		0.94–4450	(Rogge et al., 1998)[a] (Schauer et al., 2001; Fine et al., 2002; McDonald et al., 2000)
Alkyl esters	0.37–4450	(Oros & Simoneit, 2001; Fine et al., 2002)		
Methoxylated phenolic compounds	28–1000	(Rogge et al., 1998; Fine et al., 2002; McDonald et al., 2000)	1200–1500	(Schauer et al., 2001)
		Other organics		
Other substituted aromatic compounds	5.0–120,000	(Oros & Simoneit, 2001; Fine et al., 2002; Rogge et al., 1998)	110–3600	(Schauer et al., 2001; McDonald et al., 2000)
Sugar derivatives	1.4–12600	(Oros & Simoneit, 2001; Fine et al., 2002)		
Coumarins and flavonoids	0.71–12	(Fine et al., 2002)		
Phytosteroids	1.7–34.0	(Rogge et al., 1998; Fine et al., 2002)		
Resin acids and terpenoids	1.7–41,000	(Oros & Simoneit, 2001; Fine et al., 2002; Rogge et al., 1998)	21–430	(McDonald et al., 2000)
Unresolved compounds	1.2–120	(Fine et al., 2002)	20–600	(Schauer et al., 2001; McDonald et al., 2000)

[a]Only aldehydes reported.

Woodsmoke particles are generally smaller than 1 μm, with a peak in the size distribution between 0.15 and 0.4 μm (Kleeman et al., 1999; Hays et al., 2002). As with other combustion mixtures, such as diesel and tobacco smoke, fresh woodsmoke contains a large number of ultrafine particles, less than 100 μm, which condense rapidly as they cool and age. Indeed, most of the particle mass in aged woodsmoke has been formed by such condensation processes. Fine particles in this size range efficiently evade the mucociliary defense system and are deposited in the peripheral airways, where they may exert toxic effects. Particles in this size range are not easily removed by gravitational settling and therefore can be transported over long distances (Echalar et al., 1995). The transport of biomass combustion particles over hundreds of kilometers has been extensively documented (Andrae et al., 1988). Haze layers with elevated concentrations of CO, carbon dioxide (CO_2), ozone (O_3), and nitric oxide (NO) have been observed. During transport, many of the gaseous species are converted to other gases or into particles. The "black carbon" from biomass emissions is now thought to contribute to regional and global climate change as well as adverse health effects in some parts of the world (Venkataraman et al., 2005; Koch & Hansen, 2005).

Although approximately 5–20% of woodsmoke particulate mass consists of elemental carbon, the composition of the organic carbon fraction varies dramatically with the specific fuel being burned and with the combustion conditions. Detailed analysis of organic woodsmoke aerosol were conducted by Rogge et al. (1998), who measured nearly 200 distinct organic compounds, many of them derivatives of wood polymers and resins (Rogge et al., 1998). Since profiles of specific polycyclic aromatic hydrocarbons (PAHs) are likely to vary, many measurements have focused on benzo[a]pyrene (BaP), a probable human carcinogen.

A number of toxic or carcinogenic compounds are present in biomass smoke, including free radicals, PAHs, and aldehydes, as shown in Table 1 (Leonard et al., 2000; Pryor, 1992; Schauer et al., 2001). Organic extracts of ambient particulate matter (PM) containing substantial quantities of woodsmoke are 30-fold more potent than extracts of cigarette smoke condensate in a mouse skin tumor induction assay (Cupitt et al., 1994), and are mutagenic in the *Salmonella typhimurium* microsuspension and plate incorporation assays (Claxton et al., 2001). Few, if any, reports exist in which the toxicity of smoke from different biomass sources was compared and related to differences in the chemical composition of each smoke type.

Woodsmoke is enriched with several chemicals relative to pollutant mixtures from other sources of air pollution. Examples include potassium, methoxyphenols, levoglucosan, retene, and specific resin acids (e.g., abietic acid) (Khalil & Rasmussen, 2003; Fine et al., 2001, 2002; Schauer et al., 2001; Rogge et al., 1998; Hawthorne et al., 1992). Many of these chemicals have been used either individually or in multivariate analyses to quantify woodsmoke emissions (Khalil & Rasmussen, 2003; Schauer & Cass, 2000; Larsen & Baker, 2003).

Levoglucosan is sugar anhydride derived from the pyrolysis of the major wood polymer cellulose. Levoglucosan is one of the most abundant organic compounds associated with particles in woodsmoke (Fine et al., 2001, 2002). It is stable in the environment and has been used extensively to estimate woodsmoke levels in ambient PM samples (Schauer & Cass, 2000; Katz et al., 2004; Larson et al., 2004). Levoglucosan is present in other biomass smoke samples, including smoke from tobacco, grasses, and rice straw (Sakuma & Ohsumi, 1980; Simoneit, et al., 1993). Under conditions in which woodsmoke dominates the biomass smoke contribution to ambient aerosol, however, levoglucosan can be considered a unique tracer for woodsmoke (Schauer & Cass, 2000).

Methoxyphenols are a class of chemicals derived from the pyrolysis of the wood polymer lignin. This class of chemicals spans a range of volatilities from relatively volatile (e.g., guaiacol) to exclusively particle-associated (e.g., sinapinaldehyde). These chemicals are relatively abundant in woodsmoke, although the most abundant compounds are predominantly in the vapor phase (Hawthorne et al., 1989; Schauer et al., 2001). Accurate chemical analysis of the methoxyphenols, however, has proved to be an analytical challenge, and many of the methoxyphenols were found to be chemically reactive—a property that would undermine their suitability as tracers for biomass smoke (Simpson et al., 2005). Methoxyphenols have been used as woodsmoke tracers in multivariate source apportionment models to determine the proportion of urban fine PM derived from woodburning (Schauer & Cass, 2000).

The organic chemical composition has been used to distinguish smokes from different biomass fuels. Smoke from hardwood versus softwood burning can be distinguished by the relative proportions of substituted guaiacols compared to syringols (Hawthorne et al., 1989; Oros & Simoneit, 2001; Schauer & Cass, 2000). Mono- and dimethoxyphenols are also present in small amounts in grass and grain smokes, but the major phenolic compounds in grass smoke are *p*-coumaryl derivatives (Simoneit, et al., 1993). Diterpenoids (e.g., dehydroabietic acid) are abundant in smoke from gymnospems (conifers) compared to angiosperms (Schauer et al., 2001). Certain chemicals may even be unique to smoke from specific tree species (e.g., juvabione from balsam fir), although the atmospheric stability of such compounds and hence their utility as source-specific exposure markers has not been established (Fine et al., 2001; Oros & Simoneit, 2001).

Emission factors for fine particles are highly dependent on the fuel characteristics and burn conditions (smoldering vs. flaming). Similarly, emission factors for specific organic chemicals are influenced by fuel moisture content and burn conditions, although the relationships may not parallel those observed for fine particles (Khalil & Rasmussen, 2003; Guillen & Ibargoitia, 1999).

FOREST FIRE AND AGRICULTURAL BURNING: EXPOSURE AND HEALTH STUDIES

In contrast to the large amount of information relating urban PM to human health impacts, there is only a limited number of studies directly evaluating the community health impacts of air pollution resulting from the burning of biomass. Several reviews have discussed the health impacts and pollutants associated with woodsmoke air pollution (Larson & Koenig, 1994; Pierson et al., 1989; Vedal, 1993; Boman et al., 2003, 2006). Although the emphasis of these reviews was on community exposures resulting from burning of wood in fireplaces and wood stoves, many of the conclusions are relevant to the broader understanding of vegetation fire air pollution. The World Heath Organization (WHO) has published a document describing Health Guidelines for Vegetation Fire Events,* which also contains a review of evidence linking air pollution from vegetation fires with human health effects. Specific information relating agricultural and forest/brush burning with human health effects is summarized next and presented in Table 4.

On a regional basis, during vegetation fire episodes PM is the air pollutant most consistently elevated in locations impacted by fire smoke (Sapkota et al., 2005). For example, during fires in southern California, PM_{10} concentrations were 3–4 times higher than during nonfire periods, while particle number, and CO and NO concentrations were increased by a factor of 2. The concentrations of NO_2 and O_3 were essentially unchanged or even lower (Phuleria et al., 2005). Further, measurements indicate that that biomass combustion emissions can be transported over hundreds of kilometers such that local air quality is degraded even at great distances from fire locations (Sapkota et al., 2005). Smoke from African and Brazilian savanna fires has been shown to contain substantial quantities of fine particles (Artaxo et al., 1991; Echalar et al., 1995). Mass concentrations ranged from 30 $\mu g/m^3$ in areas not affected by biomass burning to 300 $\mu g/m^3$ in large areas (2 million km^2) with intense burning. Additional studies of fine particle (<2 μm) composition associated with biomass burning in the Amazon Basin was reported by Artaxo et al. (1994), who found 24-h average PM_{10} and $PM_{2.5}$ mass concentrations as high as 700 and 400 $\mu g/m^3$, respectively (Artazo et al., 1994). In one of the few measurements of rural community air pollution associated with large tropical forest fires, Reinhardt and Ottmar measured formaldehyde, acrolein, benzene, CO, and respirable PM ($PM_{3.5}$) in Rondonia, Brazil, during the peak of the 1996 biomass burning season (Reinhardt et al., 2001). Of the species measured, respirable particle levels were elevated 5-10 times above background, with mean levels of 190 $\mu g/m^3$ and levels as high as 250 $\mu g/m^3$measured during several of the 12-h sampling periods. The mean CO level was 4 ppm, which is similar to levels measured in moderately polluted urban areas, but below the level expected to be associated with acute health impacts. Benzene levels (11 $\mu g/m^3$ average) were higher than those measured in other rural areas and were comparable to those measured in cities.

Measurements from Southeast Asia also indicate that particles are the main air pollutant elevated during periods of vegetation fire-related air pollution (Radojevic & Hassan, 1999). For example, during a 2- to 3-mo period in 1994, 24-h PM_{10} levels up to 409 $\mu g/m^3$ were recorded in Kuala Lumpur (Hassan et al., 1995), and levels ranged from 36 to 285 $\mu g/m^3$ (unspecified average time) in Singapore (Nichol, 1997). In a 1997 vegetation fire episode, PM_{10} levels as high as 930 and 421 $\mu g/m^3$ were measured in Sarawak (Malaysia) and Kuala Lumpur, respectively, while 24-h levels in Singapore and southern Thailand were somewhat lower (Brauer, 1998). Closer to the fire source in Indonesia, PM_{10} concentrations as high as 1800 $\mu g/m^3$ were measured over an unspecified period (Kunii et al., 2002). In February–May 1998 a more limited vegetation fire episode affected regions of Borneo. In Brunei, 24-h PM_{10} levels as high as 440 $\mu g/m^3$ were measured during this period (Radojevic & Hassan, 1999).

Wildland Firefighters

In general, wildland firefighters experience greater exposure from forest fire smoke than members of the general public. Patterns of exposure can be intense in initial fire-suppression efforts or in situations involving thermal inversions. Workshifts are frequently 12 to 18 h and can last for more than 24 h. In large fires, prolonged work shifts can last for many days. In wildland firefighting, it is not feasible to use a self-contained breathing apparatus; often the only respiratory protection used is a cotton bandana tied over the nose and mouth. Moreover, many of the tasks in wildland firefighting are physically demanding and require elevated pulmonary ventilation rates, which can result in substantial doses of smoke to the respiratory tract. Off-shift smoke exposures may occur as well, depending on the location of the base camp (where firefighters eat and sleep) in relation to the fire and the prevailing meteorology. With the intensity of smoke exposures, it is not surprising that respiratory problems accounted for about 40% of all medical visits made by wildland firefighters during the Yellowstone firestorm of 1988 (U.S. Department of Agriculture, 1989).

There have been several investigations of both exposures and health impacts of smoke exposure among wildland firefighters. Exposure assessment can represent a major logistical challenge, considering that the work often takes place on steep terrain in remote locations and may involve extreme physical exertion. In addition, exposure assessment must of necessity be limited to relatively few of the thousands of substances in biomass smoke. By extension, the few health studies that have been undertaken have not involved concurrent exposure assessment, but have focused on cross-shift or cross-seasonal respiratory effects.

Reinhardt and Ottmar (2000) undertook an exposure assessment of breathing-zone levels of acrolein, benzene, carbon dioxide, CO, formaldehyde, and $PM_{3.5}$ among firefighters at 21 wildfires in California between 1992 and 1995. Interestingly,

*www.who.int/docstore/peh/Vegetation_fires/Health_Guidelines_final_3.pdf

TABLE 4
Summary of selected epidemiologic studies of large-scale vegetation fires

Population	Endpoints measured	Results	Reference
All ages	Emergency room visits	Increased respiratory visits in communities exposed to fire smoke	(Duclos et al., 1990)
All ages	Emergency room visits, hospital admissions	Increased emergency-room visits and hospital admissions for asthma and bronchitis during fire period relative to same period in previous year	(Sorenson et al., 1999)
All ages	Acute respiratory distress hospital visits	Increase in acute respiratory distress inhalation therapy visits associated with indirect measure (sedimentation) of air pollution during sugar-cane burning season in Brazil	(Arbex et al., 2000)
All ages	Outpatient visits	Increased visits for asthma, upper respiratory tract symptoms, and rhinitis during vegetation fire episode periods of elevated PM_{10} in Malaysia	(Brauer, 1998)
All Ages	Outpatient visits, hospital admissions, mortality	Increase in PM_{10} from 50 to 150 $\mu g/m^3$ during vegetation fire episode periods associated with increase in outpatient visits in Singapore for upper respiratory tract symptoms (12%), asthma (37%), and rhinitis (26%). No increase in hospital admissions or mortality	(Emmanuel, 2000)
All Ages	Emergency room visits	Increased asthma visits with PM_{10} during episode of exposure to biomass burning emissions in Singapore	(Chew et al., 1995)
All Ages	Emergency room visits	No increase in asthma visits with PM_{10} during episode of exposure to bushfire emissions in Australia	(Copper et al., 1994)
All Ages	Emergency room visits	No increase in asthma visits with PM_{10} During episode of exposure to bushfire emissions in Australia	(Smith et al., 1996)
All Ages	Emergency room visits	Increased asthma visits associated with PM_{10}, especially for concentrations exceeding 40 $\mu g/m^3$	(Johnston et al., 2002)
All Ages	Physician visits for respiratory, cardiovascular, and mental illness	A 46 to 78% increase in physician visits for respiratory illness during a 3-wk forest fire period in Kelowna, British Columbia	(Moore et al., 2006)
All Ages	Hospital admission for respiratory illness	Daily hospital emission rates for respiratory illness increased with levels of PM_{10} for bushfire and nonbushfire periods	(Chen et al., 2006)
All ages, >65 yr	Mortality	0.7% (all ages) and 1.8% (ages 65–74) increases in adjusted relative risk of nontrauma mortality per 10-$\mu g/m^3$ increase in PM_{10} Kuala Lumpur, Malaysia, for 1996-1997, including vegetation fore episode period	(Sastry, 2002)
Adults with COPD	Symptoms	Significant increase in symptom index (dyspnea, cough, chest tightness, wheezing, sputum production) on two days of elevated PM2.5 (65 $\mu g/m^3$) relative to control days (14 $\mu g/m^3$). Days of elevated PM attributed to fire smoke by satellite imaging	(Sutherland, 2005)
Adults	Asthma medication, lung function, asthmatic and other respiratory symptoms	Increased prevalence of respiratory symptoms and various asthma indicators, decreased lung function post-rice stubble burning period relative to period prior to burning in three communities in Iran	(Golshan et al., 2002)
Adult military recruits	Blood markers of inflammation	Bone marrow stimulated to release immature polymorphonuclear leukocytes into blood during period of exposure to forest fire smoke relative to period following smoke exposure	(Tan et al., 2000)
Children	Respiratory hospital admissions	Increased pediatric respiratory hospital admissions associated with increased biomass smoke markers (potassium and black carbon) during sugar-cane burning season in Brazil	(Cancado et al., 2002)
Children	Lung function	Decreased lung function in children during vegetation fire episode compared to preepisode measurements	(Hisham-Hashim et al., 1998)

exposures to the gases were generally well below time-weighted average occupational health standards. However, some of the fires resulted in high-level peak exposures to heavy smoke. Respirable particle ($PM_{3.5}$) exposures on multiday fires averaged 0.72 mg/m^3 on the fireline, and 0.5 mg/m^3 over the work shift, with peak concentrations of 2.3 and 2.93 mg/m^3. The corresponding exposures to CO were 4.0 and 2.8 ppm, with peak (2-h time-weighted average [TWA]) exposures of 38.8 ppm and 30.5 ppm. The particle concentrations are about 10 to 30 times higher than 24-h average ambient air quality standards for $PM_{2.5}$ (currently 65 μg/m^3 in the United States).

Materna et al. (1992) also found extremely high particle exposures among California wildland firefighters during the 1987–1989 fire seasons. Table 5 presents their data on PM exposures. These investigators also sampled for 12 PAHs and found all below 1 μg/m^3. The highest CO levels were associated with tending gasoline-powered pumping engines rather than from smoke exposure per se. An aldehyde screen detected formaldehyde, acrolein, furfural, and acetaldehyde. Most levels were well below occupational exposure limits; however, formaldehyde (which was detected in all samples) in several instances exceeded such limits (maximum TWA [226 min] = 0.42 mg/m^3). In general, these studies demonstrate that of the various measured constituents of smoke, PM tends to be the most consistently elevated during wildland firefighting in relation to health-based exposure standards.

In the first report of cross-seasonal changes in respiratory symptoms and lung function in wildland firefighters, Rothman et al. (1991) examined 69 Northern California firefighters who were nonsmokers or former smokers who had not smoked in at least 6 mo. There were significant cross-seasonal increases in reported cough, phlegm, wheeze, and eye and nasal irritation. Only eye irritation, however, was significantly associated with firefighting activity ($r = .48$, $p < .001$), while the association of wheeze with firefighting in the last 2 wk of the study was of borderline significance ($r = .25$, $p = .07$). There were small, but statistically significant, declines in several measures of pulmonary function across the season, with the strongest relationships for the highest exposure category in the final week preceding the follow-up spirometry. The associations became weaker and less significant with the progressive inclusion of additional weeks prior to the spirometry. Across the 8-wk study, several lung function metrics exhibited significant declines, including FEV1 (−1.2%, confidence interval [CI]* −0.5, −2.0%), FEV1/FVC (−0.006, CI −0.001, −0.01), and although FVC also declined, this change was not significant. Those in the highest category for hours worked in the week preceding spirometry experienced larger decrements in lung function (FEV1 = −2.9% [130 ml] and FVC = −1.9% [101 ml]). These changes were not affected by adjustment for potential confounders (not specified). The use of a cotton bandana for respiratory protection was not associated with any measurable protection.

TABLE 5
Personal TWA particle exposures among California wildland firefighters

Particle metric	Site/activity	Mean (mg/m^3)	Range (mg/m^3)
TSP	Base camp/waiting in staging area	3.3	1.8–4.4
TSP	Fireline/mop-up	9.5	2.7–37.4
Respirable	Fireline/mop-up	1.8	0.3–5.1
Respirable	Prescribed burn	1.2	0.2–2.7

Note. Modified from Materna et al. (1992).

Liu et al. (1992) examined cross-season changes in pulmonary function and airway hyperresponsiveness in 63 wildland firefighters in northern California and Montana in 1989. They were tested before the start of the fire season and within 2 wk of discharge from service. Though pre- and post-season spirometric measurements were within the normal range for all participants, there were significant cross-seasonal declines in FVC, FEV1, and FEF25–75 of 0.09 L, 0.15 L, and 0.44 L/s, respectively. There was no significant relationship with any of the covariates measured, including smoking status, history of allergy, asthma, or upper/lower respiratory symptoms, specific firefighting crew membership, or seasonal versus full-time employment. Airway responsiveness to methacholine increased significantly across the fire season, which was not affected by gender, history of smoking, allergy, full-time versus seasonal employment, or crew membership. This study suggests that, in addition to persistent cross-seasonal changes in lung function, firefighting may also be associated with increased airway hyperresponsiveness, although the effect was not significant.

Letts et al. (1991) conducted a health survey of 78 wildland firefighters in Southern California. There were no changes in symptom prevalence cross-seasonally, nor were there any significant associations with exposure (defined as low, medium, and high, based on hours of work and weighted by visual estimates of smoke intensity). There were small, nonsignificant changes in FEV1 and FVC. The decrements in FEF25–75 and FEV1/FVC, however, were both significant (−2.3%, CI −4.2, −0.5% and −0.5%, CI −1.0, −0.1%). The changes in FEF25–75 showed a nonsignificant exposure-response trend ($p = 0.08$) of: 0.5%, −1.9%, and −4.7% for the low-, medium-, and high-exposure groups, respectively. Interestingly, however, there were no associations with the number of seasons of firefighting, days since the last fire, or age. Although these investigators concluded that there was limited evidence of cross-seasonal effects of firefighting on lung function, they indicated that the season in which their survey was conducted involved an atypically low number of firefighting hours. Moreover, the baseline was established in June, reportedly "before significant smoke exposure occurred,"

*All confidence intervals reported here are at the 95% level.

though the extent of firefighting preceding the initial measurement was not documented.

In addition to examining cross-seasonal lung function changes, Betchley et al. (1997) also examined cross-shift changes among forest firefighters in the Cascade Mountains of Oregon and Washington (Betchley et al., 1997). Among 76 workers examined at the beginning and immediately after prescribed burns, mean declines in FVC, FEV1, and FEF25–75 were 0.065 L, 0.150 L, and 0.496 L/s, respectively. These changes were significant even after adjusting for respiratory infections in the preceding 4 wk, smoking status, any "lung condition," and allergy. Examining cross-seasonal changes in 53 firefighters, the values for these same measures were 0.033 L, 0.104 L, and 0.275 L/s, respectively. The changes for FEV1 and FEF25–75 were significant, and remained so even after adjustment for the same potential confounders and effect modifiers. There were no significant cross-seasonal changes in respiratory symptoms. The cross-seasonal lung function measurements and symptom reports were taken, on average, 78 days after the last occupational firefighting activities of the season. In a subsequent analysis of a subset of these workers ($n = 65$) who had been working when several combustion products were measured, the lung function decrements observed were not found to be specifically associated with $PM_{3.5}$, acrolein, carbon monoxide, or formaldehyde (Slaughter et al., 2004).

Investigators in Sardinia compared lung function among 92 wildland firefighters with a "control" group of policemen (Serra et al., 1996). The testing was undertaken in late spring, just prior to the onset of the principal fire season. The two groups had identical mean values for FVC and TLC,* and showed no significant differences for FRC,† DLCO, or DLCO‡/TLC. The firefighters, however, demonstrated modestly lower lung function test results for FEV1, FEV1/FVC, FEF50, FEF25, and RV.** Although there were significant differences in age and height between the two groups (the firefighters were older and shorter, both of which would favor lower mean lung function), the significant differences in lung function remained after multivariate control for age, height, smoking status, and pack-year history for current smokers. The investigators found no relationship of pulmonary function with years of service or with the number of fires extinguished over their careers. Cough and expectoration were more common among firefighters, but these differences were not significant.

*Total lung capacity (TLC) is the volume of air contained in the lungs after maximal inhalation.

†Functional residual capacity (FRC) measures the amount of air remaining in the lungs after a normal tidal expiration.

‡Carbon monoxide diffusing capacity (DLCO) provides an assessment of the ability of gases to diffuse across the blood–gas barrier, that is, from the alveoli into the blood.

**Residual volume (RV) is the amount of air remaining in the lungs after a maximal exhalation.

Wildland firefighting can involve intermittent prolonged exposures to high concentrations of respirable particles, which consist of mixtures unique to each situation. Exposures to elevated levels of CO and respiratory irritants such as formaldehyde also occur, but respirable particles probably represent the principal exposure of concern. The few health studies conducted on such workers have documented cross-seasonal decrements in lung function, increased airway hyperresponsiveness, and increased prevalence of respiratory symptoms. Rothman et al. (1991) demonstrated that recent cumulative exposures were more strongly associated with greater changes in lung function than were more remote exposures. At least one study has also shown acute cross-shift spirometric changes as well (Liu et al., 1992). There has been no long-term follow-up of the respiratory health of wildland firefighters, however. Among municipal firefighters, chronic pulmonary dysfunction may result from repeated smoke exposure, particularly among those who do not use respiratory protective devices (Tepper et al., 1991; Sparrow et al., 1982). It is unknown whether cessation of exposure among wildland firefighters during the off-season may allow for recovery and reversibility of effects, in contrast to municipal firefighters, who can be exposed year-round. In any case, the relatively small effects demonstrated among firefighters cannot be quantitatively extrapolated to nonoccupational exposures, as the demands of the job require a degree of physical fitness and resilience far beyond that found in most of the general population.

Forest and Brush Fires

Several studies in North America have evaluated the health impacts associated with forest and brush fires. In the first study examining the effect of wildfire smoke on the general population, Duclos and colleagues evaluated the impact of a numerous large forest fires on emergency room (ER) visits to 15 hospitals in 6 counties in California (Duclos et al., 1990). The authors calculated observed-to-expected ratios of ER visits, based on the numbers of visits during two reference periods. During the approximately $2\frac{1}{2}$-wk period of observation, ER visits for asthma and chronic obstructive pulmonary disease increased by 40% ($p < .001$) and 30% ($p = .02$), respectively. Significant increases were also observed for bronchitis (observed [O]/expected [E] = 1.2, $p = .03$), laryngitis (O/E = 1.6, $p = .02$), sinusitis (O/E = 1.3, $p = .05$), and other upper respiratory infections (O/E = 1.5, $p < .001$). Exposure assessment was problematic, however, as few PM_{10} or other monitors were located downwind of the fires. The highest PM_{10} concentration measured was 237 $\mu g/m^3$. In contrast, several measurements of total suspended particles (TSP) exceeded 1000 $\mu g/m^3$; the highest recorded value was 4158 $\mu g/m^3$. Exposure to forest fire smoke can be unpredictable, changing with wind direction, intensity of the fire, precipitation, and other variables. The few air quality measurements available to these investigators could not serve to reliably characterize population exposures, which is a general limitation of all wildfire studies. In addition, this study was subject to other typical limitations of ER analyses related

to behavioral and economic factors (e.g., perceptions of illness severity, access to other health care providers, and availability of health insurance, with the latter more problematic in the U.S. than elsewhere).

Although no air pollutant concentrations were reported, the impact of wildfires in Florida on ER visits to eight hospitals in 1998 were compared to visits during the same 5-wk period in the previous year. From 1997 to 1998, ER visits increased substantially for asthma (91%), bronchitis with acute exacerbation (132%), and chest pain (37%), while visits decreased for painful respiration (27%) and acute bronchitis (20%). Though based on smaller numbers, there were modest changes in the number of hospital admissions (increases of 46% for asthma and 24% for chest pain) (Sorenson et al., 1999). Although this study suggests that wildfire smoke exposure resulted in increased ER visits for respiratory disease and symptoms, no firm conclusions are possible. There was only one reference period selected, which might not provide a stable basis for comparison, and no statistical testing was undertaken.

In a retrospective evaluation of the health impacts of a large wildfire in a northern California Native American reservation, visits to the local medical clinic for respiratory illness increased by 52% over the same period the prior year (Mott et al., 2002). During the ten weeks that the fire lasted, PM10 levels exceeded 150 μg/m^3 (24-h average) 15 times, and on 2 days the levels exceeded 500 μg/m^3. Weekly concentrations of PM_{10} were strongly correlated with weekly visits for respiratory illness during the fire year ($r = .74$), but not in the prior year ($r = -.63$). In a community survey of 289 respondents, more than 60% reported respiratory symptoms during the smoke episode; 20% reported symptoms persisting at least 2 wk after the smoke cleared. Individuals with preexisting cardiopulmonary diseases reported significantly more symptoms before, during, and after the fire than those without such illnesses. The investigators also retrospectively evaluated the efficacy of several public health interventions in symptom reduction: (1) filtered and unfiltered masks distributed free of charge; (2) vouchers for free hotel accommodations in towns away from the smoke to assist evacuation efforts; (3) high-efficiency particulate air (HEPA) cleaners distributed for residential use; and (4) public service announcements (PSAs) about exposure reduction strategies. Mott and colleagues found that increased duration of use of a residential HEPA air cleaner was associated with decreased odds of reporting increased symptoms (odds ratio [OR] 0.54, CI 0.32, 0.89), with an inverse trend of symptom reporting with increasing duration of use. Similarly, ability to accurately recall a PSA was also associated with reduced odds for respiratory symptoms. In contrast, there was no detectable beneficial effect of evacuation from smoky areas or of the use of masks. However, the timing and duration of evacuation were not optimal. On the days with the highest recorded smoke concentrations, over 80% of the subjects had not evacuated. That mask use was not protective is not surprising; the masks were distributed without fit testing and had variable filtration efficiencies. Moreover, none of the interventions was randomized, and in fact individuals with smoke-related health effects or a prior diagnosis of respiratory or cardiovascular disease were given priority to receive hotel vouchers and HEPA air cleaners. Finally, due to the retrospective nature of the investigation, recall bias may have affected the results based on the survey.

More recently, Sutherland and colleagues reported an increase in an index of respiratory symptoms (dyspnea, cough, chest tightness, wheezing, and sputum production) among a panel of 21 subjects with COPD associated with 2 days of elevated ambient particle levels resulting from a forest fire near Denver, CO. On the 2 days in which symptom scores were increased, average $PM_{2.5}$ concentrations increased to 63 μg/m^3 relative to an average of 14 μg/m^3 on control days (Sutherland et al., 2005). During this same fire as well as several other fires in Colorado, the indoor infiltration of particulate matter was measured and the effectiveness of room HEPA-filter air cleaners was assessed in a total of eight homes. A decrease in $PM_{2.5}$ concentrations of 63–88% was measured in homes in which air cleaners were operated, relative to homes without air cleaners. In the homes without the air cleaners, measured indoor $PM_{2.5}$ concentrations were 58–100% of the concentrations measured outdoors (Henderson et al., 2005).

Moore and colleagues assessed the impact of elevated concentrations of $PM_{2.5}$ associated with forest fires on outpatient physician visits for respiratory disease. Two large fires burning adjacent to urban areas in British Columbia, Canada, resulted in intermittent elevations (140–200 μg/m^3) in daily average $PM_{2.5}$ concentrations over a 5-wk period in August and September 2003. In the city with the highest levels of PM and that was closest to a fire, weekly physician visits for respiratory disease were increased approximately 45–80% relative to average rates corresponding to those weeks during the previous 10 yr. No statistically significant increases were observed in the city with lower fire-related PM increases and neither city experienced elevated physician visits for cardiovascular diseases (Moore et al., 2006). However, as many patients experiencing symptoms characteristic of acute cardiovascular events go directly to a hospital emergency department, it is possible that the health database used in this investigation may not have been capable of identifying circulatory outcomes of interest during the study period.

During 1994, bush fires near Sydney, Australia led to elevated PM_{10} levels (maximum hourly values of approximately 250 μg/m^3) for a 7-day period. Two studies of asthma emergency room visits during the bushfire smoke episode failed to detect any association with air pollution (Copper et al., 1994; Smith et al., 1996). The report by Copper et al. (1994) was in the form of a letter to *The Lancet*, with few details provided. The investigators examined only three inner-city hospitals, preferring to avoid the influence of "patients who presented with direct effects of smoke inhalation," which might have occurred had they included hospitals with catchment areas closer to the fires. They compared the numbers of asthma ER visits for the week before the bushfires (January 1–8), the fire period (January

9–20), and afterward (January 21–31), and found no difference among the 3 periods. These comparisons were based on relatively small numbers, however, with fewer than 100 visits for asthma during the entire month for all 3 hospitals. The report by Smith et al. (1996) involved a comparison of the proportions of asthma to total ER visits to seven hospitals during the week of high smoke levels compared to the same week the prior year. There was no difference in these proportions, nor was there a relationship between the maximum daily nephelometric particle measurement and the number of asthma ER visits in multiweek regression models. Although it appears that the bushfire smoke did not have an impact on asthma ER visits, this study is limited by the use of a single reference period. In addition, the regression analysis is likely to have had very limited statistical power, with relatively few days of observation.

A recent analysis of these same fires and lung function (measured as peak expiratory flow rate [PEFR]) did not detect any association between either PM_{10} levels or an indicator variable representing the fire period and evening PEFR in 25 asthmatic children, although 20 children without airway hyperreactivity showed a significant decrease in PEFR with increasing same-day PM_{10} concentrations (Jalaludin et al., 2000). Whether this represents a true lack of association or an artifact of experimental design is difficult to ascertain. Thirty-two children in this analysis were recruited during the week of the fire. There did not seem to be any examination of whether there was a learning period for these children (during which the initial PEFR measurements might have been more variable), nor was there any discussion of the quality control for recording the measurements, or even what the PEFR protocol was. Of the 32 children (mean age = 9.2 yr), 25 had a physician's diagnosis of asthma; however, only 12 of the 32 had evidence of airway hyperresponsiveness, which is considered a hallmark of asthma. Although the regression model included indicator variables for use of asthma medications, there could nonetheless still have been residual confounding by medication use. In other words, the use of asthma medications might still have had enough of an effect on lung function to obscure a relationship between PEFR and smoke exposure, despite the attempt to control for this influence statistically. Finally, due to the timing of subject recruitment, it is not clear how many child-days of observation during the fires actually contributed to the analysis. The reported data suggest that this study is likely to have had very limited statistical power.

The results from these studies appear to conflict with those conducted in North America. As noted earlier, however, all have significant limitations that suggest caution in generalizing the results. It is also possible that there is less respiratory toxicity from bushfire smoke than from forest fire smoke due to chemical and physical differences between the two. Two more recent studies from Australia have reported associations between bushfire smoke and health impacts. For example, a study undertaken at the only hospital in Darwin (northwestern Australia) evaluated the association between daily asthma ER visits (adjusted for influenza and day-of-week effects) and measured PM_{10} over a 7-mo period, which included 2 bushfire smoke episodes. Bushfires represent the principal regional source of significant levels of air pollution in Darwin during the dry season in which this investigation took place. Increased asthma visits were associated with PM_{10} concentrations, especially for days on which PM_{10} concentrations exceeded 40 $\mu g/m^3$ (Johnston et al., 2002). The adjusted rate ratio per 10-$\mu g/m^3$ increase in PM_{10} was 1.20 (CI 1.09, 1.34). The largest association was observed for a 5-day lag, comparing days when PM_{10} exceeded 40 $\mu g/m^3$ with those on which PM_{10} was less than 10 $\mu g/m^3$ (adjusted rate ratio = 2.56 [CI 1.60, 4.09]). Unlike the prior studies of biomass smoke conducted in Australia, this investigation clearly had adequate statistical power to detect an association between PM and asthma visits. Though the time-series analysis did not control for pollen or mold, which are not routinely monitored in Darwin, the investigators considered it "extremely unlikely" that either of these would vary systematically with bushfire smoke. This assessment by the authors is probably true, but without analyzing the smoke for these bioaerosols, it is not possible to state definitively that they did not confound the results.

Chen et al. (2006) evaluated the relationship between respiratory hospital admissions in Brisbane, Australia, and particulate matter (PM_{10}) for a $3\frac{1}{2}$-yr period that included 452 days (35% of the study period) categorized as days with bushfires (>1 ha burned) in the study region, based on review of fire records. During the bushfire periods, the median of daily respiratory hospital admissions in Brisbane was 34 (range: 9–76) and the daily mean PM_{10} was 18.3 $\mu g/m^3$ (range: 7.5–60.6 $\mu g/m^3$), compared to a median of 32 respiratory hospital admissions per day (range: 7–91) and daily mean PM_{10} of 14.9 $\mu g/m^3$ (range: 4.9–58.1 $\mu g/m^3$) during non-bushfire days. The authors categorized PM_{10} values into low (<15), medium (15–20) and high (>20), rather than using a continuous variable for PM_{10}. This may have resulted in a loss of information about the potential impacts of extreme values and possibly a bias toward (or away from) the null hypothesis of no effect (Dosemeci et al., 1990). In addition, the authors noted that the single PM_{10} monitor used in this study was upwind of many of the fires, indicating that the populations affected were exposed to higher PM_{10} and smoke concentrations than those reported, which could have resulted in an overestimate of the magnitude of effect. Nonetheless, for both bushfire and nonbushfire periods, increased PM_{10} concentrations were associated with increased relative risks for respiratory hospital admissions, with some suggestion of slightly stronger associations on the days with the highest daily PM_{10} concentrations (i.e., >20 $\mu g/m^3$) on bushfire (RR = 1.19, CI 1.09, 1.30, for same-day PM_{10} concentrations) versus nonbushfire (RR = 1.13, CI 1.06, 1.23) days. The results of this study are consistent with many other time-series investigations of PM and, at a minimum, indicate that the associations between PM_{10} and respiratory health admissions on bushfire days were at least as great as those on days when other sources of PM_{10} predominated.

Major regional episodes of air pollution from vegetation fires in Southeast Asia have been the subject of several investigations

and surveillance programs. An analysis of emergency room visits for asthma in Singapore during a 1994 episode of regional pollution resulting from forest and plantation fires reported an association between PM_{10} and emergency room visits for childhood asthma. During the "haze" period, mean PM_{10} levels were 20% higher than the annual average. Although a time-series analysis was not conducted, the authors suggested that the association remained significant for all concentrations above 158 $\mu g/m^3$ (Chew et al., 1995).

Reports from surveillance monitoring activities conducted during major Southeast Asian episodes in 1997 and 1998 also indicated effects on health care utilization. In Singapore, for example, there was a 30% increase in hospital attendance for "haze-related" illnesses: A time-series analysis indicated that a PM_{10} increase of 100 $\mu g/m^3$ was associated with 12%, 19%, and 26% increases in cases of upper respiratory tract illness, asthma, and rhinitis, respectively. It is not clear why rhinitis constituted a separate diagnostic category in this investigation, rather than being included with upper respiratory tract illness. This analysis did not observe any significant increases in hospital admissions or mortality (Emmanuel, 2000). Similar findings were also observed in Malaysia (Brauer, 1998; Leech et al., 1998).

Preliminary results from a study of 107 Kuala Lumpur schoolchildren found statistically significant decreases in lung function between preepisode measurements in June–July 1996 and measurements conducted during the haze episode in September 1997 (Hisham-Hashim et al., 1998). A convenience sample questionnaire survey conducted in Indonesia during the 1997 haze episode also suggested acute impacts on respiratory and cardiovascular symptoms (Kunii et al., 2002). Of 539 interviewees, 91% reported respiratory symptoms (cough, sneezing, runny nose, sputum production, or sore throat), 44% reported shortness of breath on walking, 33% reported chest discomfort, and 23% reported palpitations. Although the numbers were small, respondents with asthma or heart disease tended to experience a greater proportion of moderate and severe symptoms relative to those without preexisting disease. Despite these findings, however, the cross-sectional nature of the sampling and reporting and the absence of an unexposed reference population weaken any inference of a causal relationship between the smoke and these symptoms.

In another study of the 1997 Southeast Asia haze episode, Tan and colleagues (2000) obtained blood samples at weekly intervals from 30 Singaporean military recruits who followed standardized outdoor routines during the episode. The mean 24-h PM_{10} level during the episode was 125.4 $\mu g/m^3$. Analyzing the numbers of immature inflammatory cells (polymorphonuclear cells or PMNs) in the subjects' blood in relation to daily measures of several pollutants, these investigators found the strongest relationship with same-day PM_{10}, though a 1-day lag of this metric was also statistically significant. Although these results are insufficient to establish a causal relationship, they suggest that smoke inhalation stimulated the bone marrow to eject immature PMNs into the circulation.*

Recently, Mott et al. reported several related examinations of the Indonesian fires on hospitalizations and survival (Mott et al., 2005). In analyses of the fire period (August through October 1997) compared with a 31-mo baseline period (January 1995 through July 1997), they reported fire-related increases of 50% and 83% for admissions due to COPD and asthma among individuals aged 40 to 64, and an increase of 42% for COPD among individuals aged 65 and older. In a time-series analysis in which the baseline period was used to generate predicted numbers of hospitalizations by age group for the fire period, the observed admissions were significantly elevated for several respiratory categories (asthma and COPD), principally among the 40–64 yr age stratum. There was no significant elevation of admissions for total circulatory diseases, though observed ischemic heart disease (IHD) admissions ($n = 6$) for the 18–39 yr age stratum were slightly above the 95% upper limit predicted ($n = 5.7$). However, the small numbers involved, coupled with the absence of a significant elevation of IHD admissions in older age groups, suggest caution in interpreting this relationship. Finally, Mott and colleagues examined repeat hospitalizations and survival during the fire period compared with the corresponding periods in 1995 and 1996. Individuals over age 65 with prior hospitalizations for any cardiorespiratory disease, any respiratory disease, or COPD in particular were more likely to be re-hospitalized during the fire period, especially for respiratory causes, compared with the corresponding periods in 1995 and 1996. In particular, individuals with a prior history of hospitalization for COPD were more likely to be rehospitalized for COPD or die from any cause during the fire period (an approximately 44% increase for both outcomes combined); this phenomenon was only manifest when smoke levels exceeded approximately 150 $\mu g/m^3$.

Only one other study has evaluated the impacts of air pollution from vegetation fires on mortality. Sastry (2002) evaluated the population health effects in Malaysia of air pollution generated by a widespread series of fires that occurred mainly in Indonesia between April and November 1997. The results showed that the haze from these fires was associated with deleterious effects on population health in Malaysia and were in general agreement with the mortality impacts associated with particles in urban air (Sastry, 2002). A 10-$\mu g/m^3$ increase in PM_{10} measured in Kuala Lumpur was associated with 0.7 % (all ages) and 1.8% (ages 65–74) increases in adjusted relative risks of nontraumatic mortality. Visibility-based estimates of PM concentrations in Kuching, a city closer to the fire sources, were also associated with increased mortality.

*In a subsequent toxicological examination involving rabbits, these same investigators found that repeated PM_{10} instillations into the respiratory tract resulted in increased production of PMNs in the bone marrow and an acceleration of their release into the blood, both of which were associated with the numbers of particles ingested by the animals' alveolar macrophages (Mukae et al., 2001).

With the exception of three of the Australian bushfire investigations, all of which have significant structural limitations, the epidemiologic studies of indoor and community exposure to biomass smoke indicate a generally consistent relationship between exposure and increased respiratory symptoms, increased risk of respiratory illness, including hospital admissions and emergency room visits, and decreased lung function. Several studies suggest that asthmatics are a particularly susceptible subpopulation with respect to smoke exposure, which is consistent with the results of many studies of the impacts of ambient air pollution. The effects of community exposure to biomass air pollution from wildfires on mortality have not been sufficiently studied to support general conclusions.

Agricultural Burning

There have been few studies of the impacts of agricultural burning, despite growing concern about its potential impact on human health (Tenenbaum, 2000). In one Canadian study, 428 middle-aged subjects with slight-to-moderate airway obstruction were surveyed about respiratory symptoms during a 2-wk period of exposure to straw and stubble combustion products. During the exposure period, 24-h average PM_{10} levels increased from 15–40 $\mu g/m^3$ to 80–200 $\mu g/m^3$. One-hour levels of CO and nitrogen dioxide reached 11 ppm and 110 ppb, respectively. Total volatile organic compound levels increased from preepisode levels of 30–100 $\mu g/m^3$ to 100–460 $\mu g/m^3$ during the episode. Although 37% of subjects were not bothered by smoke at all, 42% reported that several respiratory symptoms (cough, wheezing, chest tightness, shortness of breath) developed or became worse due to the air pollution episode and 20% reported that they had breathing trouble. Subjects with asthma and chronic bronchitis were more likely to be affected, and women appeared to be more susceptible than men for several symptoms (cough, shortness of breath, nocturnal awakening) (Long et al., 1998). In contrast, current cigarette smokers reported significantly fewer symptoms than the former smokers constituting the rest of the study population. This study indicates that, besides woodsmoke, biomass air pollution from agricultural burning is associated with increased respiratory symptoms among a susceptible population with preexisting lung disease.

A time-series study in California suggested that agricultural burn smoke was associated with serious exacerbations of asthma. The association between asthma hospital admissions and the burning of rice field stubble and waste rice straw was examined in Butte County, California, over a 10-yr period (Jacobs et al., 1997). Although burning was not associated with any measurements of major air pollutants (probably because monitors were not sited to provide optimal measurement of burn smoke), burn acreage was significantly associated with an increased risk of asthma hospitalization and showed an exposure-response trend. The greatest risk of hospitalization was observed on days when 500 or more acres were burned (relative risk [RR] 1.23, CI 1.09, 1.39).

A recent cross-sectional study in three rural villages in Iran also evaluated the relationship between rice stubble burning and respiratory morbidity, especially asthmatic symptoms (Golshan et al., 2002). During a burning period lasting several weeks, PM_{10} concentrations doubled. Based on responses to a physician-administered survey before and after this episode, the investigators reported significant increases in the prevalence of asthma attacks, use of asthma medications, the occurrence of nocturnal sleep disturbances, and other respiratory symptoms among 994 residents of an agricultural region. Several measures of pulmonary function also decreased significantly.

The relationship of rice stubble burning with asthma was also studied in Niigata prefecture, Japan (Torigoe et al., 2000). In this study, measured PM_{10} concentrations were associated with monthly asthma hospital admissions and ER visits in a region where rice straw burning emissions led to high particle concentrations during the September–October burning season. During the period 1994–1998, both asthma ER visits and hospitalizations were significantly higher in September than in almost all other months of the year except October and November (for ER visits; hospitalizations in the month of December were also not significantly different from September). Although PM_{10} levels were not associated with monthly ER visits for asthma, the investigators reported a significantly higher number of asthma ER visits on days when rice straw burning occurred and the following 2 days (7.1 ± 3.9) versus other days (4.5 ± 3.3). The latter comparison would have better time resolution than an analysis of monthly average of asthma exacerbations, and should probably be accorded greater weight. Although this investigation also involved a parental questionnaire suggesting more asthma exacerbations in children during the rice burning season than at other times of the year, an autumn peak in asthma flares is also common in other parts of the world where rice burning does not occur. In general, multiple findings in this investigation are suggestive of a rice smoke effect on asthma, but several limitations of the study design constrain both causal inference and the generalizability of the findings.

A metric commonly used as a surrogate of exposure to biomass smoke is the amount of agricultural land burned, as in the Butte County, California, study mentioned earlier (Jacobs et al., 1997). Norris (1998) evaluated the association between acres of grass seed residues burned around Spokane, WA, and visits to local emergency departments for asthma. (Norris, 1998). During one burning event, peak PM_{10} concentrations in Spokane reached 100 $\mu g/m^3$ (Figure 1). Using a bivariate indicator (20 days with >499 acres burned) for the exposure surrogate, an association with increased emergency department visits for children was observed (RR 1.30, CI 1.08, 1.58).

A few studies have specifically examined air pollution and health effects associated with the burning of sugar cane. In Brazil, daily indirect measurements (sedimentation of particle mass) of air pollution during the sugar cane burning season in 1995 were associated with the number of patients visiting hospitals for inhalation therapy for acute respiratory distress (Arbex

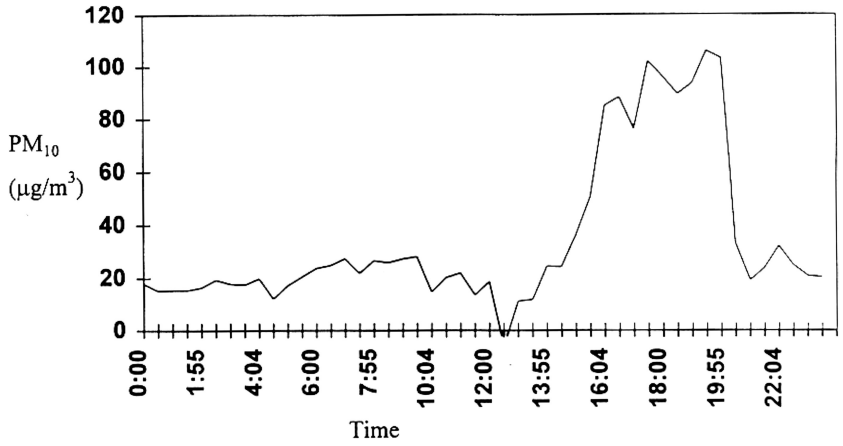

FIG. 1. PM_{10} measured downwind of a grass-burning event in Spokane, WA, at the Rockwood residential monitoring site (September 1994). *Note*: Modified from Norris (1998).

et al., 2000). The relative risk of such a hospital visit associated with an increase of 10 mg in the sediment was 1.09 (1–1.19); this association displayed an exposure-response relationship as well. Boopathy and colleagues presented a descriptive analysis of asthma hospital visits to a medical center in Houma, LA, during 1998–1999 (Boopathy et al., 2002). The area served by this medical center accounted for approximately 27% of Louisiana's sugar-cane cultivation during this period. Although no air pollution measurements were available, asthma hospital visits increased dramatically during the October–December sugar-cane burning season. As noted earlier, however, an autumn peak in asthma exacerbations is common, and respiratory infections (the main precipitating factor for severe asthma attacks) also typically increase in frequency during this time. Therefore, it would be inappropriate to infer a causal relationship between sugar-cane burning and asthma hospital visits based on this descriptive study. Boeniger and coworkers (1991) conducted an exposure assessment of smoke during sugar-cane harvesting in Hawaii in 1987 (Boeniger et al., 1991). They collected both area and personal samples. The concentration of PM increased by at least 20 and up to 70 times the measured background levels at the sampling sites chosen, but were highly variable, making exposure assessment difficult. A subsequent study of Hawaiian sugar-cane workers, however, reported no elevated morbidity or mortality rates or decreased lung function (Miller et al., 1993).

Together, these epidemiologic studies suggest that exposure to products of agricultural burning, specifically the burning of rice stubble/straw, may be associated with exacerbation of asthma. In a chamber study of smoke generated by controlled burning of rice stubble straw, Solomon and colleagues exposed 13 adults with allergic rhinitis (age range 24–55) at rest to filtered air, rice-straw smoke (RSS) at 200 $\mu g/m^3$ or at 600 $\mu g/m^3$ for 30 min, or RSS at 200 $\mu g/m^3$ on 3 consecutive days. Bronchoalveolar lavage (BAL) was conducted at 6 h postexposure. Of a variety of cell types and cytokines measured in BAL fluid, the investigators found a near doubling of epithelial cells only after the 3-day exposure, but no difference from filtered air exposures in total white blood cells, macrophages, PMNs, lymphocytes, eosinophils, or interleukin-8 under any of the RSS exposure conditions. Interestingly, this effect was not observed at a higher concentration (600 $\mu g/m^3$) delivered over a shorter time interval, suggesting that repeated exposures may be necessary, at least among individuals with allergic rhinitis (Solomon, 2003).

Several studies have also reported an increased risk (odds ratios of 1.5–2.5) of lung cancer and mesothelioma among sugar-cane workers, although specific job activities were not evaluated and exposure measurements were not made (Rothschild & Mulvey, 1982; Brooks et al., 1992). A case-control study (118 histologically confirmed lung cancer cases and 128 controls with other cancers matched by age, sex, district of residence, and timing of diagnosis) in India found an increased risk of lung cancer in sugar-cane workers associated with postharvest burning (odds ratio = 1.8, 95% CI 1.0−3.3) (Amre et al., 1999). It has been suggested that this association may be due to the liberation of asbestos-like biogenic silica fibers in sugar cane smoke.

RESIDENTIAL WOODSMOKE IN DEVELOPED COUNTRIES: EXPOSURE AND HEALTH STUDIES

During winter in areas where wood is available, woodburning is common in essentially every part of the developed world for household heating. It is also popular for recreational use in fireplaces. This has implications for area-wide ambient levels and indoor pollution as well as what can be called "neighborhood" pollution, outdoors but sometimes localized in neighborhoods where woodstoves are in use. Here we do not attempt to summarize the evidence on the contribution of woodsmoke to ambient pollution in the developed world, but provide typical examples in different regions.

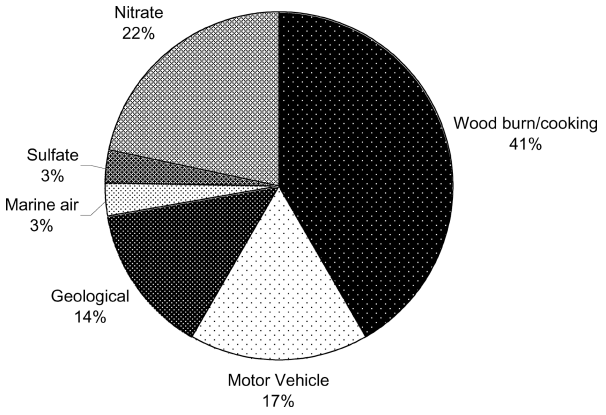

FIG. 2. Source apportionment results for wintertime PM_{10} in San Jose, CA (1993–1994). *Note*: Modified from Fairley (1990).

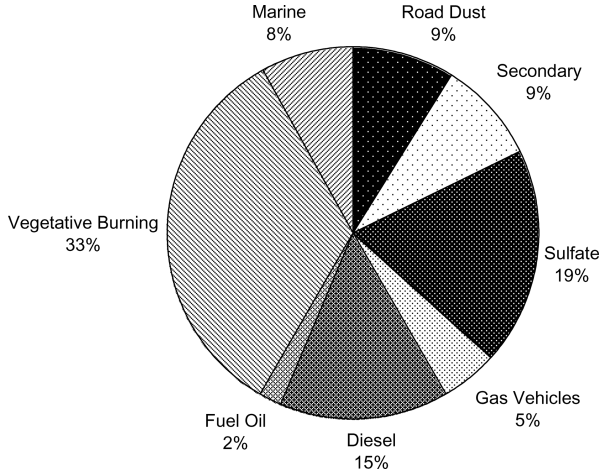

FIG. 3. Source apportionment results for PM_{10} in Seattle, WA (1996–1999). *Note*: Modified from Maykut et al. (2003).

Ambient and Neighborhood Levels

Source apportionment studies indicate that woodsmoke is a major source of ambient PM during winter months in several parts of the United States. Figure 2 shows data from San Jose, CA, that indicate that 42% of the PM_{10} during winter months could be attributed to wood burning (Fairley, 1990). Chemical mass balance receptor modeling of fine particles in Fresno and Bakersfield, CA during wintertime identified both hardwood and softwood as sources of PM and organic compounds (Schauer & Cass, 2000), which were likely to have been due to residential woodburning.

Outdoor PM levels in Seattle, WA; are also heavily influenced by residential woodstoves. Data from 3 years of sampling in Seattle were analyzed for sources using positive matrix factorization (PMF) (Maykut et al., 2003). The PMF analysis found that vegetative burning contributed 34% to the total sources of PM in Seattle over 3 yr (Figure 3).

Another study utilized a large data set from a 2-year exposure assessment and health effects panel study in Seattle during September 2000–May 2001. Indoor, outdoor, personal, and fixed-site PM monitoring data were available. The samples were analyzed for elements using XRF, and positive matrix factorization (PMF) was used to apportion sources (Larson et al., 2004). Five sources contributed to indoor and outdoor samples: vegetative burning, mobile emissions, secondary sulfate, a chlorine source, and a crustal-derived source. Vegetative burning contributed the largest fraction of PM mass in all the samples (35%, 49%, and 62% in indoor, outdoor, and personal mass, respectively).

The distribution of particle-phase organic compounds has been measured in communities with children participating in the Southern California Children's Health Study (CHS) (Manchester-Neesvig et al., 2003). Concentrations of levoglucosan, a good tracer for woodsmoke aerosol, were seen in all 12 CHS communities (Figure 4). The average concentration increased substantially in the winter, as would be expected for woodsmoke emissions. The concentrations of levoglucosan were highest at the Atascadero site, which is about 15 miles inland. Earlier, these investigators identified two additional sugar anhydride tracers of woodsmoke (galactosan and mannosan) in a study of urban sites in the San Joaquin Valley, California (Nolte et al., 2001). These data may allow a separate estimation of the effects of woodsmoke exposure on health outcomes.

In Canada, with cold winters and abundant forests, woodsmoke is a major source of particle emissions. Figure 5 shows that household woodsmoke is responsible for more than 30% of annual PM emissions in 8 provinces and more than 10% in the remaining 4. It is also more responsible for a significant fraction of VOC emissions.*

Christchurch, New Zealand, is another city impacted by woodsmoke. It is estimated that more than 90% of wintertime ambient PM comes from heating stoves and open fires burning wood (McGowan et al., 2002). Frequent periods of air stagnation compound the problem by trapping PM near the ground, and local meteorologists estimate that the relatively even mixing results in fairly homogeneous PM exposure to the population.

Emissions inventories in Launceston, Australia, indicate that household woodburning accounted for 85% of annual PM_{10} emissions in 2000 and that a 50% reduction would be needed in order the city to meet air quality standards.

Source apportionment studies in Denmark show that household woodburning was responsible for 47% of national $PM_{2.5}$ emissions in 2002. In addition, household woodburning grew by about 50% during the 1990s, as compared to only 7% for total energy use.

A recent phenomenon in the United States has been the use of backyard wood-fired boilers for heating homes, which have not

*http://www.ec.gc.ca/science/sandejan99/article1_e.html

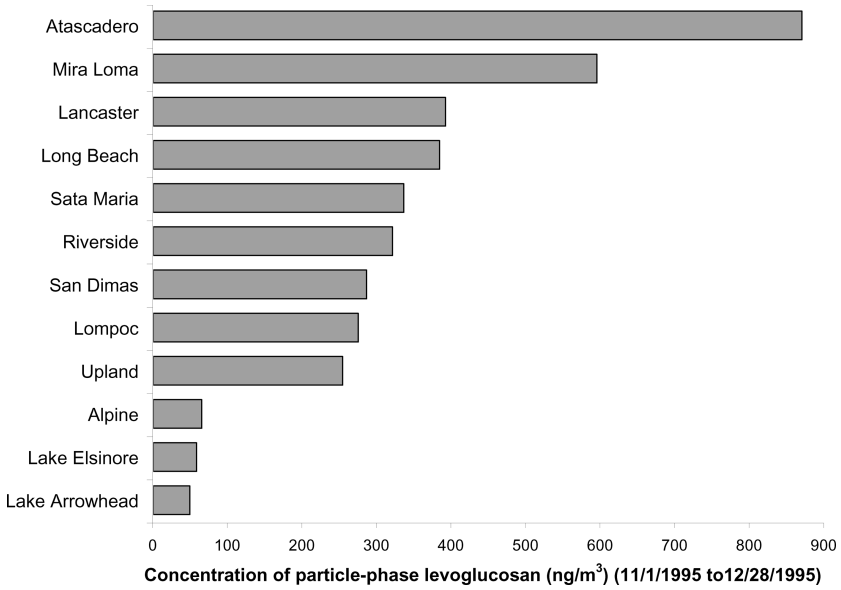

FIG. 4. Spatial distribution of winter time levoglucosan in Southern California (1995–1996). *Note*: Modified from Manchester-Neesvig et al. (2003).

been regulated and often produce substantial pollution locally (Johnson, 2006).

Indoor Levels

Relatively few measurements seem to have been reported of indoor concentrations of woodsmoke in developed-country households. A case-control study of woodstoves and health in Navajo children in Arizona did include measurements of indoor concentrations of respirable particles (PM_{10}) in 90 households (Robin et al., 1996). Cases were children from birth to 24 months of age hospitalized with acute respiratory illnesses and controls that were not hospitalized. Sixty-three percent of the cases had wood stoves in their homes, compared with 51% of the controls. TWA concentrations (15-h) ranged from 22.2 μg/m^3 in houses that used gas or electricity to 100 μg/m^3 in homes that heated with wood alone.

Early studies of woodsmoke health effects often used the presence or absence of a wood stove in the home as the

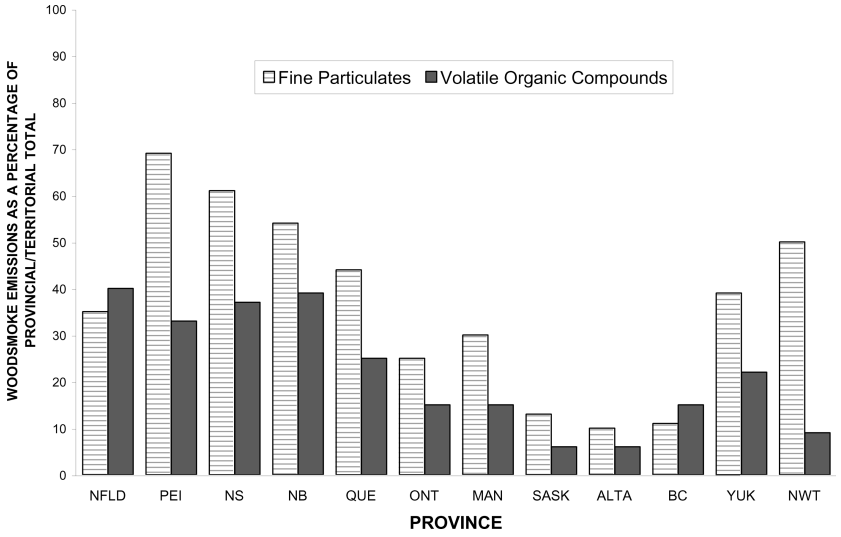

FIG. 5. Importance of woodsmoke emissions in Canada by province. *Note*: Data from Health Canada. See footnote on page 86.

indicator of exposure (see next section). Due to penetration of woodsmoke particles indoors, these exposures may not be due exclusively to indoor sources of woodsmoke. It has been shown in a woodsmoke-impacted community that particles readily penetrate inside residences (Anuszewski et al., 1998). The contribution from outdoor-generated particles to indoor and personal exposure in Seattle, WA, residences has been estimated using a recursive model (Allen et al., 2003, 2004). Nonlinear regression was used to estimate particle penetration, particle decay rate, and particle infiltration. Estimates of particle infiltration agree well with those derived from sulfur-tracer methods ($R^2 = .78$) (Sarnat et al., 2002). In a sample of 44 residences, outdoor-generated particles accounted for an average of $79 \pm 17\%$ of the indoor PM concentration. These data suggest that in epidemiologic studies of associations between health outcomes and outdoor PM, much of the exposure to outdoor particles can occur inside the home. Other factors, such as the age of the house, opening of windows, and air conditioning, can affect penetration. In one study, home air conditioning was associated with lower penetration of outdoor particles; moreover, the associations between PM_{10} and hospital admissions were lower in cities with a higher prevalence of air conditioning (Janssen et al., 2002). These findings imply that even woodstoves and fireplaces operating well that vent most smoke outside may produce substantial exposures through penetration back into the house, a characteristic of "neighborhood pollution."

Health Effects of Residential Woodburning*

To date, only a single controlled exposure study of human exposure to woodsmoke itself seems to have been published (Barregard et al., 2006; Sallsten et al., 2006). Thirteen subjects were exposed to realistic concentrations of woodsmoke (200–300 $\mu g/m^3$ $PM_{2.5}$) generated under controlled conditions for two 4-h sessions, spaced 1 wk apart. In this study, exposure to woodsmoke resulted in small exposure-related changes in levels of inflammatory mediators and coagulation factors. In addition, evidence of increased free radical-mediated lipid peroxidation was observed in 9 of the 13 subjects. Although this is the only controlled study of woodsmoke exposure published to date and it observed a small number of subjects, it is suggestive of woodsmoke-associated systemic inflammatory effects.

The majority of information regarding direct human health effects associated with woodsmoke exposure is derived from a relatively large number of epidemiologic studies have documented respiratory effects of residential woodburning, especially in children. One of the earliest studies was conducted in Michigan by Honicky et al., who compared respiratory symptoms in 31 children who lived in homes with wood stoves with 31 children who lived in homes without wood stoves (Honicky et al., 1985). Symptoms were categorized as mild, moderate, and severe. The two groups did not differ with respect to mild symptoms, but differed significantly for severe symptoms ($p < .001$). A similar study was conducted in Boise, ID, by Butterfield et al., where respiratory symptoms were tracked in 59 children under the age of $5\frac{1}{2}$ years during a winter season (Butterfield et al., 1989). Symptoms such as wheeze, cough, and nocturnal awakening were associated with presence of a woodstove.

Morris et al. (1990) evaluated the impact of indoor woodsmoke child health on a Navajo reservation in Arizona by assessing use of a well-child clinic (Morris et al., 1990). For 58 case-control pairs, the odds ratio (OR) for a serious acute lower respiratory infection (ALRI: bronchiolitis or pneumonia) associated with the presence of a wood stove was 4.2 ($p < .0012$). A more recent case-control study among slightly younger (1–24 mo) Navajo children reached similar, but nonsignificant conclusions (OR 5.0, CI 0.6, 42.8) (Robin et al., 1996). Measured 15-h PM_{10} levels above 65 $\mu g/m^3$ were more frequent in households with wood cookstoves (OR 7.0, CI 0.9 to 56.9). Adjustment for potential confounders (including the number of children living in the house, lack of running water or electricity, difficulty with transportation to the clinic, type of home, and the temperature on the PM_{10} sampling day) had relatively little effect on the magnitude of the associations. The low number of cases (45) likely affected the precision of the estimates, reducing the investigators' ability to detect significant associations between use of wood-burning devices and respiratory infections. It is noteworthy, however, that the magnitude of effect exceeds those generally found in developing-country studies of ALRI in children (discussed later).

A questionnaire study of respiratory symptoms compared residents of 600 homes in a high woodsmoke area of Seattle, WA, with 600 homes (questionnaires completed for one parent and two children in each residence) of a low woodsmoke area (Browning et al., 1990). PM_{10} concentrations averaged 55 and 33 $\mu g/m^3$, respectively. When all age groups were combined, no significant differences were observed between the high- and low-exposure areas. There were, however, statistically significantly higher levels of congestion and wheezing in 1- to 5-year-olds between the 2 areas for all three questionnaires (1 baseline questionnaire and 2 follow-up questionnaires which asked about acute symptoms). This study supports findings from the other investigations suggesting that young children are particularly susceptible to adverse effects of woodsmoke.

In Seattle, WA, 326 elementary school children were studied during the heating seasons of 1988–1989 and 1989–1990 (Koenig et al., 1993). Monthly or bimonthly spirometry values were collected during the school year. PM exposure was measured by light scattering using nephelometers. The exposure metric used was the 12-h nighttime average (7 p.m. to 7 a.m.) to reflect the hours when woodsmoke is most elevated. A random-effects statistical model compared changes in FEV_1 and FVC with changes in the light-scattering coefficient. The 26 children with asthma showed a significant decrement (18 ml/$\mu g/m^3$

*A thorough summary of emissions from woodsmoke was published several years ago (Larson & Koenig, 1994).

$PM_{2.5}$) for both measures of lung function. Children without asthma showed no significant changes in lung function associated with PM values.

A companion study evaluated the impact of particulate matter on emergency room visits for asthma in Seattle (Schwartz et al., 1993). A significant association was observed between PM_{10} particle levels and emergency room visits for asthma. The mean PM_{10} level during the 1-yr study period was 30 $\mu g/m^3$. At this concentration, PM_{10} appeared to be responsible for 125 of the asthma emergency room visits. An exposure response relationship was also observed down to very low levels of PM_{10}, with no evidence for a threshold at concentrations as low as 15 $\mu g/m^3$. The authors indicate that on an annual basis 60% of the fine particle mass in Seattle residential neighborhoods is from woodburning.

Overall, health effects research in Seattle shows associations between $PM_{2.5}$ and lung function decrements in children (Koenig et al., 1993), visits to emergency departments for asthma (Norris et al., 1999), hospitalizations for asthma (Sheppard et al., 1999), and increases in asthma symptoms in children (Yu et al., 2000), as well as increases in exhaled nitric oxide (Koenig et al., 2003, 2005). Since woodburning is the primary source of fine particles in the Seattle airshed, the health effects studies suggest a causal relationship.

Lung function in 410 schoolchildren in Klamath Falls, OR, was studied during winter in high- and low-exposure areas were studied where it has been estimated that woodsmoke accounts for as much as 80% of winter period PM_{10} (Heumann et al., 1991). Winter PM_{10} levels in the high exposure area ranged from approximately 50 to 250 $\mu g/m^3$, while levels in the low exposure area ranged from 20 to 75 $\mu g/m^3$. Lung function decreased during the wood-burning season for the children in the high-exposure area, but not in the low-exposure area.

Two studies were conducted in Montana to evaluate acute changes in lung function in children within a single community at different levels of air pollution, and also to evaluate cross-sectional differences in lung function between communities with different air quality levels, as an indication of chronic impacts (Johnson et al., 1990). Acute lung function decrements measured in 375 children were associated with increased levels of particulates. The 24-h averages ranged from 43 to 80 $\mu g/m^3$ and from 14 to 38 $\mu g/m^3$ for PM_{10} and $PM_{2.5}$, respectively. The chronic impact study also associated small decrements in lung function with residence in communities with higher levels of air pollution. Although particle composition was not measured directly in this study, measurements conducted in the acute study community during the same period attributed 68% of the $PM_{3.5}$ to woodsmoke (Larson & Koenig, 1994).

Another study examined the relationship of woodstoves to otitis media and asthma in a case-control study of home environmental air pollutants in Springville, NY (Daigler et al., 1991). That study found use of woodstoves was more likely to be present in homes of children with otitis media (OR 1.7, CI = 1.03, 2.89).

In contrast, in a larger, prospective study of 904 infants in Connecticut and Virginia, Pettigrew et al. found no relationship between either woodstove or fireplace use and either single episodes of otitis media or recurrent otitis media, which was defined as 4 or more episodes during 1 yr (Pettigrew et al., 2004). Data on infant respiratory symptoms (in this case, a physician's diagnosis of an ear infection) and hours of use of secondary heating sources were collected in telephone interviews with the mothers every 2 wk for 1 yr. Although both woodstove and fireplace use were significantly associated with the outcomes in bivariate models, these associations were absent in multivariate models that adjusted for gender, race, day care, number of children in the household, duration of breast-feeding, winter heating season, use of gas appliances, season of birth, maternal education, maternal history of asthma and allergy, and pets. On the other hand, in the same study, woodstove but not fireplace use was associated with total days of cough in these infants (RR 1.08, CI 1.00,1.16) (Triche et al., 2002).

In a panel study of adults (ages 18–70) in Denver, CO (Ostro et al., 1991), the use of a fireplace or woodstove was associated with an increase in daily moderate or severe shortness of breath (OR 1.3, CI 1.1, 1.4). Use of woodstoves or fireplaces was second only to the presence of smokers in the home, and more strongly associated with shortness of breath than use of gas stoves or occupational exposures. As this study included only subjects with moderate to severe asthma, however, the findings may not be generalizable across the entire clinical spectrum of asthma.

In a study of 888 women living in nonsmoking households in Connecticut and Virginia, Triche and colleagues analyzed daily respiratory symptom data collected during the winter heating seasons of 1994–1996 (Triche et al., 2005). Using Poisson regression and controlling for age, race, allergic status, number of children, education, type of dwelling (single-family vs. multi-unit), and state of residence, these investigators found that each hour-per-day use of a fireplace was associated with several reported respiratory symptoms, including cough (RR 1.05, CI 1.01, 1.09), sore throat (RR 1.04, CI 1.00, 1.08), chest tightness (RR 1.05, CI 0.99, 1.12), and phlegm (RR 1.04, CI 0.99, 1.09). These results suggest that use of a fireplace for 4 h would increase the risk of such symptoms by about 16–20%. No such associations were found for woodstove use, which the investigators suggested may have been due to greater indoor emissions from fireplaces.

Several large time-series studies have been conducted in communities with known woodsmoke sources. The first was conducted in Seattle over a 1-yr period (September 1989 September 1990) (Schwartz et al., 1993), during which there were 2955 emergency department visits for asthma to 8 hospitals. PM_{10} TWA over 24 h ranged from 6 to 103 $\mu g/m^3$, with a mean of 29.6. In Poisson regressions controlling for weather, season, time trends, age, hospital, and day of the week, the daily counts of emergency room visits for persons under age 65 were significantly associated with PM_{10} exposure on the previous day. The relative risk for a 30 $\mu g/m^3$ increase in PM_{10} was 1.12

TABLE 6
Woodsmoke in developed countries: A sample of studies

Location	Woodsmoke concentration	Source
Outdoors		
Santa Clara County, CA	42% of CMB	(Fairley, 1990)
Seattle, WA	49% of total $PM_{2.5}$ mass	(Larson et al., 2004)
Atascadero, CA	Levoglucosan	(Manchester-Neesvig et al., 2003)
Atlanta, GA	11% of total $PM_{2.5}$ mass	(Polissar et al., 2001)
Vermont	10–18% of $PM_{2.5}$	(Polissar et al., 2001)
Christchurch, New Zealand	90% of $PM_{2.5}$ in winter	(McGowan et al., 2002)
Indoor/personal		
Seattle, WA; personal	62% of total $PM_{2.5}$ mass	(Larson et al., 2004)
Seattle, WA; indoor	35% of total $PM_{2.5}$ mass	(Larson et al., 2004)
Fort Defiance, AZ	Indoor PM_{10} dominated by woodstove smoke	(Robin et al., 1996)

(CI 1.04, 1.2). A significant exposure-response trend was found up to nearly 60 $\mu g/m^3$. Woodsmoke contributed approximately 85% of the wintertime PM in residential areas during the study period.

Two time-series studies have been conducted in Santa Clara County, California, an area in which woodsmoke is the single largest contributor to winter PM_{10} (see Table 6). Particulate levels are highest during the winter in this area. The first study was one of the initial mortality time-series studies which indicated an association between relatively low PM_{10} levels and increased daily mortality (Fairley, 1990). A study of asthma emergency room visits in Santa Clara County and winter PM_{10} found a relative risk for an emergency visit, adjusted to a 60-$\mu g/m^3$ increase in PM_{10}, to be 1.4 (CI 1.2, 1.7) at 20°F (Lipsett et al., 1997).

A study in Christchurch, New Zealand, examined the association between hospital admissions and PM_{10} for the period 1988–1998. Ambient PM_{10} levels during the study period averaged 25 $\mu g/m^3$, with a maximum of 283 $\mu g/m^3$. The results were stratified into total cardiac and total respiratory admissions. The estimated percentage increases per interquartile increase in PM_{10} (approximately 15 $\mu g/m^3$) for all age groups was 3.37 (CI: 2.3–4.4) for respiratory admissions and 1.26 (CI: 0.3–2.2) for cardiac admissions, but with no increase for ischaemic heart disease (McGowan et al., 2002). As noted in Table 6, woodsmoke makes up 90% of wintertime PM_{10}. One interpretation of these data is that fine particles from wood burning are more closely associated with adverse respiratory effects than adverse cardiovascular effects. Data from Seattle, WA; support this interpretation, as studies show $PM_{2.5}$ in Seattle associated with asthma aggravation (Koenig et al., 1993, 2003) but do not find similar associations with cardiac events such as myocardial infarction (Sullivan et al., 2005) or sudden cardiac address (Levy et al., 2001).

On the other hand, several studies have failed to find associations between woodstove use and respiratory health (Tuthill, 1984; Eisner et al., 2002). The Tuthill study evaluated health outcomes associated with woodsmoke or formaldehyde exposures in children. An association was seen between respiratory symptoms and prevalence of respiratory disease and estimated exposure to formaldehyde but not seen between these endpoints and estimated exposure to wood smoke. Eisner et al. (2002) studied asthma outcomes in adult subjects exposed to combustion sources indoors that included woodsmoke and environmental tobacco smoke. Although higher use of woodstoves and fireplaces was associated with more severe asthma at baseline, there was no association between use of wood burning devices and asthma aggravation after the 18-mo follow-up.

Summary of Residential Woodsmoke Epidemiology

The studies discussed in this section do have some limitations. For instance, in common with most nonoccupational air pollution epidemiologic studies, few had personal exposure information. These studies, however, do encompass a gradient of health impacts associated with woodsmoke and PM. The indicators of adverse effects run from increases in respiratory symptoms to lung function decreases to visits to emergency departments and finally hospitalizations. It is highly unlikely that this pyramid of adverse effects could be built if the associations reported were not real.

In assessing the "strength" of air pollution health effects data, Bates (1992) concluded that the question of coherence is crucial. He went on to state that such coherence may exist at different levels: within epidemiologic data, and between epidemiologic and toxicological data, and between epidemiologic data and controlled studies. Woodsmoke exposure of residents inside their homes is supported by the infiltration data discussed earlier in this section. Therefore, it is reasonable to conclude that exposure to the concentrations and durations of woodsmoke associated with residential woodburning is likely to cause a variety of adverse respiratory health effects. The biological plausibility for this conclusion is supported both by the toxicology literature, limited controlled exposure studies, and the wealth of data on

health effects of biomass burning in developing countries reviewed below.

Other reviewers have come to similar conclusions (McGowan et al., 2002). Boman et al reviewed the literature relating to adverse health effects from ambient exposure to woodsmoke and, comparing the results of studies of acute exposure to those done in areas without much woodsmoke, concluded that there was no reason to think that the adverse impacts of acute woodsmoke exposure would be less than those associated with other sources of ambient PM (Boman et al., 2003).

Statisticians are attempting to derive models that will allow source apportionment data to be added to health endpoint analysis without creating undue bias (Lumley & Liu, 2003). Creation of such models will help apportion specific health outcomes to specific sources such as woodburning.

This short summary of published studies shows that significant exposures to ambient woodsmoke do occur in developed countries and that important health effects have been demonstrated to result.

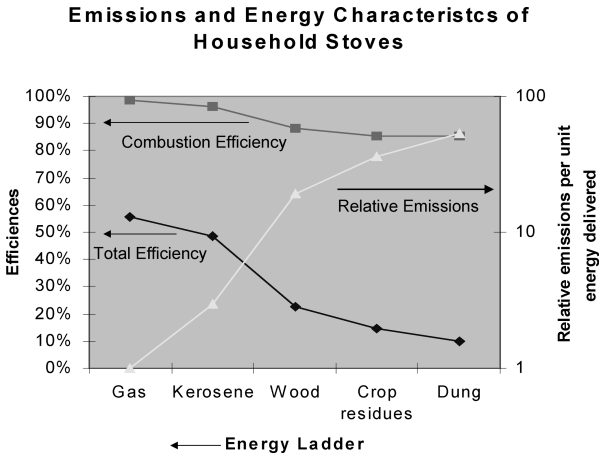

FIG. 7. Emissions and energy characteristics of typical Indian cookstoves. Note improvement in combustion and total efficiency moving from solid to liquid and gaseous fuels and great reduction in emissions per unit energy delivered. Source: Data from Smith et al. (2000).

BIOMASS USE IN DEVELOPING COUNTRIES

Indoor Air Pollution From Household Fuels

Throughout human history, the largest exposures to particle air pollution probably occurred in households through use of wood and other forms of biomass as sources of cooking, drying, and space-heating energy. Even today, such uses probably account for the majority of human exposure to respirable PM worldwide because of the continued high dependence on such household fuels (Smith, 1993). As shown in Figure 6, for example, about half of the world's households are still thought to cook with solid fuels on a daily basis (Smith et al., 2004). Of this, about 95% consists of wood and agricultural residues. Household use of mineral coal for cooking, which makes up the remainder, is mainly confined to China.

In simple devices, like the household stoves commonly used in developing countries, biomass fuel does not combust cleanly. Systematic emissions studies in India and China, for example, have generally validated the so-called "energy ladder" concept with regard to the emissions from combustion of household fuels (Smith et al., 1994). As shown in Figure 7, the energy ladder

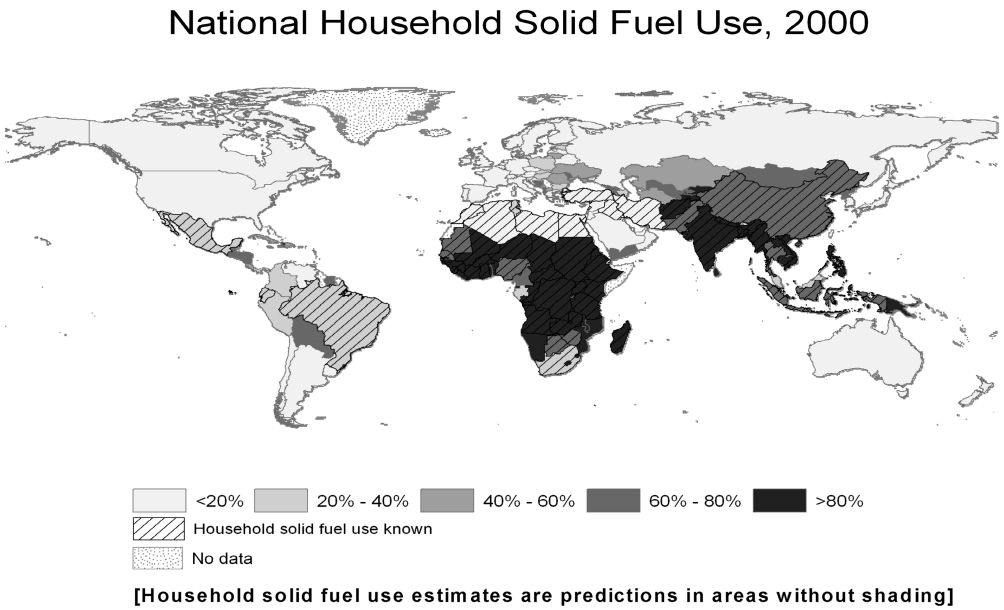

FIG. 6. Map of solid fuel use. Source: Smith et al. (2004).

starts at the bottom with low-quality biomass fuels, such as cow dung, moves up through crop residues, to wood. Further up the ladder lie liquid and then gaseous fuels (kerosene and liquefied propane gas, LPG), with electricity being at the top (i.e., with the lowest emissions). Nominal combustion efficiency (percent of fuel carbon emitted as CO_2) is as low as 80% for the poorer fuels and reaches more than 99% with gaseous fuels (Smith et al., 2000). In combination with the low thermal efficiency of solid fuel stoves, the result is differences in emissions per meal of nearly two orders of magnitude between gaseous and solid fuels. In addition to cleanliness, the cost, complexity, and ease of household use generally increase as one moves up the ladder (Office of Technology Assessment, 1992). Broadly, as average household income increases in societies, usage tends to move up the ladder, although not always to the last rung (electricity). This is shown by econometric studies at the national level (Mehta, 2003). In individual communities, however, the situation is often more complicated, particularly during transition phases, when households may straddle several rungs of the ladder at once by using multiple fuels depending on prices, seasons, availability, and so forth (Sinton et al., 2004).

As noted elsewhere in this report, poor combustion efficiency creates high emission factors for wood and other biomass across a wide range of health-damaging pollutants. High emissions, however, do not necessarily lead to high exposures unless they reach human breathing zones. Unfortunately, however, conditions in hundreds of millions of Third World households are nearly ideal to maximize exposures from emissions. A large, but unknown, fraction of daily cooking is done in unvented stoves, that is, stoves in which the emissions are released directly into the living area and not vented through a chimney or hood. Although there are not systematic surveys in developing-country settings, about 200 studies of indoor air quality (IAQ) measurements in households using solid fuels have been published, more than half from China.* Table 7 shows a summary of these studies for the two most widely measured pollutants, PM and CO. These studies have been published between 1968 and 2003. The studies in South Asia were mainly conducted in Nepal and in India, with only one reported from Bangladesh. The studies in Africa come mainly from Kenya, Gambia, and South Africa. Most of the Latin American studies have been conducted in Guatemala and Mexico.

Most of these studies were conducted in rural settings and attempted to characterize the distribution of concentration levels in the kitchens, with the earlier studies reported from the highlands in different parts of the world. Also, there is little information available on seasonal effects or differences across the various meals cooked in a day. Meal cooking time varied from study to study, generally between 30 min and 3 h, with one study reporting up to 8 h. Although several studies made comparisons between the traditional and the improved stoves, in this summary table that distinction has not been made.

A highly polluting source releasing pollution indoors at times and places when people are always present (household cooking) has a potential to produce high exposure. Put another way, the associated *intake fraction (*fraction of material released that is actually inhaled by someone) is orders of magnitude higher for indoor than for outdoor sources of air pollution (Bennett et al., 2002). Although the uncertainties are large, the available evidence would indicate that the total exposure to combustion-derived fine particles from indoor solid fuel use is larger than that from all outdoor sources of pollution in the world (Smith, 1993).†

Even in communities where most households use chimneys, however, the intake fraction can be substantially higher than for typical outdoor sources since the smoke may sit in the area among the houses in what is called "neighborhood pollution." Such pollution may not be fully reflected by ambient monitoring data, but may nevertheless substantially influence local exposures (Smith et al., 1994). This same phenomenon exists in developed countries as well, for example, from household fireplaces, as discussed earlier. Because of their almost universal role as household cooks, the highest exposures from household use of solid fuels, however, seem usually to occur to women and their youngest children who are with them during cooking, although significant exposures can accrue to other household members as well (Balakrishnan et al., 2004).

Although few studies have linked linked IAQ measurements to ill health, a growing number of epidemiologic studies have found significant risks of various exposure indicators and ill health in developing-country biomass-using households. Such exposure indicators include use of solid or "dirty" fuel versus liquid/gas "clean" fuel; using a stove with a flue or without; years cooking with solid fuel, and, for infants, being carried on their mother's back while cooking or not. Taking advantage of the increasing number of such studies, the recent global Comparative Risk Assessment (CRA) managed by the WHO included indoor as well as outdoor air pollution among the 26 risk factors examined (Ezzati et al., 2002).

The available epidemiologic evidence was divided into three categories, as shown in Table 8. Considered sufficient in quantity and quality to justify inclusion in the global CRA was evidence only in the top category: acute lower respiratory infections (ALRI: pneumonia) in young children, chronic obstructive pulmonary disease (COPD) in adults, and lung cancer in adults (for coal smoke only). The odds ratios shown in the table are the result of meta-analyses of the data in published studies that met the criteria for inclusion (Smith et al., 2004).

A number of epidemiologic studies have also been published for these populations in relation to other important diseases, however. Shown in the second category ("Moderate" evidence) are simple means of odds ratios in available studies

*See the Chinese IAQ database (Sinton et al., 1996) and the non-Chinese IAQ database (Saksena et al., 2003) both available at http://ehs.sph.berkeley.edu/krsmith.

†See also Table 10, which shows the estimate for total global health effects for indoor and outdoor air pollution.

TABLE 7
Summary of indoor air quality studies: Household pollution levels in developing countries outside of China. Values are indicative only as they were determined with various times and measurement methods.

	Number of studies	Total number of samples	TSP ($\mu g/m^3$)		PM_{10} ($\mu g/m^3$)		$PM_{2.5}$ ($\mu g/m^3$)[c]		CO (mg/m^3)	
			Meal	Daily	Meal	Daily	Meal	Daily	Meal	Daily
Bangladesh	1	53							15–26	
Bolivia	1	169			3700					
Brazil	1	23			530					
Burundi	1	2								42
Ethiopia	1	N/A							48	
Gambia	1	12				1600-2200				
Ghana	1	21			590				9	
Guatemala	7	768		280–840		190–1200	450–27,000	97–1900	2–149	1.2–17
India	13	1009	646–16,000		900–1100	506	110–2100	1300–1500	5–216	
Kenya	4	199	3776			1800–3900		630–3500		5–60
Malaysia	1	10		300						3
Mexico	5	191			280–1200	97–290		890	10–22	
Mozambique	1	114			1200				48	
Nepal	5	127	710–8800	4600–8420	4700		1700–5700		14–360	14–52
New Guinea	1	9		360–670						13–24
Nigeria	1	28		108					1076	
South Africa	1	20		1725					79–180	92
Zambia	1	89			890				10	
Zimbabwe	1	34	1357							

Note. From Saksena et al. (2003).
Unspecified averaging time for CO measurements.

TABLE 8
Health effects of use of solid household fuels in developing countries

Disease	Population affected	Relative risk (95% confidence interval)	Strength of evidence
COPD	Females >15 yr	3.2 (2.3, 4.8)[a]	Strong
	Males >15 yr	1.8 (1.0, 3.2)[a]	Intermediate
ALRI	Children <5 yr	2.3 (1.9, 2.7)[a]	Strong
Lung cancer (coal only)	Women >15 yr	1.9 (1.1, 3.5)[a]	Strong
	Men >15 yr	1.5 (1.0, 2.5)[a]	Intermediate
Blindness (cataracts)	Females >15 yr	1.3–1.6[b]	Intermediate
Tuberculosis	Females >15 yr	1.5–3.0[b]	Intermediate

Note: From Smith et al. (2004).
[a]Based on formal meta-analysis.
[b]Range of results in published studies.

showing significant impacts of use of solid fuels on tuberculosis and cataracts. There is also evidence of the impact of biomass smoke exposures on lung cancer (Behera & Balamugesh, 2005; Hernandez-Garduno et al., 2004). Although consistent, the number and character of these studies was not considered sufficiently persuasive to include these diseases in the CRA.* Similarly, although studies of outdoor air pollution, ETS, and other sources of particle exposure indicate impacts on asthma and heart disease, no convincing studies are yet available in the populations of interest for household solid fuel use.†

Using only the "Strong" evidence category in Table 8 and the distribution of solid fuel use shown in Figure 6, the total impact of IAQ from solid fuel use calculated in the CRA is shown and compared to other environmental risk factors in the CRA in Table 9.

At 1.6 (0.8–2.4) million deaths and 2.6% of the global burden of disease (as measured in lost life-years), IAQ ranks second only to poor water/sanitation/hygiene among environmental health risk factors. Among all major policy-relevant risk factors, indoor air pollution from solid fuel is tenth globally, and fourth in least-developed countries.‡ See Figure 8.

Biomass Smoke in LDC Cities

Ambient air pollutants come primarily from combustion of fossil fuel. In many cities and rural areas in developing countries, residential space heating and cooking with solid fuels, mostly biomass and coal, can also contribute significantly to the ambient pollution. Several studies have been conducted in developing countries quantifying the contribution of biomass smoke in cities. Begum et al. (2004) report contribution from biomass combustion to be 12% in Dhaka and 50% in Rajshahi, Bangladesh (Begum et al., 2004). From a study conducted by Zheng et al. (in preparation) in three sites in Hong Kong, they find that 9–10% of the organic carbon in $PM_{2.5}$ comes from biomass smoke. Chowdhury et al. report contributions from biomass in three Indian cities by season as seen in Table 10. (Chowdhury et al., 2005.).

High concentration of biomass smoke in the colder months compared to the warmer months can be explained by the regional meteorology where the monsoon rains are dominant in the summer months followed by the dry winter, when pollutants are trapped inside the inversion layer. Also, during the winter months there is a tendency to use biomass fuel for heating purposes, leading to the higher concentrations in Delhi and Kolkata, 66/51 $\mu g/m^3$ $PM_{2.5}$, representing 29/17% of the total $PM_{2.5}$. Although a bit higher in absolute concentrations, this winter pattern is also found in developed-country urban areas using wood fuels.

TOXICOLOGICAL EFFECTS OF WOODSMOKE EXPOSURE

Although studying the effects of air pollutants directly on humans offers a number of advantages, epidemiologic and controlled clinical studies are often limited by societal concerns, ethical and legal issues, as well as cost. Because of these difficulties, predictive health assessments associated with inhaled woodsmoke need to include information gained from animal exposure studies and, in some cases, *in vitro*/*ex vivo* assay systems. Furthermore, animal studies also have the potential to help uncover information concerning the mechanisms of toxicity and relative toxicity of different mixtures and sources. The discussion that follows summarizes some of the principal published toxicologic studies of woodsmoke.

*A similar conclusion was reached in 2006 in IARC Monograph #95, in which household combustion of biomass was rated as Category 2A, limited human evidence with supporting animal evidence (Straif et al., 2006).

†A recent conference abstract, however, has shown a clear effect of lowered blood pressure from women whose woodsmoke exposures were lowered in a randomized clinical trial of improved stoves with chimneys in Guatemala (McCracken et al., 2005).

‡The summary results of the CRA were released in the World Health Report (WHO, 2002; Ezzati et al., 2002) and were published in detail in Smith et al. (2004).

TABLE 9
Global burden of disease and premature death due to major environmental risk factors in 2000

Parameter	Poor countries	Mid-income countries	Rich countries	World
Population (millions)	2343	2424	1358	6125
DALYs (million disability-adjusted life years)[a]	846	406	214	1,467
Unsafe water, sanitation, and hygiene	5.5%	1.8%	0.4%	3.7%
Indoor smoke from solid fuels	3.6%	1.9%	0.3%	2.6%
Occupational risks	1.1%	2.4%	1.5%	1.5%
Lead exposure	0.7%	1.4%	0.6%	0.9%
Urban air pollution	0.3%	1.0%	0.5%	0.5%
Climate change	0.6%	0.1%	0.0%	0.4%
Total environmental burden of disease, % of total for region	11.8%	8.5%	3.4%	9.7%
Deaths (thousands)	26,700	16,000	13,000	55,700
Unsafe water, sanitation, and hygiene	1538	172	20	1730
Indoor smoke from solid fuels	1039	558	22	1619
Occupational risks	2393	640	176	3209
Lead exposure	93	69	72	234
Urban air pollution	220	426	154	800
Climate change	148	5	0	153
Total environmental mortality in region (thousands)	5431	1870	444	7745

Note: From WHO (2002).

[a]DALYs are calculated as the sum of lost years from premature mortality and lost years of illness and injury weighted by a severity factor.

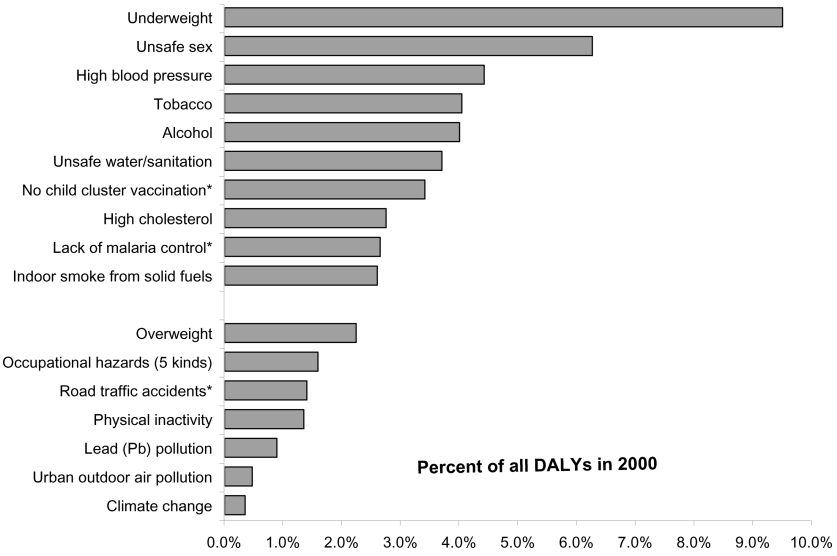

FIG. 8. Estimated burden of disease in 2000 measured as lost healthy life years (DALYs) from major preventable risk factors. Note importance of indoor smoke from solid fuels, which are mostly biomass. Those marked with asterisk are based on outcomes in the WHO Global Burden of Disease database. *Note*: Created from WHO data by Smith and Ezzati, 2005.

TABLE 10
Contribution of biomass smoke into urban ambient $PM_{2.5}$ in India

	Spring		Summer		Autumn		Winter	
Location	$\mu g/m^3$	% of $PM_{2.5}$	$\mu g/m^3$	% of $PM_{2.5}$	$\mu g/m^3$	% of $PM_{2.5}$	$\mu g/m^3$	% of $PM_{2.5}$
Delhi	25	22%	5	10%	33	21%	66	29%
Mumbai	5	13%	N/A	N/A	14	21%	11	13%
Kolkata	10	19%	6	24%	14	32%	51	17%

Note: From Chowdhury et al. (2005). Based on five to seven 24-h samples per season.

In Vivo Inhalation Studies

Although woodsmoke can be delivered to animals by a variety of methods, including open-chest ventilation, only those studies that employed exposure routes most relevant to the human woodsmoke experience (i.e., nose-only/whole body inhalation in conscious animals) are reviewed herein.

Single Woodsmoke Exposures

Acute lung injury. Because of increasing interest in the mechanisms of damage in firefighting and other high-exposure situations, the majority of toxicologic studies reviewed for this document fell within this category. As the exposure levels used for these studies are usually much greater than those to which the general public in developed countries is exposed, these studies will serve primarily to demonstrate the effects that could, potentially, occur as a result of lower level, repeated exposures. In 1982, an inhalation study by Thorning et al. (1982) described the effects of inhaled woodsmoke on pulmonary lung cell injury (as determined by changes in lung morphology). Two combustion products (i.e., CO and organic aldehydes) were monitored as a measure of woodsmoke exposure. Total aldehyde concentrations in the chamber ranged from 285 to 1707 ppm for the 25- to 45-min exposures. Smoke-exposed rabbits exhibited necrotizing tracheobronchial epithelial cell injury that peaked by 24 h post-exposure. The authors concluded that the acute lung injury and early reactions to smoke damage observed in rabbits closely resembled those pulmonary lesions seen in smoke-injured victims, and that these injuries could affect pulmonary host resistance. Particle-adsorbed aldehydes were suggested to play the major role in such injuries.

In another study using rabbits, Loke et al. (1984) investigated the effects of a 60-min inhalation of Douglas fir-generated woodsmoke (mean carboxyhemoglobin [COHb] level = 16.4%) on alveolar macrophage (Mø) response and tracheobronchial morphology. Smoke injury to the proximal tracheal lining cells was severe, with major epithelial cell loss observed in exposed animals. In addition, mucociliary escalator dysfunction was also observed. Similar to the cellular alterations observed in long-term cigarette smokers (Rasp et al., 1978), alveolar Mø from woodsmoke-exposed rabbits were flatter and contained less surface ruffling (a marker of cell activation) than their unexposed counterparts. In addition, Mø numbers were increased within the alveoli, suggesting an inflammatory response in the smoke-exposed rabbits.

In a study by Fick et al. (1984), rabbits were exposed acutely (i.e., 30–120 min) to smoke from the pyrolysis of Douglas fir wood and effects on Mø functional activity were examined immediately following exposure. At a smoke concentration yielding a COHb level of 7% and no evidence of thermal injury, pulmonary Mø-mediated bacterial phagocytosis and intracellular killing of the gram-negative bacterial pathogen *Pseudomonas aeruginosa* was dramatically reduced. Although an inflammatory response was not observed, smoke-exposed animals demonstrated a significantly greater lavageable cell yield than the unexposed controls. This investigation provided the first evidence that lower levels of woodsmoke could produce subclinical effects and alter lung properties in the absence of any acute lung injury. This well-executed toxicologic study employed the most current inhalation procedures of their time and evaluated effects in a dose-response manner based on increasing COHb levels. The authors concluded that inhalation of woodsmoke, at a relatively low level, had the potential to alter host pulmonary immune defense mechanisms in such a way as to lead to an increased susceptibility to infectious lung disease.

Woodsmoke-induced alterations in phagocyte-mediated oxidative stress response and antioxidant status were studied in a rat model designed to simulate an inhalation injury as might be encountered by firefighters and/or burn victims (Dubick et al., 2002). In this study, rats, either previously scalded or sham-burned, were exposed for approximately 16 min to clean air or smoke generated from the burning of Western bark wood (i.e., fir and pine); smoke exposure was assessed by measuring COHb levels in exposed hosts (19 $\pm$ 2%). At different time points post-exposure, animals were sacrificed and either their lungs were lavaged for evaluation of total protein and airway cellular/lung water content, or their tissues were recovered for measurements of antioxidant enzyme activities and lipid peroxidation (measured by thiobarbituric acid-reactive substances [TBARS]) (Dubick et al., 2002). Similar to that seen in other smoke injury studies, Dubick et al. (2002) observed that acute inhalation of woodsmoke produced areas of tracheal erosion resulting in the loss of epithelium. At 24 h postexposure, lipid peroxidation was increased two- to three-fold above control values in the smoke-only and burn/smoke-exposure groups; TBARS

declined after 48 h in the smoke-only group. Other investigators have also reported oxidative stress as a result of woodsmoke exposure (Demling & LaLonde, 1990; Demling et al., 1994; Lalonde et al., 1994). Minor changes in lung antioxidant enzyme activities were also observed in this study. However, in contrast to the dramatic inflammatory response observed in previous studies examining acute smoke-induced lung injury (Traber & Herndon, 1986; Hubbard et al., 1991), pulmonary immune cell infiltration was not observed. Given that leukocyte infiltration appears to "track" with woodsmoke-induced pulmonary injury, a lack of neutrophil influx was not surprising, given the modest level of lung injury produced in the aforementioned study.

In addition to tracheobronchial alterations and changes in immune cell morphology, acute inhalation (30 min) of Douglas fir-generated woodsmoke has been shown to diminish the ventilatory response of exposed guinea pigs. In a study by Wong et al. (1984), animals were exposed for 30 min to increasing woodsmoke concentrations generated by burning different amounts of wood chips. Just prior to exposure and at various time points post-exposure, guinea pigs challenged with CO_2 were placed in a whole-body plethysmograph and lung compliance (elastic properties of the lungs) was measured. Exposure to moderate levels of smoke increased baseline ΔP (change in lung relaxation pressure), but only 0.5 h after exposure. None of the exposure groups varied significantly from controls with respect to ΔP after this early time point. Reduction in pulmonary compliance has also been shown in woodsmoke-exposed dogs (Stephenson et al., 1975) and in human victims of smoke inhalation (Garzon et al., 1970). Wong et al. (1984) concluded that acute inhalation of woodsmoke can alter lung function, but that recovery occurs within several days after exposure.

Woodsmoke-induced alterations in airway responsiveness to bronchoconstrictor challenge have also been observed more recently in other studies using guinea pigs. Exposure to a PM concentration of 25 mg/m^3 produced airway hyperreactivity in response to challenge with the bronchoconstrictors substance P, capsaicin, and prostaglandins (Hsu et al., 1998a, 1998b; Hsu & Kou, 2001; Lin & Kou, 2000; Lin et al., 2001). Despite the "artificial" exposure route used to deliver woodsmoke in these studies, the results provide compelling evidence regarding the adverse effects of woodsmoke on airway responsiveness. Support for these findings in guinea pigs comes from repeated exposure studies by Tesfaigzi et al. (2002), who demonstrated a significant increase in total pulmonary resistance and dynamic lung compliance in brown Norway rats exposed by inhalation (whole body) to lower concentrations (1 or 10 mg/m^3) of woodsmoke generated from the burning of *Pinus edulis* wood for either 4 or 12 wk (3 h/day, 5 days/wk).

Some of the same investigators who examined bronchoconstriction in guinea pigs also performed studies in rats to evaluate the role of sensory receptors and nerve fibers in pulmonary ventilation following woodsmoke exposure (Kou & Lai, 1994; Kou et al., 1995, 1997, 1999; Wang et al., 1996; Lin & Kou, 1997; Lai & Kou, 1998a, 1998b, 1998c; Lin et al., 2000; Ho & Kou, 2000). For these studies, rats were exposed to either particle-free (i.e., gas only) or whole woodsmoke effluents. Results demonstrated that (among other things) woodsmoke-induced slowing of respiration is a reflex resulting from stimulation of bronchopulmonary C-fiber nerve endings (unmeylinated sensory neurons that conduct nerve impulses slowly) induced by the woodsmoke gas phase. These studies further revealed that increased hydroxyl radical (·OH) burdens following smoke exposure were actively involved in evoking the acute irritant effects of woodsmoke. Investigations by Ho and Kou (2002) also demonstrated that woodsmoke exposure increased nasal airway resistance and airway reactivity in rats exposed to woodsmoke via the nose.

In a thorough, well-executed inhalation study by Matthew et al. (2001), exposure of mice to high doses of woodsmoke (COHb level of 50% immediately after exposure): altered pulmonary histology; induced an inflammatory response; increased static lung compliance; and increased lavageable cytokine levels and cell counts (Matthew et al., 2001). Given that smoke inhalation damage is mediated in part via an upregulated immune response, increased numbers of lavageable immune cells are consistent with the observed lung pathology. The authors speculated that smoke-associated PM, with or without adhered noxious gases, were likely responsible for the majority of observed lung pathologies. This notion was supported by the studies of Zelikoff (2002), who demonstrated diminished immunotoxicity of inhaled woodsmoke effluents in rats following removal of the particulate smoke phase (Thomas & Zelikoff, 1999).

Repeated and Subchronic Woodsmoke Exposures

Pulmonary and systemic effects . Inhalation studies utilizing scenarios other than acute single-exposure regimes are extremely limited. Long-term investigations that more closely reflect smoke exposures associated with domestic pollution (i.e., home heating and cooking) are needed to assess potential long-term risks. Lai et al. (1993) examined the hematological and histopathologic responses of rats exposed repeatedly to smoke generated from the combustion of wood dust. Despite the primitive nature of the exposure system, the lack of information concerning smoke concentration and wood type, and the lack of data regarding thermal burns, many of the same smoke-induced pathologies observed in this study (i.e., epithelial lining cell desquamation, pulmonary edema, and peribronchiolar and perivascular infiltration of polymorphonuclear neutrophils [PMN]) have also been reported in the studies of acute smoke exposures (Lal et al., 1993). Bronchiolitis, parenchymatous blood vessel congestion, hyperplasia of lymphoid follicles, and mild emphysema were also observed after 15 days of smoke exposure. Although the emphysematic changes remained constant over time, other pulmonary lesions worsened dramatically with increasing exposure duration. In addition, marginal alterations were observed in hemoglobin levels, sedimentation rate, packed cell volume, and total and differential leukocyte counts from animals exposed to smoke for 15 days. Eosinophilia was also observed, but only in rats exposed for 30 and 45 days. Results

of this study demonstrated that woodsmoke-induced pulmonary lesions are progressive with repeated smoke exposures. Moreover, given that domestic woodsmoke pollution has been associated with chronic obstructive pulmonary disease (e.g., chronic bronchitis and emphysema) in developing countries and that emphysematic changes were observed in this toxicologic study, the authors concluded that the rat model of acute lung injury may prove useful for assessing the toxicologic impact and human health outcomes of inhaled woodsmoke.

Studies from this group (Zelikoff, 2000) have demonstrated that repeated short-term (1 h/day for 4 days), nose-only inhalation exposure of rats to woodsmoke generated from the burning of red oak wood (i.e., 750 μg PM/m^3, <2 ppm CO, 3 ppb NO_x, and 1.5 ng total PAH/m^3) inhibited pulmonary clearance of intratracheally instilled *Staphylococcus aureus*; the smoke concentration used in this study is reflective of that found indoors during operation of a poorly vented fireplace, a non-U.S. Environmental Protection Agency (EPA)-certified wood-burning device, or under extreme residential conditions where open fires are used for heating and cooking. The effect on bacterial clearance was observed in the absence of any lung pathology, lung cell damage (as measured by total protein and lactate dehydrogenase release) or inflammation. The lack of pulmonary injury and/or inflammation was similar to that observed in a more recent study by Reed et al. (2006) in which rats and mice were exposed (by whole-body inhalation) for longer time durations (1 wk or 6 mo) to hardwood smoke (HWS) generated from an uncertified wood stove.

In the aforementioned studies, suppressed bacterial clearance began as early as 3 h postexposure and persisted for almost 2 wk (Thomas & Zelikoff, 1999). Interestingly, similar dramatic effects on pulmonary bacterial clearance were not observed for rats exposed to particle-free woodsmoke effluents. This response demonstrates the importance of the woodsmoke-associated PM in bringing about the observed time-related effects on pulmonary host resistance. In the same study (Thomas & Zelikoff, 1999), woodsmoke exposure also suppressed production of pulmonary Mø-mediated superoxide anion ($O_2^{\cdot-}$), a reactive oxygen species critical for the intracellular killing of *S. aureus*. The authors suggested that reduced production of $O_2^{\cdot-}$ might (in part) be responsible for the observed woodsmoke-induced decrease in pulmonary host resistance against this particular pathogen. Taken together with results from earlier studies, the authors concluded that short-term, repeated inhalation of woodsmoke can compromise pulmonary immune mechanisms that are critical for host protection against infectious lung pathogens. Moreover, they concluded that the pulmonary Mø represents a sensitive target for the toxic effects of inhaled woodsmoke.

A more recent study has examined the effects of inhaled woodsmoke at 1 or 10 mg PM/m^3 on rats exposed to smoke generated from the burning of *Pinus edulis* for 4 or 12 wk (Tesfaigzi et al., 2002). In the absence of any effects on lung-associated T-lymphocyte proliferation or lavageable cytokine levels, repeated smoke inhalation produced a modest but significant reduction of CO-diffusing capacity (as demonstrated by an impairment of gas exchange) in the high-dose exposure group; increased dynamic lung compliance also in the 10-mg PM/m^3 group; and mild chronic inflammation and squamous-cell metaplasia in the larynx of all groups of exposed rats. The mucous-cell metaplasia observed after 30 days of woodsmoke exposure was transient and resolved after 90 days. The severity of alveolar Mø hyperplasia and pigmentation increased with smoke concentration and length of exposure. However, some dose-response inconsistencies and the absence of a significant change in quasistatic compliance, a more specific measure of lung elastic recoil, led the authors to conclude that the impact of woodsmoke in this study was small and, except for the observed reduction in gas exchange, of little clinical importance. Though one could dispute the investigators' conclusions regarding the nominal importance of these findings and the insensitivity of the immune system for assessing the health impacts of inhaled woodsmoke, the study was well executed, demonstrated a dose-response relationship for some endpoints, employed a well-described generation/exposure system, and incorporated extensive chemical characterization of the woodsmoke effluents.

Two rodent studies, recently published by investigators at the Lovelace Respiratory Research Institute (LRRI), examined the health effects of repeated hardwood smoke (HWS) exposure using a range of exposure concentrations at or just above those commonly experienced in the indoor and/or outdoor U.S. environment (30–1000 μg PM/m^3) (Reed et al., 2006; Barrett et al., 2006). Specifically, studies by Barrett et al. (2006) investigated the ability of short-term, repeated exposure to HWS to exacerbate allergic airway responses in already sensitized mice; two different sensitization paradigms were examined. Findings from this study demonstrated that in the absence of tissue inflammation or altered Th1/Th2 cytokine levels, a 3-day exposure to HWS following the final allergen challenge could exacerbate some indices of allergic airway inflammation, such as lavageable numbers of eosinophils and serum OVA-specific immunoglobulin E (IgE). The authors concluded that the effects of HWS on allergic airway parameters were relatively mild, but were comparable to those responses observed with other pollutant mixtures such as diesel exhaust.

A large companion study published by some of the same LRRI investigators examined the effects of longer exposure durations (1 wk or 6 mo) to HWS on general indicators of toxicity (i.e., body and lymphoid organ weights, clinical chemistry and hematology), bacterial clearance, cardiac function, and carcinogenic potential using both mice and rats (Reed et al., 2006). A range of woodsmoke levels (30–1000 μg PM/m^3) and both genders of two rodent species were examined. Exposure-related effects included increases in blood platelets; decreases in blood urea nitrogen and serum alanine aminotransferase; changes in liver, spleen and thymus weight; and increased circulating white blood cell (WBC) counts. No effects were observed upon micronuclei formation, tumorigenesis, cardiac parameters, or pulmonary clearance of the bacteria *Pseudomonas aeruginosa*. The

lack of any effects on bacterial clearance is in contrast to those effects reported by Zelikoff (2000), who demonstrated that inhalation of woodsmoke at 750 $\mu g/m^3$ for 4 days significantly reduced pulmonary clearance of *S. aureus* in exposed rats. Differences between the studies may have been due to any one of a number of factors, including disparity between rodent models, wood type, burning conditions, and/or possible adaptation of the mice to the long-term exposure scenario (6 mo). Another important difference between the two studies is the bacterial species used for challenge. *Pseudomonas* is removed mainly in the infected host by the bactericidal mechanisms of neutrophils, while those mechanisms used to remove the gram-positive cocci *S. aureus* are primarily mediated by Mø, a sensitive target for the toxic effects of woodsmoke, Thus, differences in bacterial clearance mechanisms could have played a role in the disparity observed between the studies. Reed et al. (2006) concluded, based upon observed sex and exposure duration inconsistencies, that at the woodsmoke concentrations utilized in these studies, the observed effects "posed little to small hazard with respect to clinical signs, lung inflammation and cytotoxicity, blood chemistry, hematology, cardiac effects, bacterial clearance and carcinogenic potential." While this study represents an eloquently executed investigation, the authors seem to have minimized the observed smoke-induced effects which could result in an underestimation of the actual risks associated with such exposures. The potential short- and long-term health risks associated with some of the observed effects (i.e., increased platelet number, reduced liver weight and increased spleen weight) appear worthy of further consideration, particularly in light of the recent experimental study which demonstrates that healthy humans exposed to wood smoke at 250–280 micrograms/m3 during two 4h sessions increases the levels of serum amyloid A (a cardiovascular risk factor) and plasma factors important for maintaining the balance of coagulation factors (Barregard et al., 2006).

Lung cancer. A field study was carried out in which mice and rats were placed for 15 or 19 mo, respectively, in an indoor environment to inhale either air (without combustion products) or smoke generated from burning wood or coal (Liang et al., 1988). Burning was carried out in round shallow pits of individual rooms meant to simulate those of villagers in Xuan County, China, as were the patterns and intensity of burning (241 kg/mo; 12 h smoke/day). Although smoke generated from both coal and wood contained similar total suspended particulate (TSP) levels (i.e., 14.4 vs. 14.9 mg/m^3, respectively), the BaP concentration in the wood exposure room was approximately 47 times higher than that measured in the air control environment, although substantially less than that measured in the coal-using rooms. In the woodsmoke room, measured levels of CO, SO_2 and H_2SO_4 were 80 mg/m^3, 0.05 mg/m^3, and 0.27 mg/m^3, respectively. Following exposure, animals were immediately sacrificed and the incidence of nonmalignant and malignant lung tumors was evaluated. Tumors were histologically differentiated into several groups including adenomas, adenocarcinomas (AC), adenosquamous carcinomas (ASC), and squamous-cell carcinomas (SCC). Although control mice demonstrated a lung cancer incidence of 17%, mice exposed long-term to either wood or coal smoke had incidences of 45.8% and 89.5%, respectively. Although all lung cancer types were observed in coal smoke-exposed mice, those exposed to woodsmoke demonstrated far fewer SCC and ASCs. Lung cancer incidence for rats exposed to ambient air or smoke from burning wood or coal was 0, 0, and 67.2%, respectively.

The authors of the aforementioned study concluded that woodsmoke proved to be only a weak carcinogen compared to coal smoke. Differential effects between wood and coal have also been observed in a mouse skin tumorigenicity study that compared the effects of organic extracts from smoky coal and wood combustion (Mumford et al., 1990). In this case, particle extracts from smoky coal combustion proved to be a potent complete carcinogen, whereas that from woodsmoke proved relatively inactive. The relatively modest effects observed in this early study by Liang et al. (1988) are in line with those reported by Reed et al. (2006), who demonstrated that inhalation of lower concentrations of woodsmoke (30–1000 μg PM/m^3 vs. 14.9 mg/m^3) for only 6 mo failed to significantly increase lung tumors in exposed mice (compared to control).

Ex Vivo/In Vitro Woodsmoke Exposure Studies

Although toxicologic studies using routes of exposure other than inhalation were not the primary focus of this review, several *ex vivo*/*in vitro* studies are briefly discussed inasmuch as they might contribute to a better understanding of the potential health impacts of woodsmoke. Bhattacharyya et al. (1998) examined the effects of pine woodsmoke exposure for 5–20 min on rabbit tracheal explants. Exposure of explants for 20 min led to degeneration of the mucociliary epithelial sheath; shorter smoke exposures (i.e., 10 min) resulted in retained tissue integrity, but altered epithelial morphology. Similar woodsmoke-associated pathologies have been observed *in vivo* following acute inhalation exposure.

Exposure of cultured eye lenses to woodsmoke condensates for 10 min resulted in woodsmoke metabolite-induced opacification (Rao et al., 1995). Histological analyses of smoke-exposed lenses revealed distinct morphological changes including hyperplasia, hypertrophy, and multilayering of epithelial cells. The authors concluded that exposure to woodsmoke could contribute to progressive eye lens opacification.

Leonard et al. (2000) examined the effects of pine- and Douglas fir-generated liquefied woodsmoke on cultured mouse Mø free radical generation, DNA damage, nuclear factor (NF) κB activation and tumor necrosis factor-α (TNFα) release. These studies demonstrated that exposure to liquefied woodsmoke in combination with hydrogen peroxide (H_2O_2) resulted in hydroxyl radical ($\cdot$OH)-induced DNA damage, and that co-exposure to on an $\cdot$OH radical scavenger or a metal chelator inhibited the observed genotoxicity (Leonard et al., 2000). The authors concluded that free radicals generated by woodsmoke

through the reaction of iron with H_2O_2 could produce genetic and cellular damage. Moreover, such free radicals could also play a role in the development of woodsmoke-induced pulmonary fibrosis.

A number of genetic toxicology studies have evaluated the mutagenicity of woodsmoke condensates (Hytonen et al., 1983; Alfheim & Ramdahl, 1984; Alfheim et al., 1984; Asita et al., 1991). In all cases, woodsmoke extracts were mutagenic in bacterial systems. A number of factors, including heating conditions, type of wood-burning device, wood origin, and PAH concentration, seemed to play important roles in overall mutagenic activity.

Toxicology Summary

The majority of the toxicology studies presented in this review provide biological plausibility for the epidemiologic evidence suggesting that exposure to woodsmoke emissions adversely affects human health. These animal studies also contribute to a better understanding of the possible mechanisms(s) by which woodsmoke, and its associated PM, may act to bring about increased pulmonary morbidity in exposed individuals. It appears clear from the toxicologic studies that short-term inhalation of woodsmoke can compromise pulmonary immune defense mechanisms important for maintaining host resistance against pulmonary infections. Moreover, a likely target for woodsmoke-induced immunotoxicity seems to be the lung Mø. These immune cells, which serve as the primary defense of the deep lung, provide a link between the nonspecific and specific defense systems of the respiratory tract. These studies lend support to the notion that inhaled woodsmoke contributes to the increased incidence of infectious respiratory disease reported in children living in developing nations and/or near homes heated by woodburning devices.

Effects of inhaled woodsmoke were most dramatic after acute, high-dose exposure. While effects observed at woodsmoke concentrations of 750 μg PM/m^3 may not be especially relevant for developed nations, levels much higher than 1 mg/m^3 are commonly encountered in developing countries where about 15% of the total energy supply comes from wood. Thus, high-dose studies that examine effects related to the majority of the world's population are critical and should continue to be carried out. In addition, while some evidence is also provided that long-term exposure to lower concentrations of woodsmoke, more reflective of those encountered in North America, may also pose some health risks, more studies are needed before any definitive conclusions can be reached regarding the health risks (if any) associated with such exposures.

Making interspecies comparisons between humans and other mammalian species is complicated and needs to be approached with caution due to differences in anatomy, breathing rates, metabolism, and particle deposition. However, results of these studies lend support to the applicability of laboratory animals as a model to predict woodsmoke-induced alterations of human pulmonary health. Both the similarities and differences in woodsmoke-induced effects seen between humans and laboratory models underscore the importance of comparative studies as a basis for extrapolation modeling. Although more toxicologic studies are needed to determine the effects of long-term exposure, and to identify the woodsmoke constituents responsible for the observed toxicities, it is clear that inhalation of woodsmoke can have a significant impact on pulmonary homeostasis and/or exacerbations of ongoing lung disease processes.

SUMMARY, RECOMMENDATIONS, AND CONCLUSION

Even though woodsmoke is natural, it is not benign. Indeed, there is a considerable and growing body of epidemiologic and toxicologic evidence that both acute and chronic exposures to woodsmoke in developed country populations, as well as in the developing world, are associated with adverse health impacts.

Summary

Chemical composition. Woodsmoke contains thousands of chemicals, many of which have well-documented adverse human health effects, including such commonly regulated pollutants as fine particles, CO, and nitrogen oxides as well as ciliatoxic respiratory irritants such as phenols, cresols, acrolein, and acetaldehyde; carcinogenic organic compounds such as benzene, formaldehyde, and 1,3 butadiene; and carcinogenic cyclic compounds such as PAHs. Woodsmoke contains at least five chemical groups classified as known human carcinogens by the International Agency for Research on Cancer (IARC), others categorized by IARC as probable or possible human carcinogens, and at least 26 chemicals listed by the U.S. EPA as hazardous air pollutants. Among the currently regulated pollutants in woodsmoke, fine particles ($PM_{2.5}$) serve as the best exposure metric in most circumstances and, in addition, tend to be among the most elevated in relation to existing air quality standards.

Toxicology. Most available animal studies indicate that exposure to woodsmoke results in significant impacts on the respiratory immune system and at high doses can produce long-term or permanent lesions in lung tissues. Based on relatively few studies, these effects seem most strongly associated with the particle phase. Woodsmoke is also mutagenic and possibly carcinogenic in laboratory and field studies, but less so than coal smoke. Not enough is currently known to reliably distinguish the toxicological effects of different types of biomass smoke (e.g., smoke from combustion of wood versus agricultural wastes). More work in this area is needed so as to better understand the mechanisms by which adverse effects observed in exposed individuals might occur.

Exposures. Measured in the form of fine particles, significant woodsmoke exposures, mostly in winter, occur indoors and outdoors in all areas of the developed world where wood is used for residential heating and in fireplaces. Woodsmoke often comprises a significant fraction of ambient particle levels in such areas, on both a daily and an annual basis. In developing countries, such exposures occur indoors at concentrations that can be orders of magnitude greater than those observed in the developed

world. Wildland fires and agricultural burning can generate enormous quantities of smoke and can impact populated areas, albeit infrequently. Occupational exposures can be extremely high for wildland firefighters. Woodsmoke-specific chemical tracers provide the potential for increased understanding of the contribution of ambient woodsmoke concentrations to indoor and personal woodsmoke exposures in settings where multiple sources of fine particles are present. Continuous personal monitoring of woodsmoke pollutants (e.g., CO, PM) is useful for developing microenvironmental exposure models that could subsequently be combined with questionnaire data to more accurately predict individual-level exposures to woodsmoke when personal monitoring is not feasible.

Epidemiology: Wildland fires and agricultural burning. Although rarely combined with individual exposure assessment, a number of studies have found associations between wildfires and emergency room visits for both upper and lower respiratory tract illnesses, (including asthma), respiratory symptoms, and decreased lung function. In one study, particulate matter in wildfire smoke resulting from a major episode in Southeast Asia was associated with increased cardiopulmonary mortality, although this is the only study to have specifically evaluated mortality as an outcome in relation to wildfire smoke. Though less well documented, exposures to smoke from agricultural burning may also be linked with adverse respiratory outcomes, particularly exacerbations of asthma. Only one study has examined the efficacy of various intervention strategies to reduce exposure and possibly morbidity among the general population during wildfires. Thus, there are few data on which to base recommendations to the general population on effective measures to reduce exposures. Several studies have documented cross-seasonal effects of wildland fire smoke exposure on firefighters' lung function. Long-term consequences of repeated occupational exposures to such extraordinarily high concentrations of vegetation smoke have not been investigated, however.

Epidemiology: Residential wood combustion exposures in developed countries. Surprisingly relatively few studies examining the health impacts of woodsmoke have been conducted in developed countries, partly due to the difficulty of disentangling risks due to woodsmoke from those associated with other pollutants also present. In addition, most available studies are ecologic in design, limiting the ability to infer causality. Those that have been done, however, indicate that exposure to the smoke from residential woodburning is associated with a variety of adverse respiratory health effects, which are no different in kind and, with present knowledge, show no consistent difference in magnitude of effect from other combustion-derived ambient particles. The few studies that are available seem to indicate a somewhat smaller effect of woodsmoke on cardiovascular than respiratory effects, but the actual public health implications would depend on the background rates of these diseases as well as other factors. No studies seem to be available related to cancer endpoints in developed countries.

Exposures and epidemiology in developing countries. Exposures to biomass smoke are common in nearly half the households in the world that use wood, crop residues, or animal dung for cooking and heating. Although poorly characterized overall, such biomass smoke exposures are substantially higher than those in developed countries. In more than a dozen studies each, both chronic obstructive lung disease and acute lower respiratory tract infections have been strongly associated with these household exposures, leading to an estimate by WHO of 1–2 million premature deaths per year globally. Multiple studies have also shown relationships with tuberculosis, cataracts, adverse birth outcomes, lung cancer, and asthma. Biomass smoke is also an important part of outdoor air pollution in many developing-country cities, although no studies seem to have been done to separate out its impacts from those of other pollutants.

Hundreds of studies have examined the relationships between outdoor pollutants and disease around the world. To the extent that woodsmoke contributes to individual airborne chemicals, such as CO, nitrogen oxides, and benzene, the conclusions of these studies can be applied to those same chemicals in woodsmoke because, being specific molecules, they do not vary by source. It is less clear, however, whether woodsmoke-associated particles are differentially toxic relative to particles from other sources that have been dominant in most epidemiologic studies of ambient air pollution. Since particles are probably the single most important disease-associated constituent of woodsmoke,* an assessment of their hazard is crucial for evaluating the overall hazard of the woodsmoke mixture.

Perhaps because of long human associations with woodsmoke particles and the consequent perception that they are "natural" and thus somehow less hazardous than particles from modern sources, such as fossil fuel combustion, there has been some reluctance to treat them equally, for example, in emissions standards. This effectively constitutes a decision that woodsmoke particles are actually less hazardous per unit concentration than "average" ambient particles, such as those in diesel exhaust. Although the database is not as extensive as those for other major air pollutants, the weight of the evidence, consisting of animal and in vitro toxicology, the human exposure data, and epidemiologic studies of wildfires and of household wood combustion, indicates that woodsmoke particles are hazardous to human health. Specifically, our review suggests that there is sufficient evidence from the available literature that air pollution from biomass combustion is associated with a range of adverse respiratory health impacts and little evidence to suggest reduced or altered toxicity from these particles relative to the more commonly studied urban air PM. Most of the epidemiologic studies, however, have focused on respiratory health outcomes, in contrast to the recent emphasis on cardiovascular effects of urban

*In terms of health impact. The largest constituents of woodsmoke in terms of mass are, in order, CO_2 and CO.

and regional airborne particle exposures. Likewise, there are no toxicological data examining the effects of woodsmoke on cardiovascular outcomes. Thus, at present there are insufficient data to assess the extent to which ambient woodsmoke pollution might affect the circulatory system.

Since source apportionment studies show that woodsmoke is a major contributor to PM in many communities, it is likely that woodsmoke exposure plays a role in the spectrum of adverse effects linked to PM exposure. The large effects seen at higher exposures in the developing world provide additional evidence of the toxicity of woodsmoke. Further, there is evidence that biomass combustion globally is not decreasing and may in fact increase as a result of climate change-related increases in wildfires and shifts to the use of renewable fuels as the costs of fossil fuels, including natural gas, continue to rise. Although not reviewed here, the evidence of health impacts from exposures to the most well-studied biomass smoke (i.e., from burning tobacco) also strongly support this conclusion. Most relevant in this regard are the well-documented health impacts of environmental tobacco smoke (ETS or SHS, "second-hand smoke"), for which exposure levels are much closer to those typically experienced for woodsmoke in developed countries than exposures experienced by active smokers.

Recommendations for Further Research

Although many potential research avenues exist, here we note what we believe are the most critical gaps in our knowledge of the health effects of smoke from combustion of wood and other biomass:

Chemical composition

- Better understanding of the similarities and differences of smokes generated by combustion of different categories of biomass in different conditions, including wood and major crop residues.
- Utilization of recent advances in analytical chemistry (e.g., LC/MS techniques) to identify and quantify a wider range of chemicals in biomass smoke.
- Linkage of toxicological studies with comprehensive chemical composition measurements to identify specific chemicals or compounds classes responsible for the toxicity of biomass smoke.
- Fate of these chemicals in ambient air. Which ones break down and which ones linger?

Toxicology

- More long-term animal inhalation studies at concentrations relevant to indoor and outdoor woodsmoke levels found in both developed and developing countries, as well as comparative studies of both acute and chronic effects of exposures to particle phase woodsmoke and particles from other sources.
- Studies that provide information concerning the constituents associated with adverse health outcomes should be considered for developing intervention strategies.

Controlled human exposures

- More chamber studies that can elucidate the acute effects of high exposure to major types of biomass smokes.
- Following research in the second-hand smoke (ETS) literature, it would be useful to look for short-term physiologic responses to short-term changes in woodsmoke exposures in controlled or semicontrolled settings.

Exposure assessment

- More source and exposure apportionment studies are needed to determine the degree to which residential wood combustion contributes to both indoor and outdoor particle exposures in areas where wood smoke is likely to represent a significant source of particle exposure.
- Reliable biomarkers are needed to assist in epidemiologic studies.

Epidemiology

- Find better ways to combine source and exposure apportionment assessments in epidemiologic studies designed to determine the proportion of particle-associated health effects attributable to woodsmoke.
- Undertake studies among populations exposed primarily to woodsmoke particles, at least seasonally.
- Conduct studies focused on cardiovascular and cancer effects to compare with risks from fossil fuel-derived ambient particles, particularly for cardiovascular effects.
- More research is needed to assess both potential exposure reductions achievable through interventions and the real health impacts of such interventions to protect the public exposed to smoke from wildfires.

*Exposures and epidemiology: Developing countries**

- Accelerate efforts to quantify the potential exposure reductions and health benefits of practical interventions to control indoor exposures to biomass smoke, including better ventilation such as chimneys, better combustion, and better fuels.
- The high household exposures to biomass smoke common in developing countries present opportunities for research on heath effects of complex pollutant mixtures (fine and ultrafine PM, CO, benzene, PAH, etc.) of interest globally and to reduce significant health risks to hundreds of millions of the poorest people in the world.

*Because the primary emphasis of this article is not developing countries, only two recommendations are listed here.

Conclusions

Recognizing the limitations of current knowledge and need for additional information, we nevertheless offer preliminary answers to the questions raised in the introduction:

The hazards of woodsmoke as a mixture. Because woodsmoke is made up of such a large mixture of different chemicals, it is impossible at present to attempt to accurately assess its health impacts by simply summing the potential effects of individual constituents. (Indeed, there are few if any examples in which the effects of mixtures are fully reflected by the summed toxic potentials.) Particularly in high-exposure situations with fresh woodsmoke, as with occupational exposures or vegetation fire episodes, there may be a need to derive indices of exposure that take into account a range of toxic endpoints due to woodsmoke, for example, including acute-acting as well as chronic toxicants, so that appropriate protective actions can be adequately taken. Use of fine particles or any other single metric by itself may not be sufficient in these circumstances.

Woodsmoke particles. Nevertheless, at the present time fine particles may represent the best metric to characterize exposures to smoke from residential wood combustion and from wildfire smoke. There is no persuasive evidence that woodsmoke particles are significantly less dangerous for respiratory disease than other major categories of combustion-derived particles in the same size range. There is too little evidence available today, however, to make a judgment about the relative toxicity of woodsmoke particles with respect to cardiovascular or cancer outcomes.

Table 6 indicates that millions of people are exposed to smoke from household combustion of wood and other sources of biomass burning. Given the recent upward trend in the costs of oil and natural gas, it is likely that residential biomass combustion will become even more widespread throughout both the developed and developing world. More explicit efforts to reduce emissions from small-scale biomass smoke sources are likely to become even more important in the near future in order to meet air quality goals set to protect health.

Finally, returning to the questions posed at the start, we conclude that although there is a large and growing body of evidence linking exposure to wood/biomass smoke itself with both acute and chronic illness, there is insufficient evidence at present to support regulating it separately from its individual components, especially fine particulate matter. In addition, there is insufficient evidence at present to conclude that woodsmoke particles are significantly less or more damaging to health than general ambient fine particles.

Nevertheless, given the importance of woodsmoke as a contributor to particle concentrations in many locations, strategies to reduce woodsmoke emissions may be an effective means of lowering particle exposures. In addition, given the weight of toxicologic evidence, additional epidemiologic studies are needed to confirm our conclusions.

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Review

A Review of Community Smoke Exposure from Wildfire Compared to Prescribed Fire in the United States

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Received: 30 March 2018; Accepted: 9 May 2018; Published: 12 May 2018

Abstract: Prescribed fire, intentionally ignited low-intensity fires, and managed wildfires—wildfires that are allowed to burn for land management benefit—could be used as a land management tool to create forests that are resilient to wildland fire. This could lead to fewer large catastrophic wildfires in the future. However, we must consider the public health impacts of the smoke that is emitted from wildland and prescribed fire. The objective of this synthesis is to examine the differences in ambient community-level exposures to particulate matter ($PM_{2.5}$) from smoke in the United States in relation to two smoke exposure scenarios—wildfire fire and prescribed fire. A systematic search was conducted to identify scientific papers to be included in this review. The Web of Science Core Collection and PubMed, for scientific papers, and Google Scholar were used to identify any grey literature or reports to be included in this review. Sixteen studies that examined particulate matter exposure from smoke were identified for this synthesis—nine wildland fire studies and seven prescribed fire studies. $PM_{2.5}$ concentrations from wildfire smoke were found to be significantly lower than reported $PM_{2.5}$ concentrations from prescribed fire smoke. Wildfire studies focused on assessing air quality impacts to communities that were nearby fires and urban centers that were far from wildfires. However, the prescribed fire studies used air monitoring methods that focused on characterizing exposures and emissions directly from, and next to, the burns. This review highlights a need for a better understanding of wildfire smoke impact over the landscape. It is essential for properly assessing population exposure to smoke from different fire types.

Keywords: wildfire; prescribed fire; smoke; particulate matter; public health; exposure

1. Introduction

Wildfire has long been an important ecological process of our natural world, only requiring three ingredients—fuel, oxygen, and heat [1]. Prior to European settlement, many forests in the United States were historically shaped by wildfires [2]. Native Americans historically used wildfire as a vegetation management tool to increase density of edible plants, provide material for basketry, and control insects and plant diseases [3]. Historically, in the Western US, frequent fires of low severity burned on the forest floor and resulted in coniferous forests that are more vulnerable to the effects of fire [4]. In California,

Stephens et al. (2007) estimated that during the prehistoric period wildland fires emitted 47 billion kilograms of fine particulate matter ($PM_{2.5}$) annually [5].

Prescribed fire; planned and intentionally ignited low-intensity fires, and managed wildfires; wildfires that are allowed to burn for land management benefit, could be used to treat the abundance of fuel in forests and restore fire-adapted landscapes across a larger area [2]. However, smoke-caused air quality impacts and compliance to air quality regulations can be an impediment to the use of prescribed fire, and the public health impacts of the smoke that is emitted from wildfire and prescribed fire must be considered [2,6]. Wildfire smoke can contain fine to inhalable particulate matter ($PM_{2.5}$–PM_{10}), acrolein, benzene, carbon dioxide, carbon monoxide, formaldehyde, crystalline silica, total particulates, and polycyclic aromatic hydrocarbons (PAHs) [7,8]. Individuals can be exposed occupationally, if they work as wildland firefighters, or from ambient air that is contaminated with smoke from a nearby or distant wildfire [9].

Past health studies of wildfire exposure have generally examined the relationship between exposure to $PM_{2.5}$ from wildfire smoke and associated adverse health outcomes [9,10]. Fine particulate matter is derived primarily from combustion and can absorb and retain toxic substances, such as volatile and semi-volatile organics (PAHs and quinones), transition metals, reactive gases (ozone and aldehydes), and sulfate and nitrate particles [11,12]. Particulate matter can be deposited in the human respiratory tract through three main mechanisms—impaction, sedimentation, and diffusion [13]. Inhalable particles with diameters of 0.5 to 2 μm are deposited in the respiratory tract through sedimentation. Larger particles, usually up to 10 μm in diameter, are deposited in the respiratory tract through inertial impaction, whereas smaller particles <0.5 μm are deposited though diffusional deposition [14]. Fine particulate matter can be deposited in respiratory bronchioles and alveolar regions where gas exchange occurs in the human lung [13,14]. There is evidence that $PM_{2.5}$ can cause adverse health outcomes through multiple biological mechanisms, such as increased local lung oxidative stress and inflammation, leading to acute and chronic respiratory effects; the lung inflammatory responses can spill over into systemic circulation contributing to acute and cardiovascular effects [15–18].

Although there are many epidemiological studies that have provided evidence of adverse health outcomes associated with long and short-term exposure to $PM_{2.5}$ in urban environments, there are fewer studies examining health outcomes and exposures to $PM_{2.5}$ from wildfire smoke. It is important to study exposures to $PM_{2.5}$ from wildfire smoke, as the chemical composition of $PM_{2.5}$ in wildfire smoke can differ from that of urban sources of $PM_{2.5}$ [8,9]. Previous studies have suggested that $PM_{2.5}$ from wildfire smoke causes adverse respiratory health effects and possibly increased mortality and cardiovascular health effects [19–22]. A recent systematic review of health impacts from wildfire smoke by Reid et al. (2016) found evidence that wildfire smoke was associated with respiratory morbidity, including exacerbations of symptoms of asthma and chronic obstructive pulmonary disease. There was some evidence, not conclusive, that wildfire smoke exposure is associated with respiratory infections and all-cause mortality [10]. Additionally, there are a few studies that found associations between wildfire smoke exposure and adverse birth outcomes, such as low-birth weight; however, these studies were limited and do not provide conclusive evidence. Holstius et al. (2012) demonstrated that average birth weight was slightly reduced among infants that were in utero during the 2003 Southern California wildfires [23]. Fann et al. (2018), estimated that wildfire events affected additional premature deaths and respiratory hospital admissions in Louisiana, Georgia, Florida, northern California, Oregon and Idaho. Additionally, the short and long term economic value of exposure to wildfire events were $63 and $450 billion (in present value), respectively [24].

Smoke from wildfire is inevitable, particularly in fire prone ecosystems. Exposure to smoke can to some extent be controlled by suppression and other anthropogenic actions. Historically, in the United States, full suppression has been utilized in an attempt to eliminate smoke and fire from the landscape [25]. The understanding that this practice is unsustainable has led to increased interest in using fire on the landscape to improve ecological health [26]. Human health is intrinsically coupled to

ecological health, but this relation is confounded by smoke exposure [27]. Understanding relative risk from fire management actions is essential to informed protection of public health.

The objective of this synthesis is to examine the differences in ambient community-level exposures from smoke in the United States from two smoke exposure scenarios—wildfire and prescribed fire. Several key questions will be addressed: (1) What are the $PM_{2.5}$ concentration differences between prescribed fire and wildfire smoke exposures? (2) How do $PM_{2.5}$ concentrations from each exposure scenario compare to the National Ambient Air Quality Standards (NAAQS)? (3) How long are communities exposed to $PM_{2.5}$ during each exposure scenario? This synthesis will provide public health practitioners, air quality regulators, and natural resource managers with more information on the exposure differences of smoke exposure from wildfire compared with prescribed fire. Ultimately, this information can be used to understand and quantify the health risks associated with smoke exposure from wildfire compared with prescribed fire.

2. Materials and Methods

A systematic search was conducted to identify scientific papers from peer-reviewed journals to be included in this review. The systematic search followed the Guidelines for Systematic Review and Evidence Synthesis in Environmental Management [28].

The Web of Science Core Collection and PubMed, for scientific papers, and Google Scholar were used to identify any grey literature or reports to be included in this review. The search strategy used the following search terms—wildfire, wildland fire, prescribed fire, grass fire, peat fire, prescribed managed fire, prescribed natural fire and smoke, exposure assessment, air quality. For each search that was performed, we recorded the search date, search terms that were used, database that was searched, and titles that were returned from the search.

The synthesis was restricted to scientific papers that met the following inclusion criteria: (1) studies that were conducted in the United States and (2) reported $PM_{2.5}$ concentrations during specific wildfire or prescribed fire events. Studies were appraised for the quality of the methods used for air monitoring or modeling used for concentration estimation. Studies that reported only $PM_{2.5}$ occupational exposures during a wildfire or prescribed fire event were not included.

The systematic search resulted in 271 journal articles from PubMed, with 229 unique titles, and 2023 journal articles from Web of Science, with 1093 unique titles (Figure 1). Once merged, there were 1449 unique scientific journal articles. Next, we reviewed the journal titles and selected 79 relevant articles. During the title review, reasons for articles to be excluded included: (1) were not conducted in the United States; (2) indicated a focus on developing models to estimate $PM_{2.5}$ emissions, source apportionment, or plumes; (3) conducted an occupational exposure study; (4) measured other air contaminants; (5) indicated that they were conducted in a laboratory. Of the selected articles, we reviewed their abstracts for extractable information that was relevant to the synthesis objectives. Based on the information provided in the abstracts, such as study methods and results, we selected the article to be further reviewed by reading the full article (N = 34). Sixteen peer-reviewed scientific journal articles met the study criteria and were included in this synthesis.

From each selected journal article, information was extracted and inputted into a table for comparison and analysis (Table 1). Extracted data from each article included: information on the wildfire or prescribed fire event name and date range, reported concentration mean and range, number of reported days that exceeded the NAAQS 24-h standard ($PM_{2.5}$ concentration $\geq$ 35 $\mu g\ m^{-3}$) [29], number of days sampled, the data source of the reported concentrations, and what type of average concentration average or sampling time was used for each study.

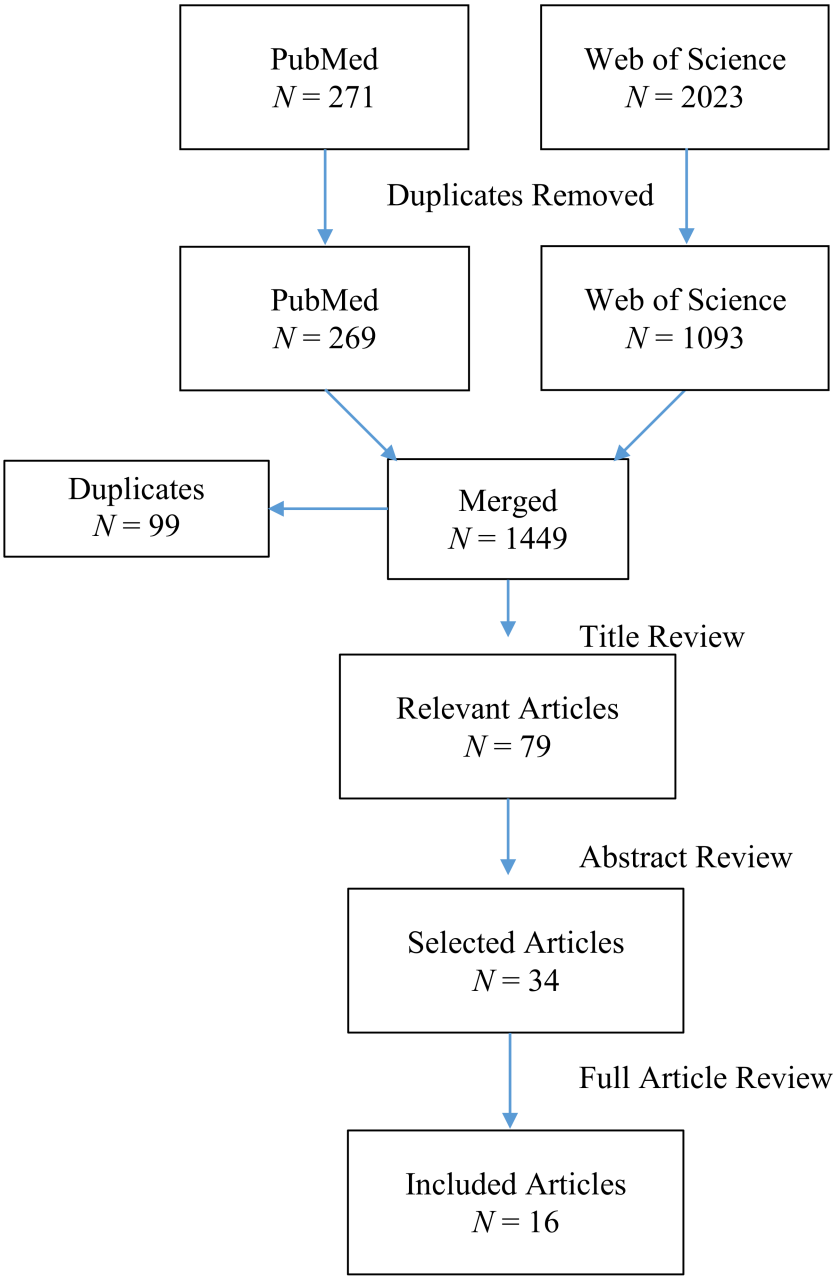

Figure 1. Flow diagram of study selection.

Table 1. Characteristics of included studies and answers to synthesis objectives.

Study	Event Location and Name, (Dates)	Fire Size (ha) [a]	$PM_{2.5}$ Concentration (μg m^{-3})		NAAQS Exceedance [b]	# of Days Sampled	Data Source	Sampling Time Range
			Mean	Range				
Wildfire Events								
Ward and Smith 2005 [30]	Montana Missoula Fire Season (8/13 and 8/25/2000)	-	39.9 and 42.2	Not Reported	2 days	2	Monitor	24 h Average
Ward et al. 2006 [31] [c]	Montana Missoula Wildfires (8/14–8/18/2003)	-	87.5	46–136.8	7 days	4	Monitor	24 h Average
	Montana Missoula Wildfires (8/31–9/2/2003)	-	54	37–69		3		
Viswanathan et al. 2006 [32]	California Cedar, Paradise and Otay Fires (10/26–11/4/2003)	113,424 22,945 18,988	Not reported	Max-104.6, 170	2 days	10	Monitor	24 h Average
Herron-Thorpe et al. 2010 [33]	Pacific Northwest Wildfires (7/3–8/22/2007) Pacific Northwest Wildfires (6/22–8/27/2007)	- -	16.8 15.9	Not reported Not reported	10 days 19 days	51 67	Model	24 h Average
Strand et al. 2011 [34] [d]	Idaho Frank Church Fire (8/11–9/14/2005) Washington Tripod Fire (7/24/2006–Mid Oct/2006) Region-fire wide event Western MT (8/2007–Mid Oct/2007) Region-fire wide event Northern CA(6/21/2008–9/2007)	22,194 70,820 - -	2–22 3–69 3–57 4–95	8–244 49–1659 21–575 28–472	3 days 47 days 11 days 40 days	13–77	Monitor	Hourly Average
Schweizer and Cisneros 2014 [35]	California Lion Fire (7/8–9/7/2011)	8370	7.7–20.1	Max-166.7	0 days	62	Monitor	24 h Average
Burley et al. 2016 [36]	California Aspen Fire (7/22–8/11/2013) California Rim Fire (8/17–10/24/2013) California French Fire (7/28–8/17/2014) California King Fire (9/13–10/9/2014)	9227 104,131 5202 39,546	41.5 8.7 14.4 6.6	11.7–92.7 1.3–69.9 7.9–21.9 1.6–27.8	13 days 2 days 0 days 0 days	20 49 20 26	Monitor	24 h Average
Navarro et al. 2016 [37]	California Rim Fire (8/17–10/24/2013)	104,131	6–121	1–450	Not Reported	49	Monitor	24 h Average
Zu et al 2016 [38]	Quebec Wildfires-Impacts in Boston (7/7–7/16/2002) Quebec Wildfires-Impacts in New York City (7/7–7/16/2002)	- -	23 25.2–27.3	4.1–64.5 4.8–84.2	Not Reported Not Reported	28 28	Monitor	24 h Average
Prescribed Fire Events								
Robinson et al. 2004 [39]	Arizona (Flaming Phase Samples) Oct/Nov 2001–2002 Arizona (Smoldering Phase Samples) Oct/Nov 2001–2002	20–80	Not reported	523–6459 155–904	Not Reported	6 6	Monitor	1.5–2 h Samples 4–51 h Samples
Lee et al. 2005 [40]	Georgia Prescribed Burn (4/15 and 16, 4/28 and 29/2004)	82–154	1810	Not Reported	Not Reported	4	Monitor	Total Average
Naeher et al. 2006, Achtemeier et al. 2006 [41,42]	Georgia Non-chipped plot (2/13/2003) Georgia Chipped plot (2/12/2003)	1 1	519.9 198.1	13.6–805.7 94.3–300.3	Not Reported Not Reported	1 1	Monitor Monitor	12 h Average 12 h Average
Hu et al. 2008 [43]	Prescribed Fire impacts on Atlanta (2/28/2007)	1200	37.8	NA	1 day	1	Model	24 h Average
Robinson et al. 2011 [44]	Northern Arizona Broadcast Burns (2001–2007) Northern Arizona Pile Burns (2001–2007)	10–40	2800 3000	523–8357	Not Reported Not Reported	15 6	Monitor	1–3 h Samples
Pearce et al. 2012 [45]	South Carolina Savannah River Site Burns (2003–2007)	10–1111	74.01	5.69–1415.96	Not Reported	55	Monitor	22 h Average

[a] Fire size is reported for studies that examined specific fire events; [b] Days that were reported to be above the US EPA NAAQS for $PM_{2.5}$ (35 μg m^{-3}) [29]; [c] Ward et al., (2006) [31] used PM_{10} monitoring concentration data to estimate $PM_{2.5}$ concentrations; [d] Strand et al. (2011) [34] reported hourly median and maximum concentration, and these values are used in place of the concentration mean and range, respectively. PM: particulate matter; NAAQS: National Ambient Air Quality Standards.

3. Results

The systematic review identified 16 studies that characterized exposures to $PM_{2.5}$ from wildfire and prescribed fire events (Table 1). Generally, studies directly measured $PM_{2.5}$ concentrations with existing air monitoring networks or temporary monitoring stations placed in communities that were deployed specifically for fire events. Although there were studies that attempted to model concentrations of $PM_{2.5}$ from wildfire or prescribed fire smoke, they did not report $PM_{2.5}$ concentrations associated with a specific fire event and did not meet the inclusion criteria.

The systematic search identified nine scientific studies that examined exposure to $PM_{2.5}$ from wildfire smoke. The studies covered a wide geographic area and were focused on wildfires that occurred in California, Montana, the Pacific Northwest, and Canada that impacted major cities in the United States. The selected papers reported $PM_{2.5}$ concentrations from several large wildfires (region-wide events), occurring at one period or during specific wildfire events. For example, Ward et al. (2006) measured $PM_{2.5}$ concentrations in Missoula, Montana, while 298,172 ha burned throughout all of Montana [31].

In the five studies that examined the impacts of specific wildfire events, the wildfires ranged in size from 5202 to 113,424 ha for the French and Cedar fires in California, respectively. Only three studies reported where the $PM_{2.5}$ monitors were located in relation to the fire events. Strand et al. (2011) [34] deployed monitors in local communities and small towns, at a minimum of 12 to 36 km from the fire locations in Idaho, Washington, Western Montana, and Northern California. Navarro et al. (2016) and Schweizer et al. (2014) [35,37] both used permanent and temporary monitors that were located 7–189 km from the Rim Fire and 16.6–242.8 km from the Lion Fire, respectively.

Eight studies that were selected used direct air monitoring methods to assess $PM_{2.5}$ exposures, while Herron-Thorpe et al. (2010) [33] used a modeling approach to estimate $PM_{2.5}$ concentrations from specific wildfire events during 2007 in the Pacific Northwest. From the data extracted from the studies, we focused on comparing studies that used the same averaging time (24 h average) to calculate a mean and range of $PM_{2.5}$ concentrations. Mean $PM_{2.5}$ concentrations from wildfires ranged from 8.7 to 121 $\mu g\ m^{-3}$, with a 24 h maximum concentration of 1659 $\mu g\ m^{-3}$. The 2013 Rim Fire and 2003 Montana Fires reported the highest mean $PM_{2.5}$ concentrations of 121 and 86.5 $\mu g\ m^{-3}$, respectively [31,37]. On average, $PM_{2.5}$ concentrations from wildfires were sampled and reported for 30 days; events ranged from 2 to 77 days. During wildfire events, the number of days that exceeded the NAAQS ranged from 2 to 47 days and averaged 11 days. The $PM_{2.5}$ concentrations from the Tripod Fire smoke in Eastern Washington resulted in 47 days that were above the NAAQS [34].

Seven scientific studies were identified that measured exposure to $PM_{2.5}$ at prescribed fires in Arizona, Georgia and South Carolina. Six studies used air monitoring equipment to measure $PM_{2.5}$ concentrations, while one study Hu et al. (2008) [43] simulated $PM_{2.5}$ concentrations using fire and atmospheric conditions from a specific prescribed fire event. Almost all sampled prescribed fires were performed as broadcast burns, where fire was applied directly across a predetermined area and was confined to that space. One sampled prescribed fire was conducted as a pile burn operation, where only piles of cut vegetation are ignited and burned [44]. Naeher et al. (2006) and Achtemeier et al. (2006) [41,42] reported $PM_{2.5}$ concentrations from the same prescribed fire event where researchers examined the effects of mechanical chipping on smoke measurements. The size of the prescribed fires ranged from 1 to 1200 ha, with the largest event being two adjacent prescribed fires in the Southeast United States, outside of Atlanta (Hu et al., 2008) [43].

Generally, the prescribed fire air sampling occurred during the burn operation and monitors were placed inside or next to the fire perimeter. For example, Robinson et al. (2011) [44] placed monitors next to the fire perimeter on Day 1 of sampling and inside the fire perimeter on Day 2 to capture emissions during the smolder phase of the fire. Naeher et al. (2006) and Achtemeier et al. (2006) [41,42] also placed monitors inside the prescribed fire and along the fire perimeter on the downwind side of the prescribed fire burn unit. Pearce et al. (2012) [45] measured concentrations using a grid of 18 monitors that were placed 10–12 km on the downwind side of the prescribed fire burn unit. Hu et al. (2008) [43]

was the only study to report $PM_{2.5}$ concentrations from a prescribed fire in an urban center—Atlanta, Georgia—which was 80 km from the prescribed fire.

Reported mean concentration of $PM_{2.5}$ from the selected studies ranged from 37.8 μg m^{-3}, in Atlanta, Georgia, to 3000 μg m^{-3} at a prescribed fire in Arizona [43,44]. Additionally, the same prescribed fire in Arizona during the flaming phase produced the highest maximum $PM_{2.5}$ concentration of 8357 μg m^{-3} [44]. Only Hu et al. (2008) [43] examined the impacts of a prescribed fire on NAAQS exceedances and reported that one day exceeded the NAAQS (24 h mean = 37.8 μg m^{-3}) during the prescribed fire event. Unlike the wildfire studies that generally used a consistent averaging time (24 h), prescribed fire studies averaged concentration over many different time periods. Averaging times ranged from 1.5–2 h samples to a four-day total average.

4. Discussion

Due to differences in study objectives and methodology, $PM_{2.5}$ concentrations from wildfire smoke were found to be lower than reported $PM_{2.5}$ concentrations from prescribed fire smoke. Although the acres burned on wildfires was up to 100 times larger, monitoring location, distance and concentration averaging time was shown to have an impact on the reported $PM_{2.5}$ concentrations. Wildfire studies focused on assessing air quality impacts to communities that were close to the fire (for example 12–36 km) and urban centers that were far from the wildfire. However, prescribed fire studies used air monitoring methods that focused on characterizing $PM_{2.5}$ exposures and emissions directly from, and next to, the burns site.

Wildfire and prescribed fire smoke exposure, similar to other emissions, is dependent on proximity to the source. Wildfire studies that were examined measured smoke at locations that ranged from 7 to 242.8 km from the wildfires, while prescribed locations ranged from next to the burn perimeter (0 km) and up to 80 km away from the burn. The dependence on proximity and smoke direction was demonstrated by Burley et al. (2016) [36], showing that megafires, such as the Rim and King fires, largely missed their monitoring site due to smoke plume direction, while the smaller and closer Aspen Fire transported more directly and had the highest exposure impacts at Devils Postpile National Monument. Hu et al. (2008) [43] was the only prescribed fire study identified that assessed the air quality impact from $PM_{2.5}$ to a large urban area. The 24-h $PM_{2.5}$ concentration in an urban area (Atlanta, Ga) that was estimated from this prescribed burn was 37.8 μg m^{-3} and in the range of the measured wildfire concentrations. In addition, the distance of the burn (80 km) was also similar to the monitor distance for wildfires.

The selected wildfire studies largely reported $PM_{2.5}$ mean concentrations that were generally averaged over a 24 h time period. However, the prescribed fire studies reported mean concentrations that were sampled over time periods ranging from 1–96 h. The short duration prescribed fire sampling events resulted in mean concentrations (198.1–3000 μg m^{-3}) that were higher than the prescribed fires that reported 22–24 h average $PM_{2.5}$ concentrations (37.8–74.01 μg m^{-3}). The shorter prescribed fire sampling events captured the periods of higher smoke emissions, while the longer averaging time for wildfire studies resulted in lower mean $PM_{2.5}$ concentrations.

Wildfire exposures are often episodic and short-term, but if they happen often, over a course of a fire season over many years, they could be considered long-term exposures. From the studies that were reviewed, the wildfire events that were included occurred over multiple weeks and months, while the prescribed fire events occurred over a few days. The duration of an event is important to consider because the longer exposure durations can lead to higher cumulative exposures to air contaminants [46].

This review highlights the lack of consistent information about exposures to $PM_{2.5}$ from fire smoke, especially from prescribed fires. Monitoring for prescribed fire was more focused on capturing the smoke emission directly next to the fire and not downstream from the burn, while wildfire studies either used existing urban sites and/or monitored for sensitive receptors. There were many studies identified during the initial search that have assessed smoke from wildfires or prescribed fires,

but there were few studies that directly reported concentrations of $PM_{2.5}$ to meet the inclusion criteria. Characterization of $PM_{2.5}$ air quality impacts to communities from prescribed fire smoke is needed to better understand how $PM_{2.5}$ exposures are different compared to those of wildfires. Prescribed fire exposure studies should be designed to examine emissions directly from the burn but also consider and measure the impacts on downwind communities. Additionally, one could use an area of the United States that is prone to frequent wildfires and estimate exposure through modeling from recent specific wildfires and prescribed fires to examine exposure differences. This approach was suggested by Baker et al. (2016), as it would lead to better model inputs for fire size and emissions, and could be validated against an existing monitoring network [47]. An additional approach that could be used would be a health impact assessment used by Fann et al (2018) [24] to estimate the incidence and economic value of human health impacts attributable to wildfire smoke compared to prescribed fire smoke [24]. Lastly, improved exposure estimates could be used to quantify the risk of adverse health effects from each of these different exposure scenarios [48].

5. Conclusions

Destructive wildfires have higher rates of biomass consumption and have greater potential to expose more people to smoke than prescribed fires. Naturally ignited fires that are allowed to self-regulate can provide the best scenario for ecosystem health and long-term air quality. Generally, prescribed fire smoke is much more localized, and the smoke plumes tend to stay within the canopy, which absorbs some of the pollutants, reducing smoke exposure. Land managers want to utilize prescribed fire as a land management tool to restore fire-adapted landscapes. Thus, additional work is needed to understand the differences in exposures and public health impacts of smoke of prescribed fire compared to wildfire. One way to do this would be for managers to collaborate with air quality departments (internal to agency or external) to monitor $PM_{2.5}$ concentrations in communities near a prescribed fire.

Consistent monitoring strategies for all wildland fires, whether prescribed or naturally occurring, are needed to allow the most robust comparative analysis. Currently, prescribed fire monitoring is often focused on capturing the area of highest impact or characterizing fire emissions, while wildfire monitoring often relies on urban monitors supplemented by temporary monitoring of communities of concern. A better understanding of smoke impact over the landscape and related impacts is essential for properly assessing population exposure to smoke from different fire types.

Funding: This research was funded by United States Department of Agriculture Forest Service Pacific Southwest Research Station: #A17-0121-001.

Acknowledgments: We would like to thank Penny Morgan for feedback on an earlier draft of this review. This work was supported by the United States Department of Agriculture Forest Service Pacific Southwest Research Station (#A17-0121-001). The manuscript reflects solely the opinion of the authors and not of the funding source.

Conflicts of Interest: The authors declare no conflicts of interest.

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toxics

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Sustained Effects on Lung Function in Community Members Following Exposure to Hazardous $PM_{2.5}$ Levels from Wildfire Smoke

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Received: 9 July 2020; Accepted: 1 August 2020; Published: 5 August 2020

Abstract: Extreme wildfire events are becoming more common and while the immediate risks of particulate exposures to susceptible populations (i.e., elderly, asthmatics) are appreciated, the long-term health effects are not known. In 2017, the Seeley Lake (SL), MT area experienced unprecedented levels of wildfire smoke from July 31 to September 18, with a daily average of 220.9 μg/m^3. The aim of this study was to conduct health assessments in the community and evaluate potential adverse health effects. The study resulted in the recruitment of a cohort (n = 95, average age: 63 years), for a rapid response screening activity following the wildland fire event, and two follow-up visits in 2018 and 2019. Analysis of spirometry data found a significant decrease in lung function (FEV_1/FVC ratio: forced expiratory volume in first second/forced vital capacity) and a more than doubling of participants that fell below the lower limit of normal (10.2% in 2017 to 45.9% in 2018) one year following the wildfire event, and remained decreased two years (33.9%) post exposure. In addition, observed FEV_1 was significantly lower than predicted values. These findings suggest that wildfire smoke can have long-lasting effects on human health. As wildfires continue to increase both here and globally, understanding the health implications is vital to understanding the respiratory impacts of these events as well as developing public health strategies to mitigate the effects.

Keywords: wildfire smoke; community; spirometry; health effects

1. Introduction

Wildfires have become a major global concern, and in the United States (US) there are hundreds of thousands to millions of acres burned [1,2]. Consequently, wildland smoke emissions are progressively being recognized as a public health concern, due to large scale wildfire fire events [3]. The increased number of these events are attributed to anthropogenic climate change, including warmer temperatures, early spring melt, and decreased winter precipitation [4]. Lightning and human ignition of excess forest fuels from years of previous fire suppression activity, as well as forest management practices, have contributed to large scale wildland fire events [5]. It has been projected that there will be an ~50% increase in burned areas across the western US between 2009 and 2050 and future predictive models show that this area will continue to see rapidly growing fire activity with increases of 80% burned areas in the Pacific Northwest alone [6,7]. While the western states (Washington, Oregon, Montana, Idaho, California, Wyoming, Nevada, Arizona) shoulder a majority of fires/acres burned (7 million+ in 2017), the Midwest and South had hundreds of thousands of acres of wildfires in 2017. Because of fire location and prevailing wind patterns, western Montana communities in the Northern Rockies are

annually inundated with smoke from increasing seasonal wildfires [8–10], and the region presents an opportunity to study the health effects of wildfire smoke exposures in historically at-risk communities.

Given recent climate trends, a growing incidence of 'historic' fires is appearing to represent the new normal in the western US. While most fire seasons result in significant levels of exposures, the 2017 Seeley Lake region in Montana experienced an unprecedented level of smoke exposure from nearby wildfires in terms of sustained PM concentrations. Due to numerous factors, including close proximity to multiple fires and the presence of significant inversions (atmospheric conditions that trap pollution closer to the ground), community residents were exposed to EPA-designated "very unhealthy" and "hazardous" $PM_{2.5}$ levels for 35 of the 49 days of exposure (1 August–19 September) in the summer of 2017 with a median 24 h average of 220.9 mg/m^3 for the entire period. Historically, studies attempting to assess the health effects of wildfire smoke in local communities have focused on medical records including emergency department visits, hospital admissions, or provider visits categorized with specific ICD codes for respiratory or cardiovascular diagnoses [11–15]; however, the long-term human health implications of these exposures have not previously been assessed. Wildfire smoke exposure is ascribed to an average 339,000 deaths each year, and studies have reported that wildland smoke PM is associated with respiratory effects [16–18]. The present study addresses this gap in knowledge with two-year follow up of evaluating community members exposed to these significant levels of wildfire smoke in Seeley Lake, MT.

During the 2017 wildfires, the Missoula City-County Health Department, Division of Environmental Quality (MCCHD-DEQ) contacted the University of Montana about the exposed community. A multi-disciplinary team headed by the IPHARM (ImProving Health Among Rural Montanans) program in the School of Pharmacy at the University of Montana was assembled to enroll and screen community members in Seeley Lake, MT and for comparison, the similarly sized town of Thompson Falls, MT whose smoke exposure during the same time period was five-fold less $PM_{2.5}$. Participants were given multiple surveys in addition to screening of health parameters: blood pressure, pulse-oximetry, and spirometry. The present study focused on respiratory effects from exposure to wildfire smoke. Analysis of full spirometry testing on community members from Seeley Lake showed significant decreases in lung function parameters up to two years post exposure, with clinically significant decreases in FEV_1 and changes in the FEV_1/FVC ratio indicating obstruction. The present work represents one of the first of its kind to assess and follow an exposed cohort to determine potential long-term health effects of wildfire smoke.

2. Methods

2.1. Study Design

The study was designed to enroll and assess multiple health parameters of persons living in Seeley Lake, MT area following exposure to unprecedented levels of wildfire smoke during the summer of 2017. Using the IPHARM health screening program infrastructure, five screening visits were conducted: one initial screening in 2017 within 24 h following the last day of elevated smoke, two in 2018 and two in 2019. Screening tests included spirometry, blood pressure, heart rate, oximetry, subject survey data, and collecting blood and saliva samples for later epigenetic testing. An additional cohort was enrolled and similarly screened in July of 2018 in Thompson Falls, MT, USA.

2.2. Study Population

The study population consisted of male and female subjects living in Seeley Lake and Thompson Falls, MT, USA during the summer of 2017. Subjects were between 23 and 85 years of age. Exclusion criteria excluded persons under the age of 18, inability to answer survey questions, or inability to perform spirometry based on the following screening questions: 1. In the last 3 months have you had a chest injury or surgery involving the eye, ear, chest, abdomen, or been hospitalized for a heart attack? 2. Do you experience hemoptysis? 3. Have you had a respiratory infection, such as flu, pneumonia, bronchitis or

chest cold, in the last 3 weeks? 4. Have you ever had a pneumothorax? 5. Do you experience regular chest pain? 6. Have you ever had thoracic, abdominal, or cerebral aneurysms? An affirmative answer to any of these questions precluded a participant from the spirometry testing. Additionally, participants undergoing current treatment for hypertension were evaluated for control (i.e., <130/80) before undergoing spirometry testing. The study protocol was approved by the Institutional Review Board (IRB) at the University of Montana and participants provided informed consent. Initial study approval was obtained by the University of Montana-Missoula Institutional Review Board on 14 September 2017 (#185-17), with annual continuation approval on 23 August 2018 and 26 August 2019.

2.3. Particle Exposures

The MCCHD-DEQ has monitoring stations in Seeley Lake and Thompson Falls, MT. Daily $PM_{2.5}$ levels were chronicled using the EPA NowCast method which registered $PM_{2.5}$ concentrations for every 12 h and then calculated a weighted average of those hours. In Seeley Lake, the air quality monitor is located outside and in close proximity to the elementary school athletic field in the town and is an average of 1.755 miles from each participant's listed address. In Thompson Falls, the monitor is located near the parking lot of the high school and is an average of 4.74 miles from the participants' listed addresses.

2.4. Study Procedures

2.4.1. Surveys and Participant Screening

Clinical history was obtained through surveys with special reference to smoking habits; asthma; allergies; systemic cardiovascular and respiratory diseases; ownership of wood burning stoves; COPD/Emphysema; bronchitis; and past and current health history in two weeks prior to the screening event. General physical examination included height, weight, and arterial and brachial blood pressure measurements (PulseWave analysis system; SphygmoCor AtCor Medical).

2.4.2. Pulmonary Function Tests

Pulmonary function tests were performed according to NIOSH approved guidelines using a NIOSH approved spirometer, the ndd EasyOne spirometer (ndd Medical Technologies Inc., Andover, MA, USA) or the Vitalograph asma-1 monitor (Vitalograph, Inc., Lenexa, KS, USA) with testing conducted in a seated position. After screening spirometry was performed with the acceptability of each test determined by the NIOSH-certified tester and spirometry software. The EasyOne™ spirometer has an inbuilt test quality grading system (A–D, F) that provides feedback to the operator on test acceptability and repeatability. A goal was set for three acceptable tests of A or B session quality with a limit of no more than eight attempts. This corresponded to three acceptable tests with between-test repeatability of 150 mL or less, as per ATS/ERS criteria. The forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), and FEV_1/FVC ratio was calculated automatically by the spirometer as the percentage of predicted values based on age, height, and gender as defined by NHANES III prediction equation (third National Health and Nutrition Examination Survey, 1999) [16]. In addition, the lower limit of normal (LLN) was calculated using z-scores ((measured-predicted)/standard deviation), where z-score = −1.64 (5th percentile) is defined as the LLN [17]. Spirometry quality and lung function values were compared between testing events to assess the reliability and changes in results over time.

2.5. Data Analysis

Intergroup differences were evaluated using three-way ANOVA followed by Sydak's Test for comparing multiple group means while controlling type I error. Odds ratios were generated using 2×2 contingency tables with one factor being the presence or absence of a clinically significant decrease in FEV_1 over a year (>30 mL) and the other factor being the presence or absence of a health attribute (e.g., asthma, allergies, etc.). The dependent variable was simply the frequency of occurrence. Fisher's Exact

test was used to test for independence between factors with a probability of Type I error set at 5 percent (two-tailed). The 95% confidence intervals for the odds ratios were calculated using the Baptista-Pike method. Significance for odds ratios was determined by the confidence interval. If the 95% CI included '1' within the interval it was not statistically significant. Statistical significance of the frequency of clinically significant decreases in FEV_1 was determined by binomial probabilities, contrasting observed frequency of clinically deceased FEV_1 with the expected frequency for any given 1-year period.

3. Results

3.1. Cohorts

Random, volunteer participants were enrolled from two wildfire smoke-exposed communities in western Montana: Seeley Lake and Thompson Falls. Initial enrollment events were preceded by multiple methods of recruitment including flyers, community meeting announcements, online (Facebook), and word-of-mouth. In Seeley Lake, 95 participants were originally enrolled and included in the study with thirteen new participants added in 2019. Their demographics are summarized in Table 1. Sexes were fairly evenly divided between males and females (44 to 51) with almost all identifying as "white" (one participant identified as "white" and "Hispanic"). In addition, the vast majority of respondents indicated at least a minimum of a high school diploma level of education, and a distribution of household income levels with the majority in the $30,000–$75,000 range. In Thompson Falls, 24 participants were enrolled in the comparison cohort with the majority screened identifying as female (n = 19) and all identifying as "white". All patients had at least a minimum of a high school diploma level of education, and a distribution of household income levels with the majority in the $30,000–$75,000 range. Demographics are summarized in Table 1.

Table 1. Patient demographics.

Variable	Seeley Lake			Thompson Falls
	2017	**2018**	**2019**	**2018**
Participants	95	42	62	24
Age * (years)	63 ± 1.5	63 ± 2.1	64 ± 1.5	59 ± 2.5
Sex				
Male	44	18	26	5
Female	51	24	36	19
Race				
White	93	40	60	24
Asian	1	1	1	0
African American	0	0	0	0
Hispanic	2	1	2	0
Education				
Less than High School	2	1	1	0
High School Diploma or GED	25	11	14	5
Some College	27	12	19	7
College Degree	41	16	26	12
Income				
Less than $29,999	18	9	11	5
$30,000–$74,999	52	22	28	15
Greater than $75,000	18	11	18	3

* Data are given as mean with ± SE; * Not all participants completed all surveys.

3.2. Exposures

In order to describe the extent of the $PM_{2.5}$ exposures to the affected populations, the MCCHD-DEQ data was used to record the air quality throughout Montana during the wildfire season of 2017. The MCCHD-DEQ posts the $PM_{2.5}$ levels for the local air quality while using the EPA guidelines to determine air quality in regards to human health. Between the dates of 1 August and 19 September 2017, Seeley Lake experienced daily $PM_{2.5}$ averages as shown in Figure 1. The daily average values between these dates was 220.9 $\mu g/m^3$, while 35 of those days had daily $PM_{2.5}$ averages of >150 $\mu g/m^3$ which fell within the range of very unhealthy (150.5 to 250.4 $\mu g/m^3$ $PM_{2.5}$), and had a peak of 638 mg/m^3 which exceeded hazardous levels (250.5 to 500.4 $\mu g/m^3$ $PM_{2.5}$). For perspective, since 2013, there were only two years (2015 and 2018) with points above the daily threshold of 35 $\mu g/m^3$, with one of those years, 2015, showing multiple days but only a few approaching the 100 $\mu g/m^3$ level (data not shown). In summary, the $PM_{2.5}$ levels in Seeley Lake were very high during the 2017 season for a sustained period of time. During this same time period Thompson Falls, another community in the Northern Rockies region, located 50 miles northwest of Seeley Lake, experienced a daily $PM_{2.5}$ average of 47 $\mu g/m^3$, which is still above the EPA standard of 35 $\mu g/m^3$ in the unhealthy designation (Figure 1).

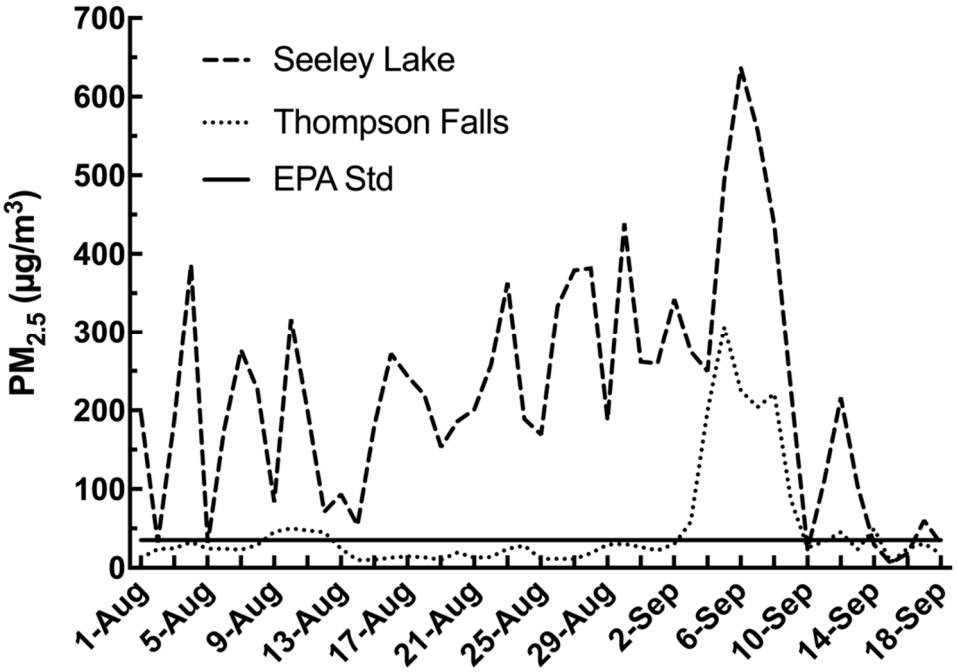

Figure 1. $PM_{2.5}$ levels in two western Montana communities during 2017 fires. The above graph illustrates the $PM_{2.5}$ levels during the peak wildfire time period for Seeley Lake and Thompson Falls. As can be seen, 2017 levels Seeley Lake were significantly above the NAAQS daily target of 35 $\mu g/m^3$ for almost the entire period with an average of 220.9 $\mu g/m^3$. In comparison, while Thompson Falls $PM_{2.5}$ levels followed a similar trend and the overall daily average was 47 $\mu g/m^3$, a large portion of the period saw daily levels below the target.

3.3. Lung Function Assessments

3.3.1. FEV_1/FVC Decrease

A large portion of studies assessing health effects of wildfire smoke have utilized hospital medical record databases and focused on respiratory and cardiovascular ICD-10 codes as these are considered the most likely affected outcomes. To this end, full spirometry was performed and assessed as described in order to evaluate impacts of the extensive wildfire smoke $PM_{2.5}$ exposures on lung function. At the initial visit to Seeley Lake in 2017, 59 of 95 participants were able to go through full spirometry testing.

Of the 36 without full spirometry two were unable to undergo the procedure as per the screening questionnaire (recent heart attack and pneumothorax one week prior) and the rest were due to time and personnel constraints. In the following year, 2018, 38 of 42 participants who returned were able to be reassessed with full spirometry. In 2019 there were 59 of 62 participants both new and old that performed spirometry. In 2018 and 2019 participants unable to undergo spirometry were those that answered in the affirmative to any questions during screening (including pneumothorax, abdominal aneurysm, collapsed lung, recent eye surgery, and current respiratory illness). The average lung function, FEV_1/FVC, for the total Seeley Lake cohort in 2017 immediately following the fires was 77.5% compared to the predicted average of 77.05%. However, the difference between observed and predicted changed dramatically in the subsequent years with FEV_1/FVC in 2018 (71.6% observed; 77.35% predicted) and 2019 (73.4% observed; 76.52% predicted) (>70% is considered normal) (data not shown). Examination of the variance between sexes, the average FEV_1/FVC values for males fell below those of females. The decrease for the population was significant with an additional significant difference between males and females (Figure 2). The data shows that in 2018 and 2019 values (FEV_1/FVC) for both sexes fell below their predicted values. In the comparison cohort there was also a significant decrease (−5.62%, $p < 0.001$) in the observed versus the predicted values in 2018, but no statistical variance between male and female (data not shown).

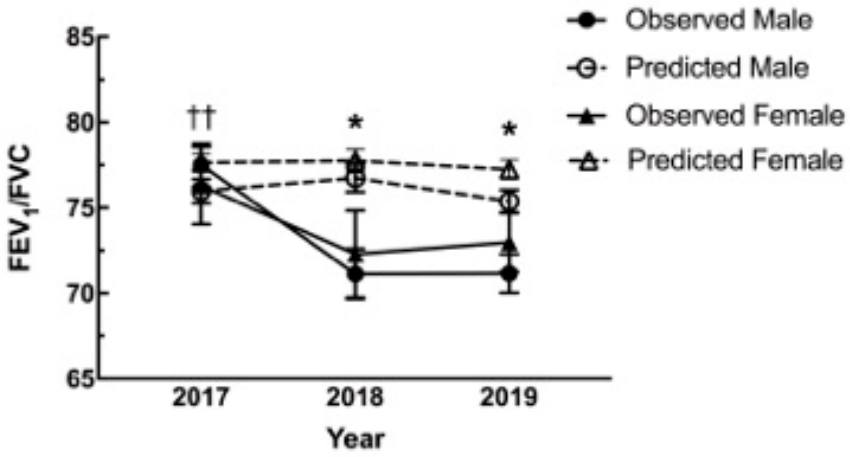

Figure 2. Pulmonary function changes from predicted values. The graph depicts the changes to lung function (FEV_1/FVC) in 2018 and 2019 following the Rice Ridge fire in Seeley Lake, MT. In both years following the 2017 exposure the observed (solid lines) was significantly lower than predicted (dashed) for males (* M, circles), while significantly lower in 2019 for the females (* F, triangles). In addition, the male values were significantly lower as compared to the observed values in 2017. (* $p < 0.05$ Observed vs. Predicted within Sex; †† $p < 0.01$ Significant compared to 2017 for corresponding group).

3.3.2. Lower Limit of Normal (LLN)

The NHANES III set of predicted equations was used for comparison purposes and the lower limit of normal was calculated using z-scores ((measured-predicted)/standard deviation). In 2017, six Seeley Lake participants fell below the LLN with one of the participants having (diagnosed) COPD. In the following year, 2018, 17 of the Seeley Lake participants had FEV_1/FVC values that fell below normal and in 2019 there were 14 Seeley Lake participants whose values fell below normal (Figure 3). For comparison, the Thompson Falls cohort exhibited a similar percentage of participants (45.8%) with FEV_1/FVC values below the LLN one year following the fires (Figure 3).

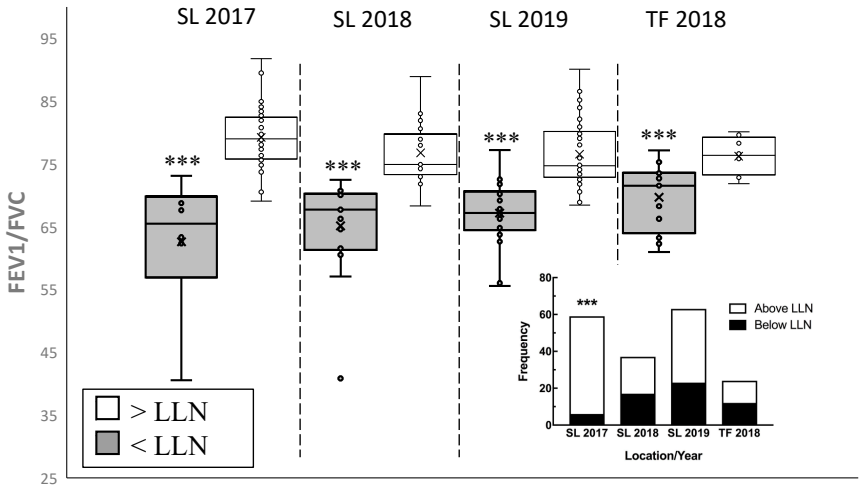

Figure 3. Community members falling below lower limit of normal for FEV_1. To determine clinically significant decreases in lung function, individual FEV_1 values are compared to the lower limit of normal (LLN) based on individual parameters (height, sex, age). LLN was calculated using z-scores (z-score < −1.64) and the following equation: (measured-predicted)/standard deviation. All three years of assessments in Seeley Lake and the one year in Thompson Falls found the average FEV_1 values for the participants below LLN to be significantly lower that of the rest of the cohort. In addition, there is a significant increase in proportion of participants falling below the LLN, contrasted to the 2017 SL values, as shown in the inset contingency table (n = 24–59, *** $p < 0.001$).

3.3.3. Peak Expiratory Flow

Additionally, peak expiratory flow (PEF) values were compared for all three years (2017–2019) in Seeley Lake and 2018 for Thompson Falls. As shown in Figure 4, while there was a small decrease in the year following the fire (2018), there were no significant changes in PEF for the Seeley Lake or Thompson Falls cohorts over the time of the study (Figure 4). This is routinely a highly variable measure of lung function and generally multiple values are averaged over a short time period for a more accurate assessment.

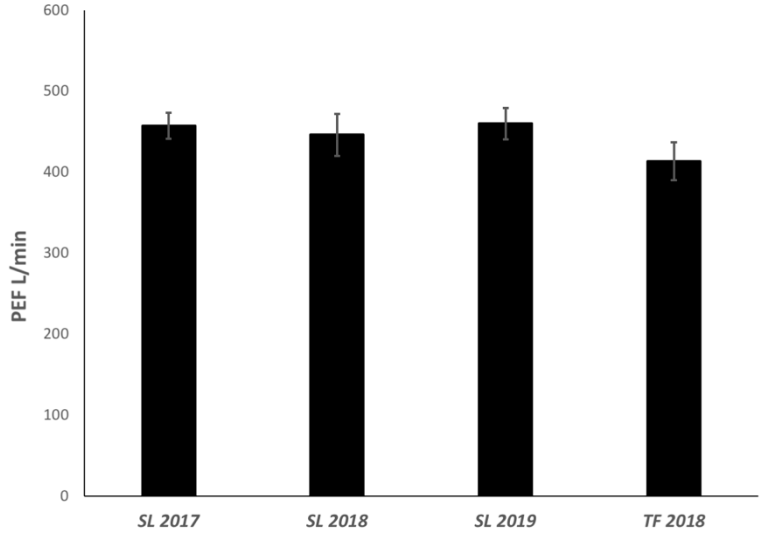

Figure 4. Peak expiratory flow values in the Seeley Lake and Thompson Falls cohorts. In the above graph the average peak expiratory flow (PEF) was calculated (±sem) for each year following the fires in 2017. Despite a slight decrease one year post exposure in 2018, there were no significant changes to the aggregate PEF values for the cohort and no difference with the control cohort in Thompson Falls.

3.3.4. Annual FEV_1 Decline

In Seeley Lake, from 2017 to 2018, 19 participants had FEV_1 values that decreased more than their expected annual decline and from 2018 to 2019, 18 participants had more than their expected annual decline as shown in (Table 2). From 2017 to 2018, there were 19 participants (Table 2) that had a clinically significant decrease in FEV_1 (>30 mL for males and >25 mL for females), which is the limit of acceptable loss of lung function per year [18]. From 2018 to 2019, there were 17 participants (Table 2) that continued to have decreased FEV_1 values that were greater than the expected decline per year. Overall, the FEV_1 values were decreased, with a greater effect on males, following the wildfires (Figure 5A). However, the average FEV_1 for all participants under the age of 65 was closer to their predicted FEV_1 averages, while the average FEV_1 for the >65 year-old participants was significantly lower compared with the <65 values (Figure 5B), suggesting the greatest effect is on the elderly.

Table 2. Annual change of FEV_1 in Seeley Lake cohort—males vs. females.

		n	Clinically Decreased (%)	Average Decrease (mL)
2017–2018	Total	37	19 (50%)	−0.231 ± 0.056
	Males	18	8 (44%)	−0.289 ± 0.114
	Females	19	11 (55%)	−0.208 ± 0.060
2018–2019	Total	30	17 (57%)	−0.123 ± 0.029
	Males	14	7 (50%)	−0.172 ± 0.043
	Females	16	10 (63%)	−0.135 ± 0.025

For the above table, "clinically decreased" is defined as a decrease of at least 25 mL for females and 30 mL for males over one year. The '*n*' correspond to the number of participants that have full spirometry results for both of the years indicated and the first number is for the period 2017–2018 and the second for the period 2018–2019.

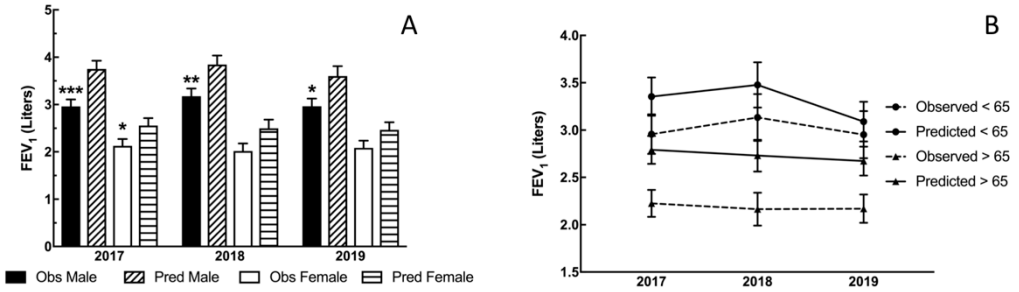

Figure 5. Annual changes between sex (**A**) and age (**B**) in FEV_1 in the Seeley Lake Cohort. Changes to FEV_1 from predicted values. The graph in panel A depicts the deviations from predicted of observed FEV_1 values in 2017, 2018, and 2019 following the Rice Ridge fire in Seeley Lake, MT. In all three assessments following the exposure the observed FEV_1 values were lower than predicted based on age, sex, race, and height (NHANES III–Hankinson 1999), and significantly lower all years for males and in 2017 for female participants. In addition, the male values were significantly lower as compared to the observed values in 2017. (** $p < 0.01$; *** $p < 0.001$ Observed vs. Predicted within Sex). The effect of age on FEV_1 changes is in panel B. The observed FEV_1 values versus the predicted FEV_1 for the Seeley Lake, MT cohort for the three visits (over two years) following the wildfires. The two groups depicted in the graph above are categorized as either >65 years old or <65 years old. All FEV_1 values were analyzed on individual parameters (age, sex, height) for each year. Both sets showed decreased, compared with predicted values, FEV_1 values. The younger group (<65 years) was not significantly lower and appears to approach predicted levels by the second year after the fires; while the older (>65 years) group remained significantly lower observed values for the >65 group (* $p < 0.05$).

3.4. Covariates

Multiple parameters in the Seeley Lake cohort, were evaluated as potential covariates including asthma, airborne allergies, emphysema/COPD, diagnosed cardiovascular disease, or the presence of a woodstove in the home. Analyses found no associations between allergies (pollen, dust, hay fever), emphysema/COPD, or the presence of a woodstove. While more than half (55 of 89) have a woodstove in their home, of the 37 participants that returned in 2018 for a second spirometry, half of those with stoves (10 of 20) had a significant decrease in FEV_1 (data not shown) and the average decrease was greater in the non-woodstove participants (289.3 vs. 189.7 mL). In addition, 39 of the full cohort in 2017 listed a history of allergies, but of the 37 participants that returned for a subsequent spirometry testing in either 2018 or 2019 less than half (8 of 19 and 7 of 18, respectively) had a significant decrease in FEV_1. With only four participants indicating a history of emphysema/COPD, only one of those presented with a significant FEV_1 decrease (data not shown). Lastly, of the participants listing a history of asthma, only 3 of 8 had a clinically significant FEV_1 change from 2017 to 2018, but all 8 decreased from 2018 to 2019, with seven presenting with a significant change consistent with a potential long-term risk for respiratory health effects of wood smoke exposures. In the assessment of potential risks from any of these factors, none had an odds ratio indicating an increase risk. However, while not statistically significant, with an odds ratio of 0.182, asthma is approaching being a risk of a smoke-induced decrease in FEV_1 in the long-term (i.e., after two years).

4. Discussion

Wildfires are a growing and significant concern globally. Most studies assessing potential health effects of exposures to the resulting smoke have focused on historical data of emergency department visits, hospital admissions, or provider visits [11–15]. The previous studies reported an increase in visits with ICD codes including cardiovascular and respiratory complications in the time frame following a wildfire event. In contrast, the present study was designed to evaluate and longitudinally follow a cohort of individuals in a community impacted with significant levels of smoke from wildfires. The cohort in Seeley Lake, MT is an older population (average age: 63 years) with a fairly even distribution of sexes (Table 1), while the comparison community of Thompson Falls had an average age of 59 years with the majority of patients being female. Participants were screened for inclusion, given health and demographic surveys, and underwent spirometry testing to assess potential effects on respiratory function parameters from the exposures.

Residents living in Seeley Lake, MT during the summer of 2017 were exposed to extremely high levels of $PM_{2.5}$ (Figure 1). $PM_{2.5}$ is a major component of air pollution and one of the criterion air pollutants designated by the EPA and has established the $PM_{2.5}$ cutoff to be 35.4 $\mu g/m^3$ for "unhealthy" designations. Seeley Lake had 35 consecutive days with $PM_{2.5}$ levels at 150.5 $\mu g/m^3$ (a designation of "very unhealthy") and above and 9 days where levels were greater than 250.4 $\mu g/m^3$ ("hazardous"). There were four fires burning within a 50-mile radius of Seeley Lake in 2017, contributing to the smoke exposure of the residents. The valley location of this community allowed for the smoke from the nearby wildfires to be trapped on the valley floor with temperature inversions, a weather phenomenon that occurs when cold air at night traps air pollution in a valley and prevents it from blowing away or rising higher in the atmosphere. In other recent studies examining health impacts of smoke from wildfire events, levels of $PM_{2.5}$ had not reached the levels of those in the Seeley Lake exposure. In Australia, during a particularly significant wildfire period in 2006/2007, the daily average for the two-month timeframe was 15.81 $\mu g/m^3$ (max. 294.95 $\mu g/m^3$) [15]; while during the 2007 San Diego fires there was a five-day average of 89.1 $\mu g/m^3$ (max. 803.1 $\mu g/m^3$) [13]. Our study's comparison community of Thompson Falls is also located in the Northern Rockies region, in the Clark Fork river valley. For the same time period in 2017 this community was also exposed to EPA designation of "unhealthy" levels of wildfire smoke (daily average of 47 $\mu g/m^3$ $PM_{2.5}$), however, it was 5-fold less average $PM_{2.5}$ than Seeley Lake, MT (Figure 1). These exposures are unprecedented and have afforded researchers the opportunity to follow a cohort longitudinally.

The most likely health effects from wildfire smoke exposures are on the respiratory and cardiovascular systems. To this end studies have generally taken the form of historical evaluations of medical records and ICD codes for respiratory and cardiovascular outcomes [11–15]. In the 2007 San Diego wildfires, increased respiratory medical encounters were found to correlate with peak smoke periods [13]. Likewise, Alman, et al. noted an increase in hospitalizations and ED visits for cardiorespiratory codes during the 2012 Colorado wildfires [11]. These studies are able to illustrate immediate effects of these exposures, but without individual longitudinal data we cannot appreciate, or identify, long-term complications of wildfire smoke exposures. The main physiological parameter assessed in the present study was lung function via spirometry testing. Spirometry is a common method of assessing pulmonary function that can be used to diagnose asthma, chronic obstructive pulmonary disease (COPD) and other respiratory pathologies. Spirometry is often used to evaluate lung physiology instead of X-rays and CAT scans because it can detect abnormalities in lung function even when no signs or symptoms of a disease are evident. When assessing lung function and the potential effects of environmental exposures, spirometry generates multiple parameters for comparison [19–21]. The main values utilized from spirometry assessments include, but are not limited to, FEV_1 (forced expiratory volume in the first second), FVC (forced vital capacity), and PEF (peak expiratory flow). The volume (FEV_1) is compared to the FVC volume, which is the total amount air exhaled during testing, and the FEV_1/FVC ratio is considered a reliable indicator of lung function. In analyses of these types of data, the ratios are age-matched and individuals are evaluated based on their lower limit of normal (LLN) [22–24]. In addition, FEV_1 values are used to determine whether lung function is declining at a normal rate based on age. According to the Mayo Clinic, the expected annual decline in pulmonary function in FEV_1 is 30 mL for males and 25 mL for women.

Both short-term and long-term studies of populations exposed to pollution have found significant correlation between fine particle pollutants and respiratory morbidity and mortality [25]. Exposures to $PM_{2.5}$ have also been consistently associated with decreases in pulmonary function in epidemiological studies [26]. Participants in our Seeley and Thompson Falls cohorts were assessed by an OSHA-certified staff scientist. The data from these studies suggest a significant decrease in lung function one year following the exposure and the decrease was maintained up to two years post smoke exposure (Seeley Lake). In Seeley Lake, a decrease in FEV_1/FVC (Figure 2) was observed in both 2018 and 2019 with a significant difference in males. In addition, there was an increase in the number of individuals that dropped below the LLN for this parameter (Figure 3). This type of change suggests an obstruction (as opposed to a restriction) that is in the category of asthma or COPD (chronic obstructive pulmonary disease). Participants were also evaluated for clinically significant annual changes in FEV_1 (>25 mL in females and >30 mL in males) where at least half of the participants showed a clinically significant decrease in FEV_1 with the largest average drop in the first year after exposure (Table 2). The decreased FEV_1 values in the Seeley Lake cohort at the initial screening suggests this parameter is more sensitive (temporally) to the smoke exposure (Figure 5A), and presents in a more vulnerable population (>65 years; Figure 5B). This decrease in FEV_1 means that the lung is restricted from filling to its normal capacity. These lung function changes, while being statistically significant are, more importantly, clinically significant as depicted in the annual decrease of FEV_1 volumes and the increase in the number of participants dropping below the LLN for the FEV_1/FVC ratio. In fact, the combination of these results are considered key in diagnosing obstructive changes [27]. In addition, while previous studies found that increases in 9–10 $\mu g/m^3$ increased the risk of asthma emergency department visits following exposure [13,15], the present study suggests a long-term implication for asthmatics. Because all seven asthmatics in the Seeley Lake cohort presented with decreased FEV_1 two years after the wildfire event, and 6 of 7 were clinically significant decreases, it suggests asthma as a risk factor for long-term complications and not just in the context of a short-term trigger.

Exposures to significant levels of wildfire smoke may result in obstructive lung pathology [28,29]. Past studies in air pollution have focused on the effects of pollutants on the airway epithelial lining and subsequent activation of the innate immune system. Recent studies have shown that ozone, a major

toxic air pollutant, induces IL-33 production by airway epithelial cells that results in activation of type 2 innate lymphoid cells (ILC2) [30,31]. The ILC2 are important sources of IL-13 [32] and have been linked to asthma, an obstructive lung pathology. Additionally, studies in both firefighters and in vitro studies reported increased IL-6, a contributor to inflammatory lung pathology [33,34], in response to wildfire or wildfire smoke extract, respectively [35,36]. The firefighters were assessed in the acute phase and found increased serum levels of IL-6, IL-8 and decreased IL-10, while the lung epithelial cultures presented with increased IL-6 production in addition to other markers of COPD including dysfunction of tight junctions. Therefore, the present study showing an increase in obstructive pathology based on spirometry results suggests a model of increased acute inflammation and activation of the innate immune system from wildfire smoke that results in tissue remodeling and decreased lung function (Figure 6).

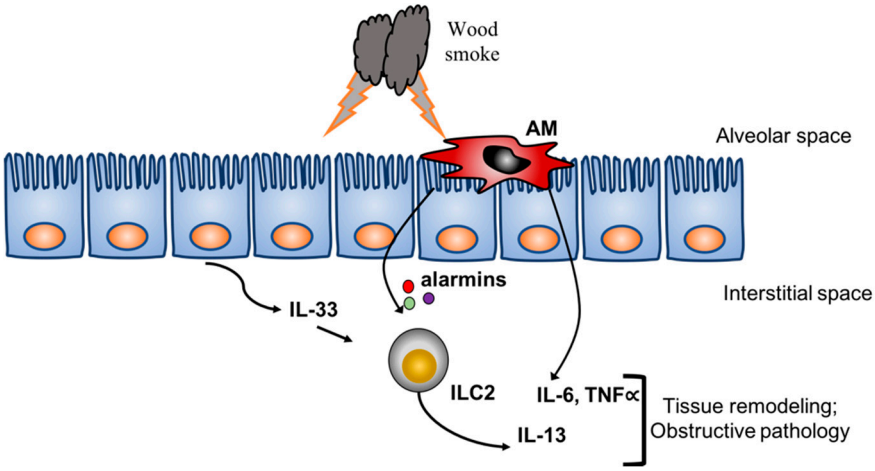

Figure 6. Theoretical molecular mechanism of the effects of wildfire smoke on pulmonary function. The working hypothesis is that both alveolar macrophages (AM) and lung epithelial cells directly interact with smoke particles. The combined responses result in production of key cytokines and alarmins that activate innate lymphoid cells (ILC2) and lung parenchyma resulting in tissue remodeling and an obstructive pathology (i.e., COPD, asthma).

5. Conclusions

Wildfires are increasing globally, both in duration and frequency and the potential for long-term implications must be considered in anticipating the public health response. It is vital to understand the long-term health implications of exposure to smoke from wildfire events. The observed changes in lung function parameters in our cohort illustrate the potential for long-term adverse health effects following a significant exposure to wildfire smoke. While the event in the present study was singular in its level and duration of smoke exposure, this is not the first, nor will it be the last exposure for these communities, due to the history of wildfires in this region (Northern Rockies) [8–10]. While the present study has shown a significant effect on the respiratory system of individuals in wildland smoke-exposed communities in the Western United States, it is important to note that this is an older cohort that is part of an historical at-risk population. The participants in the cohorts presented with altered lung functions categorized as an obstruction (decrease in FEV_1/FVC ratio) similar to asthma or COPD, and while not part of the design of the present study, the addition of bronchodilator testing would be key in determining the nature of the obstruction [18]. Additionally, the data suggests asthma as a potential risk factor of a longitudinal effect that warrants further research. In addition, these respiratory effects could have long-term health impacts on a variety of physiological systems. While beyond the scope of the present study, other biological systems need to be assessed for similar effects (i.e., cardiovascular, immunological) from these exposures. While the present cohort is categorized at-risk, studies have determined that mitigation strategies aimed at this group are cost-effective [37]. To expand

on these present observations, future studies will need to enroll additional age groups for comparison. Developing public health strategies to mitigate the risks to communities will be paramount in protecting the well-being of the impacted populations.

Author Contributions: A.O. collected patient data, contacted patients for follow-up, organizer of screening events, database generation/management, data analysis, contributor in writing manuscript. C.A.L.M. was an organizer of events, major role in patient contact/follow-up, database maintenance, major contributor in writing of manuscript. M.B. performed all spirometry and analysis of results, organizer of events, contributed to manuscript preparation. S.B. assisted with event organization, collected data, database generation, contributed to manuscript preparation. C.T.M. is PI on project, managed all personnel at events and was responsible for conduct of study, data acquisition/analysis, and manuscript preparation. All authors have read and agreed to the published version of the manuscript.

Funding: The present work was funded by HRSA U1QHP28733 and the National Institutes of Health/National Institute of Environmental Health Sciences (NIH/NIEHS) 1R21ES029679. Additional support was provided by NIGMS 1P30GM103338-01A1.

Acknowledgments: Authors thank Raymond Hamilton for assistance on statistical analyses and feedback on manuscript preparation.

Conflicts of Interest: The authors declare no conflict of interest.

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Published in final edited form as:

Sci Total Environ. 2018 January 01; 610-611: 802–809. doi:10.1016/j.scitotenv.2017.08.024.

The health impacts and economic value of wildland fire episodes in the U.S.: 2008–2012

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Abstract

Introduction: Wildland fires degrade air quality and adversely affect human health. A growing body of epidemiology literature reports increased rates of emergency departments, hospital admissions and premature deaths from wildfire smoke exposure.

Objective: Our research aimed to characterize excess mortality and morbidity events, and the economic value of these impacts, from wildland fire smoke exposure in the U.S over a multi-year period; to date no other burden assessment has done this.

Methods: We first completed a systematic review of the epidemiologic literature and then performed photochemical air quality modeling for the years 2008 to 2012 in the Continental U.S. Finally, we estimated the morbidity, mortality, and economic burden of wildland fires.

Results: Our models suggest that areas including northern California, Oregon and Idaho in the West, and Florida, Louisiana and Georgia in the East were most affected by wildland fire events in the form of additional premature deaths and respiratory hospital admissions. We estimated the economic value of these cases due to short term exposures as being between $11 and $20B (2010$) per year, with a net present value of $63B (95% confidence intervals $6-$170); we estimate the value of long- term exposures as being between $76 and $130B (2010$) per year, with a net present value of $450B (95% confidence intervals $42-$1,200).

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Disclaimer

The research described in this article has been reviewed by the National Health and Environmental Effects Research Laboratory, U.S. Environmental Protection Agency, and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the Agency, nor does the mention of trade names of commercial products constitute endorsement or recommendation for use.

Competing financial interests: The authors declare no competing financial interests.

Conclusion: The public health burden of wildland fires—in terms of the number and economic value of deaths and illnesses—is considerable.

Keywords

Health impact assessment; source apportionment; $PM_{2.5}$; ozone; CMAQ; wildland fires; wildfires

1. Introduction

The increasing frequency and intensity of large wildfires deteriorates air quality and adversely affects human health (Crimmins et al. 2016; Henderson et al. 2009; Liu et al. 2015, 2016; Westerling et al. 2006). These events in turn both promote, and are exacerbated by, long-term changes to the climate; current trends in these events are expected to continue (Crimmins et al. 2016; Stavros et al. 2014). While the level and type of pollutants emitted during wildfires vary according to region and fuel type, all fires release directly emitted particulate matter (PM) as well as precursors to fine particles ($PM_{2.5}$) and can contribute to downwind formation of ozone (Knorr et al. 2012).

While risks to human health from exposure to PM are especially well characterized in the epidemiological, toxicological and controlled human exposure literature (US EPA 2009), health impacts from PM stemming from wildland fires have been less extensively studied, though epidemiological literature has consistently observed adverse human health impacts attributable to wildfire-related $PM_{2.5}$ (Liu et al. 2015). For example, Rappold et al., (2012b, 2011) found that a peat fire episode in eastern North Carolina was associated with increasing numbers of Emergency Department visits for cardiopulmonary and respiratory outcomes. Similarly, Delfino et al., (2009) observed increasing rates of respiratory and cardiovascular hospital admissions resulting from a month-long wildland fire episode in southern California. Epidemiological studies conducted in other countries, including Australia, have observed similar affects (Johnston et al. 2007a; Morgan et al. 2010b). A systematic review of literature from the U.S., Australia and elsewhere by Liu et al., (2014) found that wildland fire-related coarse particles (PM_{10}) was most consistently associated with respiratory outcomes.

Despite the growing body of epidemiological studies, there are a relatively small number of air pollution risk assessments that attribute the number of premature deaths and illnesses and the economic value of health impacts to wildfire episodes. The risk assessments performed thus far have been limited in their temporal scope, using a single (2005) and projected (2016) year (Fann et al., 2013), or have been limited to examining one fire at a time (Jones et al. 2015; Kochi et al. 2012; Rappold et al. 2014; Rittmaster et al. 2006). This paper builds upon this literature to estimate the number and economic value of wildland fire $PM_{2.5}$-related premature deaths and illnesses in the contiguous United States using chemical transport model predictions of $PM_{2.5}$ from wildland fire episodes over a 5-year period beginning in 2008. Considering a national scope allows us to more fully capture the impact that wildland fires may have on human health.

2. Materials and Methods

In this study we characterized the overall magnitude and distribution of adverse health impacts by age and race that were associated with exposure to fire-$PM_{2.5}$ during wildfire smoke episodes. We used health impact functions derived from epidemiological studies that assessed the relationship between fire-$PM_{2.5}$ and expected incidence of health outcomes. We also performed a systematic literature review and meta-analysis; however, using results from the meta-analysis in a health impact function would have introduced considerable uncertainty into the estimates, and thus were not used.

2.1. Health Impact Function

The risk assessment employs a health impact function to quantify the number of wildland fire-attributable premature deaths and illnesses in each of the five years we modelled. We estimated the number of $PM_{2.5}$-related deaths and hospital admissions (y_{ij}) during each year i (i=2008, 2009, 2010, 2011, 2012) among individuals in each county j (j=1,…,J where J is the total number of counties) as:

$$y_{ij} = \Sigma_a y_{ija}$$

$$y_{ija} = m0_{ija} \times \left(e^{\beta \cdot C_{ij}} - 1\right) \times P_{ija},$$

where, β is the risk coefficient, m_{0ija} is the baseline death rate or hospital admission rate for the population in county j in year i among individuals for 5-year age strata a, C_{ij} is annual mean wildfire-attributable $PM_{2.5}$ concentration in county j in year i, and P_{ija} is the number of residents in county j in year i for five-year age strata a.

To perform a health impact risk assessment we used baseline incidence rates, population counts, and health impact functions included in the environmental Benefits Mapping and Analysis Program—Community Edition (BenMAP-CE, v1.1) (U.S. Environmental Protection Agency 2014) to estimate counts of $PM_{2.5}$ attributable deaths and respiratory hospital admissions in each of five years from 2008 to 2012. These inputs have previously been used to estimate health and economic impacts in the national ambient air quality standards reviews, and the methods have been validated in previous publications (Berman et al. 2012; Fann et al. 2011; Office of Air Quality Planning and Standards 2011). Below we describe how we specify inputs and use the BenMAP-CE tool with the appropriate input data.

2.2. Air quality modeling predictions

We simulated daily air quality from 2008 to 2012 using the Community Multiscale Air Quality version 5.1 (CMAQ v5.1) model with and without emissions from wildland fires in the contiguous United States. Wildland fires in our study included wildfires, prescribed fires, and other significant fires but it excluded agricultural fires. The difference between the two model runs represents the contribution of fire-$PM_{2.5}$ and $PM_{2.5}$ precursor emissions. Inputs

to the model included gridded meteorological fields, emissions data, and boundary conditions. Gridded meteorological fields were provided by annual CONUS Weather Research and Forecasting (WRF) model simulation. Meteorological fields were defined on a 12 ×12 km horizontal grid with 35 vertical layers of variable thickness extending up to 50 hPa. The lowest model layer, which extended to approximately 20m above ground was used to calculate the annual mean concentration of $PM_{2.5}$ (Δx) in the health impact function.

The CMAQ input emissions were based on a 12 km national U.S. domain with speciation for the Carbon-Bond 05 chemical mechanism (Yarwood et al. 2005). The emission inventory and ancillary files were based on the 2008 emissions modeling platform for 2008, 2009, and 2010 (EPA, 2012) and on the 2011 emission modeling platform for 2011 and 2012 (EPA, 2016). Since the focus of this study is wildland fires, any additional information about the non-fire emission sources is noted in the references. The fire emissions were based on year specific daily fire estimates using the Hazard Mapping System fire detections and Sonoma Technology SMARTFIRE system version 2 (Sonoma Technology 2007). Smartfire2 is a framework for producing fire activity data and allows for the merging of multiple data sources. Some of the fires included in the fire inventory come from satellite based remote sensing sources which cannot distinguish large prescribed or debris burning fires from wildfires with certainty. After multiple sources of fire information are reconciled to create fire activity estimates, the fire emissions are estimated using fuel moistures (via the USFS Wildland Fire Assessment System), consumption estimates from the Consume model (Ottmar, 2014; US Forest Service 2015), emission factors from the Fire Emission Production Simulator (Ottmar, 2014; US Forest Service 2015), and fuel loading from the United States Forest Service Fuel Characteristic Classification System (FCCS) database (McKenzie et al., 2012). Plume rise for all point sources including the wildland fires was calculated within the CMAQ model. Biogenic emissions were processed in CMAQ and are based on the Biogenic Emissions Inventory System v3.14 (Schwede et al, 2005; Carlton et al, 2011)

2.3. Effect coefficients

To identify $PM_{2.5}$ effect coefficients suitable for the health burden impact assessment, we consulted two sources of evidence. The first is the U.S. EPA's 2009 Integrated Science Assessment for Particulate Matter (PM ISA), which classified human health endpoints as having a "causal" or "likely to be causal" relationship with short-term and long-term exposure to $PM_{2.5}$. The PM ISA synthesizes the epidemiological, toxicological and controlled human exposure studies published to that point, and was peer reviewed by the independent Clean Air Scientific Advisory Committee (U.S. EPA, 2009). The PM ISA indicated that mortality and cardiovascular outcomes were causally related, and respiratory outcomes were likely to be causally related, to short-term and long-term exposure to fine particle levels (U.S. EPA 2009). The ISA did not differentiate these effects by particle composition or source.

Next, we performed a systematic review and quantitative meta-analysis. We included epidemiological studies that looked at associations between $PM_{2.5}$ during smoke events and various health endpoints. Studies identified were conducted in the U.S., Australia, South America, and Asia, and were limited to health endpoints identified by the PM ISA as being

causally related to $PM_{2.5}$ exposure. The systematic review employed a machine learning technique to identify relevant literature. We report detailed information regarding the search terms, our procedure for identifying and screening eligible literature, and a Preferred Reporting Items for Systematic Reviews and Meta-Analyses diagram in the Supplemental Materials. The machine learning literature review identified a total of 276 epidemiological studies, of which we judged 21 to be suitable to be included in the quantitative meta-analysis (Supplemental Figure 1). These 21 studies reported a total of 902 relative risks or odds ratios for respiratory (n=455) or cardiovascular (n=308) hospital or emergency department visits, or all-cause or non-accidental deaths (n=139).

A subset of 4 studies reported risk estimates for respiratory hospital admissions that reported effect coefficients for a common endpoint, PM indicator (in this case, PM_{10}), lag structure and population age strata and so were suitable for pooling in a quantitative meta-analysis (Henderson et al. 2015; Johnston et al. 2007b; Morgan et al. 2010a; Tham et al. 2009). Using the Metafor library in the R statistical package, we perform a random effects meta-analysis (R Core Team 2016; Viechtbauer 2010). A forest plot illustrating the studies included in the meta-analysis, a random-effects pooled estimate, and additional information regarding tests for funnel plot asymmetry can be found in the Supplemental Materials (Supplemental Figures 2 and 3).

Ultimately, for the health impact assessment, we did not use this pooled estimate because the exposure of interest for our nationwide risk assessment was $PM_{2.5}$, not PM_{10}. Additionally, the risk coefficients may not be generalizable because all 4 studies were based on wildland fire events outside of the U.S. (Henderson et al. 2015; Johnston et al. 2007b; Morgan et al. 2010a; Tham et al. 2009).Populations in other countries may respond differently to wildland fire episodes to those in the U.S., have access to a different healthcare system, may be more or less susceptible to wildland fire smoke, and may differ in other ways that we cannot observe using the available data. For these reasons, we used a risk coefficients drawn Delfino et al. (2009), which reported risk estimates for both respiratory and cardiovascular hospital admissions during the wildland fire episodes in Southern California.

We also selected $PM_{2.5}$ epidemiological studies that reported short-term and long-term $PM_{2.5}$ effect coefficients that did not specifically report effect estimates for exposures to wildland fire $PM_{2.5}$. We used risk estimates U.S. EPA previously employed to evaluate the health benefits of alternative air quality standards (EPA 2011), recognizing that these studies do not consider $PM_{2.5}$ particles originating from wildland fire events specifically and they generally consider $PM_{2.5}$ concentrations at levels significantly below those observed during wildland fire episodes. The short-term studies include Zanobetti et al. (2009), a multi-city time-series study that reported hospital admissions for respiratory outcomes, and Zanobetti and Schwartz (2009), a multi-city time series study of $PM_{2.5}$-related mortality. Because wildfire episodes can affect long-term levels of $PM_{2.5}$, we also employ effect coefficients from long-term epidemiological studies; these include an extended analysis of the American Cancer Society (Krewski et al. 2009) and an extended analysis of the Harvard Six Cities cohort (Lepeule et al. 2012).

2.4. Baseline rates of death and hospital admissions

The epidemiological studies noted above report estimates of risk that are expressed as being relative to a baseline rate. In this analysis we used effect coefficients to quantify cases of hospital admissions and premature deaths, and thus we applied baseline rates of rates of all-cause mortality and hospital admissions. We selected county-level age-stratified all-cause death rates from the Centers for Disease Control (WONDER) database for the year 2010 (Centers for Disease Control and Prevention 2016). We selected hospital visit rates from the Healthcare Cost and Utilization Program (HCUP); these are a mixture of county, state and regional rates (See Supplemental Table 2).

2.5. Assigning $PM_{2.5}$ concentrations to the population

We quantified changes in population-level exposure by assigning the predicted $PM_{2.5}$ concentrations to the population in each 12km by 12km model grid cell. The BenMAP-CE tool contains 2010 U.S. Census reported population counts stratified by age, sex, race and ethnicity, assigned to each air quality grid. We used the census-reported population counts for the years 2010 and then projected these counts to the years 2011 and 2012 using the Woods and Poole forecast (Woods and Poole 2012). Population data in each year are stratified by age, sex, race, and ethnicity.

To calculate a national wildland fire $PM_{2.5}$ concentrations for each of the five years that was weighted to the size of the population exposed to wildland fire $PM_{2.5}$ concentrations for all counties combined (C_i) in year i as

$$C_i = \frac{\sum_j C_{ij} \times P_{ij}}{P_i}$$

where C_{ij} is the wildfire-attributable annual mean $PM_{2.5}$ concentration in county j in year i, P_{ij} is the population in county j in year i, and P_i is the total population over all counties combined in year i.

2.6. Economic Values

We estimated the value of avoided premature deaths using a Value of Statistical Life (VSL) recommended by the U.S. EPA's Guidelines for Preparing Economic Analyses (US EPA 2010). Following U.S. EPA guidelines, we indexed this value to the inflation and income year of the analysis. Using a 2010 inflation year and assuming 2016 income levels, we calculated a VSL of $10.1M. To value changes in respiratory hospital admissions, we used a cost of illness estimate U.S. EPA employed in its Regulatory Impact Analysis for the $PM_{2.5}$ National Ambient Air Quality Standards (US EPA 2012). This value of $36,000 reflects the direct medical costs associated with the hospital visit as well as lost earnings.

3. Results

3.1. Air Quality

The median of the predicted annual mean wildland fire-attributable $PM_{2.5}$ concentrations across all model grid cells ranges from between 0.3 $\mu g/m^3$, in 2009 and 0.8 $\mu g/m^3$ in 2012, while the population-weighted annual mean $PM_{2.5}$ concentration ranges from between 0.6 $\mu g/m^3$ in 2009 to 1.1 $\mu g/m^3$ in 2008 (Table 1). In general, the distribution of wildfire attributable $PM_{2.5}$ concentrations are greatest in the year 2008 and lowest in the years 2009 and 2010 of the years included in this analysis.

A small number of states are most greatly affected by wildland fire events across the 5-year period (Figure 1). In the western U.S, states including California, Oregon, Idaho and Montana experience wildland fires in each year. Among the Southeastern states, North Carolina, South Carolina, Georgia, Louisiana, Arkansas, Florida and eastern Texas are most impacted.

3.2. Estimated health impacts and economic values of wildland fire events

We estimate between 5,200 to 8,500 respiratory hospital admissions per year (Table 2) (From 2008 to 2012) from wildland fires when using the concentration-response relationship from the Delfino et al. (2009) study. This range is within the same order of magnitude as the values estimated using a concentration-response relationship from the Zanobetti et al. (2009) multi-city study that did not explicitly account for wildland fire episodes (3,900 to 6,300). Using an effect coefficient from the Delfino et al. (2009) study, we also estimate between 1,500 and 2,500 cardiovascular hospital admissions. As noted above, we could not identify a suitable wildland fire study that reported a mortality effect coefficient and so used a risk coefficient from a multi-city time-series study (Zanobetti & Schwartz). We quantified between 1,500 and 2,500 wildland-attributable $PM_{2.5}$-related deaths from short-term changes in $PM_{2.5}$ concentrations over the five-year period (Table 2). We estimate the largest number of excess deaths and hospital admissions for the year 2008, when wildland fire attributable $PM_{2.5}$ concentrations are the greatest out of the years included here.

To provide context for the estimates above, in the secondary analysis we also applied $PM_{2.5}$ concentration-response relationships recently employed in U.S. EPA health impact assessments, estimating thousands of non-fatal heart attacks, thousands of respiratory and cardiovascular hospital admissions, hundreds of thousands of cases of upper and lower respiratory symptoms and millions of cases of acute respiratory symptoms (Supplemental Table 4).

Summing the economic value of the short-term premature deaths and hospital admissions we estimated a total dollar value of between $11B and $20B per year (2010$) (Table 3). The present value of these economic values across the 5-year time period is $63B (3% discount rate, 2016$). We estimated the value of the long-term $PM_{2.5}$ related premature deaths and hospital admissions to fall between $76B and $130B per year (2010$). The present value of these economic values across the 5-year time period is $450B (3% discount rate, 2010$).

3.3. The distribution of wildland fire-attributable health impacts among population subgroups

We next sought to better understand how the 5-year wildland fire episodes that we modeled in CMAQ were affecting populations across the U.S. For the 2008 to 2012 time period in which we modeled wildland fire episodes we mapped the cumulative CMAQ predicted wildland fire $PM_{2.5}$ concentrations as a distribution, and identified the upper 75^{th}, 90^{th} and 95^{th} percentile. We then identified the portions of the CMAQ modeling domain at or above this level across the continental U.S. (Figure 2). We next characterized the populations exposed to these elevated concentrations according to their race (Table 4).

When comparing the wildland fire-attributable $PM_{2.5}$ concentrations occurring among the highly affected and less affected areas, we found that: (1) in locations of the U.S. that are most affected by wildland fires, black populations represent a larger share of the individuals exposed to wildland fire $PM_{2.5}$ and a smaller share in those locations of the U.S. that are less affected by wildland fires; (2) by contrast, white populations experience a smaller share of the population in the highly affected areas and a larger share in the less affected areas.

3.4. Sensitivity Analysis

The estimated number of premature deaths and illnesses are sensitive to the predicted daily change in wildfire related $PM_{2.5}$ concentrations. Smoke plume height has been repeatedly identified as one of the critical sources of uncertainty in air quality modeling (Baker et al 2016).

This parameter in turn affects the range in which particles disperse and therefore the predicted level of concentrations are most sensitive at both very short and far distances from fires. Examining model performance indicated that the model tends to over-predict at low concentrations, possibly over representing small fires, as well as at very high concentrations. Over-dispersion of particles in the model can yield an excess number of low impact days that can have a cumulative impact on the estimate of exposure.

To understand the sensitivity of our results to the high number of days with low concentrations we quantified the number of wildland fire-attributable deaths from daily changes in $PM_{2.5}$ that were at least 1, 3 and 5 $\mu g/m^3$ in size (Supplemental Table 3). We find that when quantifying $PM_{2.5}$-related deaths on days in which wildland $PM_{2.5}$ concentrations are at or above 1 $\mu g/m^3$, the estimated number of premature deaths is on average 17% lower than when we do not apply this threshold; when estimating premature deaths occurring on days when $PM_{2.5}$ levels at or above 5 $\mu g/m^3$, there are about 44% fewer premature deaths. These results suggest that our results were sensitive to low levels of model-predicted wildland $PM_{2.5}$ concentrations.

4. Discussion and Conclusions

To our knowledge, this is the first manuscript to characterize the $PM_{2.5}$-related incidence and economic value of wildland fire impacts in the continental United States across an extended time period. The number of wildland fire-attributable $PM_{2.5}$-related hospital admissions, emergency department visits and other outcomes we estimated in this analysis

are comparable to those reported in Fann, Fulcher and Baker (2013) for the year 2016. That analysis, like this one, employed photochemical modeling surfaces and used a similar array of health impact functions. However, Fann, Fulcher and Baker (2013) calibrated the air quality modeling predictions to monitored air quality data, which would affect the level and distribution of the wildland fire-attributable $PM_{2.5}$ concentrations. Here we do not calibrate model predictions with observed data because it has been noted that high concentrations of particles during wildfires restrict air flow to the pumps, shutting the monitors down and missing high concentration episodes, thus leading to potentially biased observed values. We also recognize the potential for CMAQ to overestimate wildland fire impacts (Baker et al. 2016). More complex calibration and data fusion models specific to the wildland fires are likely to become available with the increased usability of remote sensing in this area of research as well as improved parameterizations based on results from field campaigns such as the Fire and Smoke Model Evaluation Experiment (FASMEE) (http://www.fasmee.net).

The epidemiological literature reporting risks from wildland fire-related $PM_{2.5}$ is sparse; this makes quantifying wildland-attributable risks challenging. The epidemiology studies that the U.S. EPA and others commonly use to quantify PM-related mortality and morbidity impacts, and reported in the Supplemental Materials to this article, were not explicitly designed to characterize risks from this emission source. However, it is reasonable to expect that many epidemiologic studies that did not explicitly address wildland fire impacts will have at least partially accounted for them, given that wildland fire particles tend to account for large portion of PM in the atmosphere (Verma et al. 2014). The National Emissions inventory of 2011 estimates that 41% of $PM_{2.5}$ emissions originate with wildland burning (U.S. EPA, 2011).

In light of this limitation, when we designed the health impact assessment, we took two steps to ensure that we were using effect coefficients that were well matched to the unique characteristics of wildland fire smoke events. First, we performed a random effects quantitative meta-analysis of respiratory hospital admission epidemiological studies of smoke events in the U.S. and elsewhere in the world. Second, we selected concentration-response relationships from epidemiological studies that we believe would better account for the episodic nature of smoke events and the corresponding elevated levels of particles (Zanobetti et al. 2009; Zanobetti and Schwartz 2009).

The overall economic value of wildland fire-attributable premature deaths and respiratory hospital admissions is considerable. Depending on whether we quantify short-term $PM_{2.5}$-related premature deaths or long-term $PM_{2.5}$-realted deaths, the cumulative 5-year economic value is in the tens to hundreds of billions of dollars.

Nearly all states in the U.S. experienced elevated fine particle concentrations from wildland fire events over the 5-year period, according to our simulation of air quality. Certain states were affected by severe wildland fire events occurring across two or more years, including Louisiana (some of which may be due to debris burning after an active hurricane season because remote sensing of fires cannot distinguish between large debris burning from wildfires or prescribed fires), California, Idaho, and Georgia. Within these states, certain population subgroups were affected disproportionately. In particular, black populations, and

to a lesser extent Asian populations, accounted for a greater share of wildland fire-attributable health impacts. State and local officials may wish to consider how best to communicate with subgroups that are most affected when deploying warning systems that alert the public to the health risks of wildland fire smoke. State and local officials deploying warning systems to alert the public to the health risks of wildland fire smoke may wish to consider how best to reach these subgroups.

This analysis is subject to certain limitations and uncertainties that shape the way in which the results may be interpreted. Considering first the air quality inputs, we assessed the performance of the chemical transport model by matching CMAQ grid locations to the locations of environmental monitors and comparing predicted and observed values. We found that the model is biased high when predicting low levels of $PM_{2.5}$ concentrations. The model also over predicts $PM_{2.5}$ (mainly Organic Carbon and Elemental Carbon) during all seasons for fire events. Baker et al (2016) found the Flint Hills fire overestimated OC and EC but the Wallow fire did not overestimate to the same degree suggesting there is a lot of complexity in terms of how different fires are characterized in CMAQ. Another limitation is that only fire events that are part of the emission inventory have been evaluated. Any misspecification of emissions in the inventory fires is not included in our analysis. Further scientific advances and research efforts aimed to improve characterization of fire, fuel and emissioninventories, and to improve measure characterization of differential toxicity of smoke emitted from different fuel types can potentially improve future health risk assessments.

The health impacts quantified using concentration-response relationships described in this paper are also subject to uncertainties. More specifically, questions remain regarding the extent to which certain species of fire-attributable PM may be more or less toxic than others (Sullivan et al. 2008). Thus, using effect coefficients from the epidemiological studies that did not specifically consider wildland fire episodes, may under- or over-estimate impacts. By contrast, the quantitative meta-analysis draws upon epidemiological studies of wildland fire events, but is limited to respiratory hospital admissions and PM_{10}. Several of these studies were conducted outside of the U.S.; differences between the health care system, wildland fire particle composition, behavioral responses to smoke events and other factors may bias the meta-analysis pooled risk coefficient. We also apply the effect coefficients from the Delfino et al. (2009) study of wildfires in Southern California nationwide; this may bias our estimates of risk high or low depending on the area that it is applied to. Finally, the present value calculation assumes that the wildland fire-related premature deaths estimated for each year are independent of those estimated in all other years. For example, individuals modeled as dying prematurely from wildland fire smoke exposure in year 1 cannot also die in year 2, but the net present value calculation introduces the possibility that deaths may have been counted more than once across years. However, given that the fraction of total deaths accounted for by air pollution episodes is relatively small (approximately 6% on a national basis), the potential for this approach to bias-high the number of wildland-attributable deaths is likely small (Fann et al. 2011).

Despite these uncertainties, this manuscript also exhibits a number of strengths and unique features. First, this analysis is, to our knowledge, the first to characterize the incidence and

economic value of human health impacts attributable to wildland fire-emitted fine particles in the continental U.S. over a 5-year period. Second, while previous studies have incorporated a systematic review of wildland fire studies, none to our knowledge have performed a quantitative meta-analysis. Third, we characterize both the size, and distribution, of wildland fire-related premature deaths and hospital admissions across population subgroups. Taken together, the results in this analysis suggest that the number and value of wildland fire events is considerable and that these impacts are not shared equally across the U.S. population.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements:

We gratefully acknowledge the comments received from Bryan Hubbell and Karen Wesson, who reviewed early versions of this manuscript.

Abbreviations

BenMAP-CE	Environmental Benefits Mapping and Analysis Program—Community Edition
CMAQ	Community Multi-Scale Air Quality Model
U.S. EPA	United States Environmental Protection Agency
ICD	International Classification of Disease
NH_3	Ammonia
NOx	Nitrogen Dioxide
O_3	Ground-level ozone
$PM_{2.5}$	Particulate matter, 2.5 microns or less in diameter
SO_2	Sulfur Dioxide
WHO	World Health Organization

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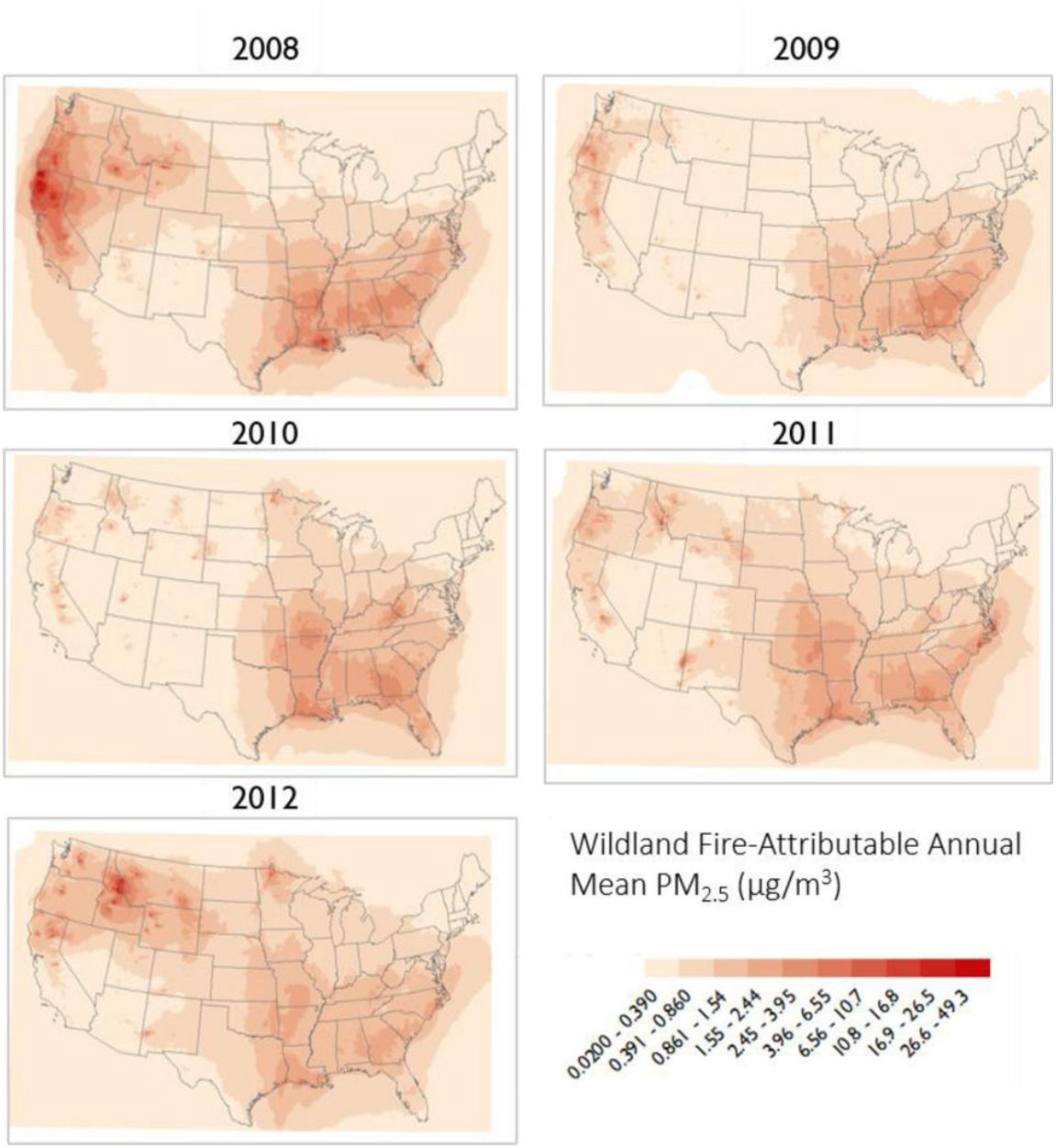

Figure 1.
Annual mean wildland fire-attributable $PM_{2.5}$ concentrations (2008– 2012)

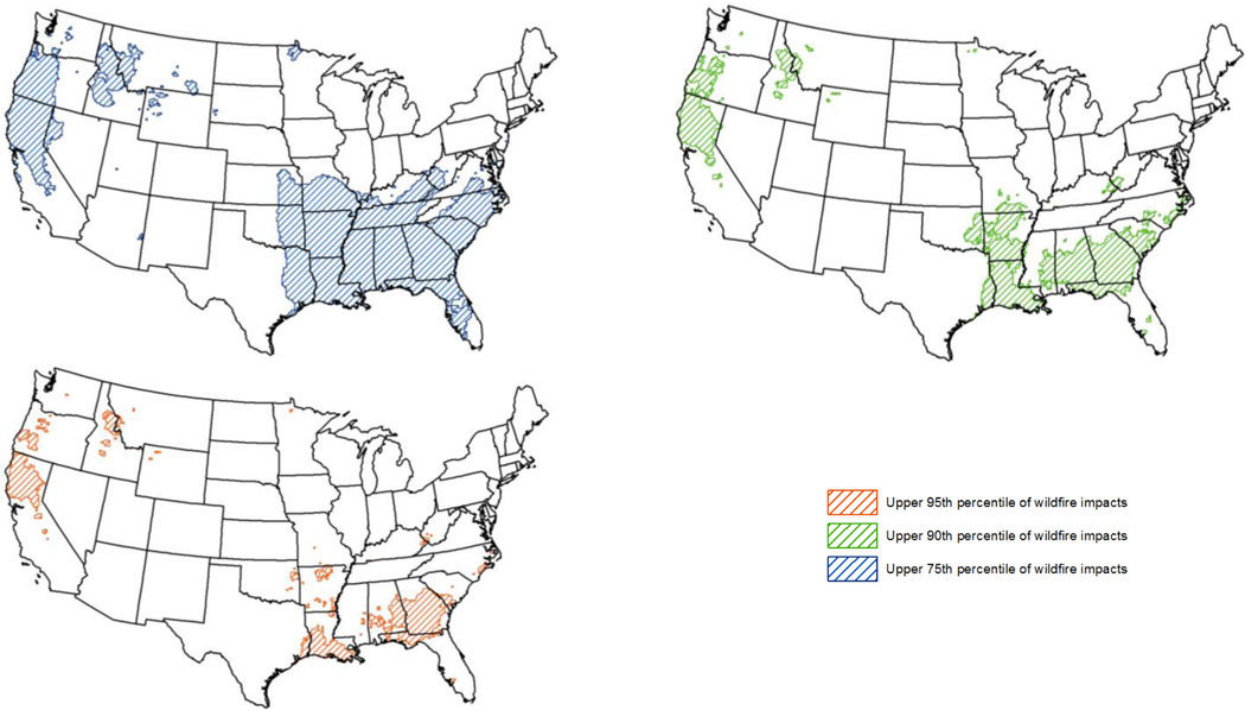

Figure 2.
Locations of the U.S. Experiencing Elevated Wildfire-Related $PM_{2.5}$ Concentrations Over a 5-year Period

Table 1.

Summary statistics of annual mean $PM_{2.5}$ ($\mu g/m^3$) predictions across 12km grid cells attributable to wildfires (2008 to 2012)

	Year[A]				
	2008	***2009***	***2010***	***2011***	***2012***
10^{th} %ile	0.26	0.14	0.14	0.26	0.33
25^{th} %ile	0.37	0.18	0.23	0.40	0.52
50^{th} %ile	0.61	0.33	0.43	0.60	0.81
Mean	1.1	0.56	0.70	0.80	0.92
75^{th} %ile	1.4	0.79	0.96	1.1	1.2
90^{th} %ile	2.3	1.3	1.7	1.7	1.6
Max	42	6.5	6.5	23	17
Population-weighted $PM_{2.5}$ level	1.1	0.61	0.68	0.73	0.75

[A] Values rounded to two significant figures

Table 2.

Premature deaths and illnesses attributable to wildfire-related $PM_{2.5}$ concentrations in each year calculated using alternative concentration-response functions (95% confidence intervals)

Endpoint	Year				
	2008	*2009*	*2010*	*2011*	*2012*
Respiratory Hospital Admissions					
Delfino et al. (2009)	8,500 (4,400—12,000)	5,200 (2,700—7,700)	6,200 (3,200—9,100)	6,300 (3,300—9,300)	6,400 (3,300—9,400)
Zanobetti et al. (2009)	6,300 (3,600—9,000)	3,900 (2,300—5,500)	4,600 (2,600—6,500)	4,700 (2,700—6,700)	4,800 (2,800—6,800)
Cardiovascular Hospital Admissions					
Delfino et al. (2009)	2,800 (−500—6,000)	1,700 (−320—3,700)	2,100 (−380—4,400)	2,100 (−380—4,500)	2,100 (−390—4,600)
Premature deaths from short-term exposure to $PM_{2.5}$					
Zanobetti & Schwartz (2009)	2,500 (1,900—3,000)	1,500 (1,100—1,800)	1,700 (1,300—2,100)	1,900 (1,400—2,200)	1,800 (1,400—2,200)
Premature deaths from long-term exposure to $PM_{2.5}$					
Krewski et al. (2009)	14,000 (9,700—19,000)	8,700 (5,800—11,000)	10,000 (6,900—14,000)	11,000 (7,300—14,000)	11,000 (7,600—15,000)
Lepeule et al. (2012)	32,000 (16,000—48,000)	19,000 (9,800—29,000)	23,000 (12,000—35,000)	24,000 (12,000—36,000)	25,000 (13,000—38,000)

[A] Values rounded to two significant figures; all functions estimated for populations ages 0–99

Table 3.

Estimated economic value of wildfire-attributable $PM_{2.5}$-related premature deaths and respiratory hospital admissions (2008 to 2012) (Billions of 2010$, 95% confidence intervals)[A].

Health Endpoints	Year					Present Value
	2008	*2009*	*2010*	*2011*	*2012*	
Sum of mortality from short-term exposures and respiratory hospital admissions [A]	$20 ($2—$53)	$12 ($1—$31)	$14 ($1—$37)	$11 ($1—$30)	$12 ($1—$31)	$63 ($6—$170)
Sum of mortality from long-term exposures and respiratory hospital admissions [B]	$130 ($12—$340)	$76 ($7—$210)	$90 ($8—$250)	$96 ($9—$260)	$100 ($9—$270)	$450 ($42—$1,000)

[A] Sum of Delfino et al. (2009) respiratory hospital admission estimates and Zanobetti & Schwartz (2009) mortality.

[B] Sum of Delfino et al. (2009) hospital admission estimates and Krewski (2009) mortality.

Table 4.

The percentage of individuals living in locations highly affected, and less affected, by wildland fires (2008 to 2012)[A]

Race	National Average	Location of the U.S.[B]		Difference between highly and less affected
		Highly affected	*Less affected*	
Asian	5%	4%	5%	–1%
Black	13%	18%	13%	12%
Native American	1%	2%	1%	1%
White	81%	75%	81%	–6%
Total	*100%*	*100%*	*100%*	

[A]Highly affect subgroups are those individuals who have experienced cumulative levels of wildland fire attributable $PM_{2.5}$ concentrations that are at or above the 75^{th} percentile, identified in Figure 2 with blue hatched shading. Less affected subgroups are those not living in these areas.

[B]Estimates rounded to two significant figures

Journal of the Air & Waste Management Association

ISSN: 1096-2247 (Print) 2162-2906 (Online) Journal homepage: https://www.tandfonline.com/loi/uawm20

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To cite this article: Vikram Ravi, Joseph K. Vaughan, Michael P. Wolcott & Brian K. Lamb (2019) Impacts of prescribed fires and benefits from their reduction for air quality, health, and visibility in the Pacific Northwest of the United States, Journal of the Air & Waste Management Association, 69:3, 289-304, DOI: 10.1080/10962247.2018.1526721

To link to this article: https://doi.org/10.1080/10962247.2018.1526721

Published online: 13 Dec 2018.

JOURNAL OF THE AIR & WASTE MANAGEMENT ASSOCIATION
2019, VOL. 69, NO. 3, 289–304
https://doi.org/10.1080/10962247.2018.1526721

Taylor & Francis
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TECHNICAL PAPER

Impacts of prescribed fires and benefits from their reduction for air quality, health, and visibility in the Pacific Northwest of the United States

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ABSTRACT

Using a WRF-SMOKE-CMAQ modeling framework, we investigate the impacts of smoke from prescribed fires on model performance, regional and loc al air quality, health impacts, and visibility in protected natural environments using three different prescribed fire emission scenarios: 100% fire, no fire, and 30% fire. The 30% fire case reflects a 70% reduction in fire activities due to harvesting of logging residues for use as a feedstock for a potential aviation biofuel supply chain. Overall model performance improves for several performance metrics when fire emissions are included, especially for organic carbon, irrespective of the model goals and criteria used. This effect on model performance is more pronounced for the rural and remote IMPROVE sites for organic carbon and total $PM_{2.5}$. A reduction in prescribed fire emissions (30% fire case) results in significant improvement in air quality in areas in western Oregon, northern Idaho, and western Montana, where most prescribed fires occur. Prescribed burning contributes to visibility impairment, and a relatively large portion of protected class I areas will benefit from a reduced emission scenario. For the haziest 20% days, prescribed burning is an important source of visibility impairment, and approximately 50% of IMPROVE sites in the model domain show a significant improvement in visibility for the reduced fire case. Using BenMAP, a health impact assessment tool, we show that several hundred additional deaths, several thousand upper and lower respiratory symptom cases, several hundred bronchitis cases, and more than 35,000 workday losses can be attributed to prescribed fires, and these health impacts decrease by 25–30% when a 30% fire emission scenario is considered.

Implications: This study assesses the potential regional and local air quality, public health, and visibility impacts from prescribed burning activities, as well as benefits that can be achieved by a potential reduction in emissions for a scenario where biomass is harvested for conversion to biofuel. As prescribed burning activities become more frequent, they can be more detrimental for air quality and health. Forest residue-based biofuel industry can be source of cleaner fuel with co-benefits of improved air quality, reduction in health impacts, and improved visibility.

PAPER HISTORY
Received February 11, 2018
Revised August 21, 2018
Accepted September 17, 2018

Introduction

Both wildfires and prescribed burning are significant sources of aerosols, as well as carbon monoxide (CO), oxides of nitrogen (NO_x), ammonia (NH_3), carbon dioxide (CO_2), and volatile organic compounds (VOCs) in the atmosphere (Wiedinmyer et al. 2006). Wildfires are uncontrolled and natural, whereas prescribed fires are widely used as a management tool for avoiding catastrophic wildfires by reducing the available fuel. The characteristics of emissions from wildfire and prescribed burns may be different since the factors governing the emissions, such as type of fuel (wildfires often consume canopy biomass), temperature (wildfires are much hotter, prescribed fires are cooler and lower intensity), and time of the year, differ between the two types (Kennard et al. 2005; Pyne, Andrews, and Laven 1996). Another important distinction is that prescribed fires recur periodically whereas wildfires are unpredictable. Even though these characteristics make prescribed fires different from wildfires, the contribution of prescribed fires to the emissions of particulate matter of diameter less than 2.5 μm ($PM_{2.5}$) can be a significant fraction of total $PM_{2.5}$ emissions in the emission inventory. For example, in 1989 in Georgia, the 211,000 tons of $PM_{2.5}$ emitted from prescribed fires were 30% of the state's total $PM_{2.5}$ emissions (Sandberg et al. 2002); in Washington and Oregon, annual $PM_{2.5}$ emissions of

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182,000 and 91,600 tons, respectively, were 21% and 46% of total non-wildfire $PM_{2.5}$ emissions for 2011 (USEPA 2011). These emissions can be even more significant on a seasonal basis, since emissions from prescribed fires are confined to only a few months in the fall and spring.

Emissions from prescribed fires can have a significant impact on air quality, visibility, and health (Sandberg et al. 2002). These impacts on air quality can be described at three different scales: (1) occupational exposure in the immediate vicinity, where the personnel involved in conducting the prescribed fires may be exposed to very high $PM_{2.5}$ concentrations (Naeher et al. 2006), (2) exposure to smoke of the communities that are at short downwind distances from sources (Naeher et al. 2006), and (3) exposure caused by long-range transport of the smoke plumes, which can be associated with severe air pollution episodes in large metropolitan areas (Hu et al. 2008; Tian et al. 2009; Zeng et al. 2008). Exposure to air pollution is associated with an increase in premature mortality and several diseases such as lung cancer, asthma attack, myocardial infarction, shortness of breath, and so on (Dockery et al. 1993; Lelieveld et al. 2015; Schwartz, Dockery, and Neas 1996). An increase in wildfire intensity in the future could also drive an increase in the demand for prescribed burning; hence it is necessary to study adverse health impacts from prescribed fires as studied recently by (Haikerwal et al. 2015).

In addition to air quality and health impacts, high $PM_{2.5}$ concentrations can also degrade visibility in class I areas (national parks, monuments, and wilderness areas), many of which are in the Pacific Northwest. The role of $PM_{2.5}$ and its constituent species in light scattering and absorption and thereby reducing visibility has been highlighted in several studies (NAPAP 1991). Visibility is considered an important part of public welfare since it plays an important role in public recreational activities and is addressed through the secondary PM National Ambient Air Quality Standard (NAAQS). Under the regional haze rule (USEPA 1999), visibility conditions in class I areas should be restored to natural conditions by the year 2064. This means that while the visibility during the cleanest 20% days should not deteriorate, visibility should also improve during the most impaired 20% days. The regional haze rule defines natural conditions as the visibility that would be observed in the absence of any human impairment. In the western United States, the average visibility in many class I areas varies between 14 and 10 deciviews, which corresponds to a visual range of 100–150 km (USEPA 1999). This visual range is one-half to two-thirds of the natural visibility condition (i.e., visibility without any human-caused impairment). EPA's interim air quality policy for wildland and prescribed fires maintains that "Air quality and visibility impacts from fires managed for resource benefits should be treated equitably with other source impacts" (USEPA 1998). In order to work toward the goal of attaining natural visibility conditions, the EPA requires states to prepare regional haze state implementation plans. Since prescribed fires are an important source of $PM_{2.5}$, though contributing to emissions only for a part of the year, they can cause significant visibility impairment and their impact can be reflected in both the 20% worst and 20% best visibility days.

This paper is motivated by the Northwest Advanced Renewables Alliance (NARA) project (www.nararenewables.org), which aims to create a sustainable biofuel supply chain in the Pacific Northwest region of the United States using forest residue (which is otherwise burned) and to meet requirements imposed by the Energy Independence and Security Act of 2007. Such an industry will replace or reduce fossil fuel usage and thus will reduce climate impacts through lower greenhouse gas emissions. Additionally, it will also have beneficial air quality impacts due to avoided prescribed burns through harvesting. While fires are an integral part of the natural ecosystem and various forests in the Pacific Northwest have adapted to them, and prescribed fires are often used as a fuel management tool (Wimberly and Liu 2014), our analysis here assumes the scenario where biomass harvesting for biofuel production may be used besides prescribed burning, as represented by our scenario selection in a later section. To investigate the implications of a NARA-like supply chain for biomass-to-biofuel conversion, we use a high-resolution advanced air quality modeling system to assess prescribed burn effects on air quality for three different emission scenarios. We assessed the air quality and health effects associated with two specific supply chain regions in the Pacific Northwest that included emissions from hauling activities, biorefinery, and pile burning, and found that most benefits are due to reduction in burning (Ravi et al. 2018). In this study, we expand our study domain to the entire Pacific Northwest, but only assess impacts from a reduction in prescribed burning. We also assess the effects on visibility in the Class I areas in the Pacific Northwest. Specifically, we address three different questions:

(1) How does inclusion of prescribed burn emissions affect the model performance at various monitoring sites? We use observations from the IMPROVE network for remote/rural locations and the AQS network for urban areas. We quantify this using standard air quality model performance evaluation metrics (see Table 1).

Table 1. Metrics used for performance evaluation (Boylan and Russell 2006; Chen et al. 2008).

Metric	Equation
Mean fractional bias (%)	$FB = \frac{1}{N}\sum_{i=1}^{N}\frac{(C_m - C_o)}{(C_m + C_o)/2} * 100$
Mean fractional error (%)	$FE = \frac{1}{N}\sum_{i=1}^{N}\frac{\lvert C_m - C_o\rvert}{(C_m + C_o)/2} * 100$
Normalized mean bias (%)	$NB = \frac{\sum_{i=1}^{N}(C_m - C_o)}{\sum_{i=1}^{N} C_o} * 100$
Normalized mean error (%)	$NE = \frac{\sum_{i=1}^{N}\lvert C_m - C_o\rvert}{\sum_{i=1}^{N} C_o} * 100$
Mean bias	$MB = \frac{1}{N}\sum_{i=1}^{N}(C_m - C_o)$
Mean error	$ME = \frac{1}{N}\sum_{i=1}^{N}\lvert C_m - C_o\rvert$
Root mean square error (RMSE)	$RMSE = \left(\frac{1}{N}\sum_{i=1}^{N}(C_m - C_o)\right)^{1/2}$
Correlation coefficient (r)	$r = \frac{\sum_{i=1}^{N}(C_m - \overline{C_m})(C_o - \overline{C_o})}{\left[\sum_{i=1}^{N}(C_m - \overline{C_m})^2 \sum_{i=1}^{N}(C_o - \overline{C_o})^2\right]^{1/2}}$
Mean fractional bias goal (%)	$\lvert MFB\rvert \le 170\, e^{\left[\frac{-0.5(\overline{C_o} + \overline{C_m})}{0.5}\right]} + 30$
Mean fractional bias criteria (%)	$\lvert MFB\rvert \le 140\, e^{\left[\frac{-0.5(\overline{C_o} + \overline{C_m})}{0.5}\right]} + 60$
Mean fractional error goal (%)	$MFE \le 150\, e^{\left[\frac{-0.5(\overline{C_o} + \overline{C_m})}{0.75}\right]} + 50$
Mean fractional error criteria (%)	$MFE \le 125\, e^{\left[\frac{-0.5(\overline{C_o} + \overline{C_m})}{0.75}\right]} + 75$

(2) How would a scenario for reduced prescribed fire emissions improve the regional air quality and what health benefits should be expected with such a scenario?
(3) To what extent do prescribed fires affect visibility in the national parks and wilderness areas during the simulation period.

Methodology

Modeling framework

For this study, we used the AIRPACT-4 (Air Information Report for Public Access and Community Tracking version 4) air quality modeling framework for the Pacific Northwest (Chen et al. 2008; Vaughan et al. 2004). The modeling domain encompasses all of Idaho, Oregon, and Washington with peripheral areas and uses a 258 × 285 grid of 4 km × 4 km horizontal grid cells with 21 vertical layers of varying thickness. Retrospective runs of AIRPACT-4 for this study used archived meteorological simulations from forecast meteorology from the Weather Research and Forecasting modeling system (WRF; Skamarock et al. 2005) operated by the University of Washington (http://www.atmos.washington.edu/mm5rt; Mass et al. 2003). WRF output was processed through the Meteorological Chemical Interface Processor (MCIP; Byun et al. 1999; Otte and Pleim 2010). Emissions for area, mobile, and point sources based on the National Emission Inventory (NEI) 2007 were compiled using the Sparse Matrix Operator Kernel for Emissions (SMOKE) tool (https://www.cmascenter.org/smoke/). Vehicular emissions were processed using MOVES-2010. Biogenic emissions were estimated using the Model for Emission of Gases and Aerosols model (Guenther et al. 2012). Model boundary conditions are derived from MOZART-4 global chemistry model results (Emmons et al. 2010). For the gas phase chemistry, the SAPRC99 chemical mechanism (Carter 2000) was used, with the AE5 aerosol module. AIRPACT-4 uses the Community Multiscale Air Quality (CMAQ) model version 4.7.1 for chemical transport and transformation (D. Byun and Schere 2006).

For the current work, estimated emissions from prescribed fires were extracted from the NEI 2011 fire dataset for the states within the AIRPACT domain. EPA estimates the fire emissions through the use of the BlueSky fire modeling framework (Larkin et al. 2009). The BlueSky framework uses fire information from a variety of sources (SMARTFIRE satellite reporting [Raffuse et al. 2009], groundbased Incident Command System [ICS-209] reports, and prescribed-burn reporting systems). Once the fire information is available, fuel load maps and a fuel consumption model are used to estimate the total fuel consumed. Given total fuel consumption, emissions of different pollutants including $PM_{2.5}$, CO, CH_4, NO_x, SO_2, NH_3, and VOCs are generated using an emission module. Emissions are distributed spatially and temporally using SMOKE, which also calculates the plume rise for the fires (Herron-Thorpe et al. 2014).

Simulation period and modeling scenarios

The monthly total emissions from the prescribed fires in 2011 are shown in Figure 1, which shows that the emissions from prescribed burns peak during the fall months and are maximum during October and November. Based on this pattern of fall burning, we selected October and November 2011 as our simulation period. To assess the potential impact of prescribed burns on air quality, we consider three different emission scenarios:

100% Fire (or with fire) case: Includes all the prescribed burn emissions in the domain as per NEI 2011

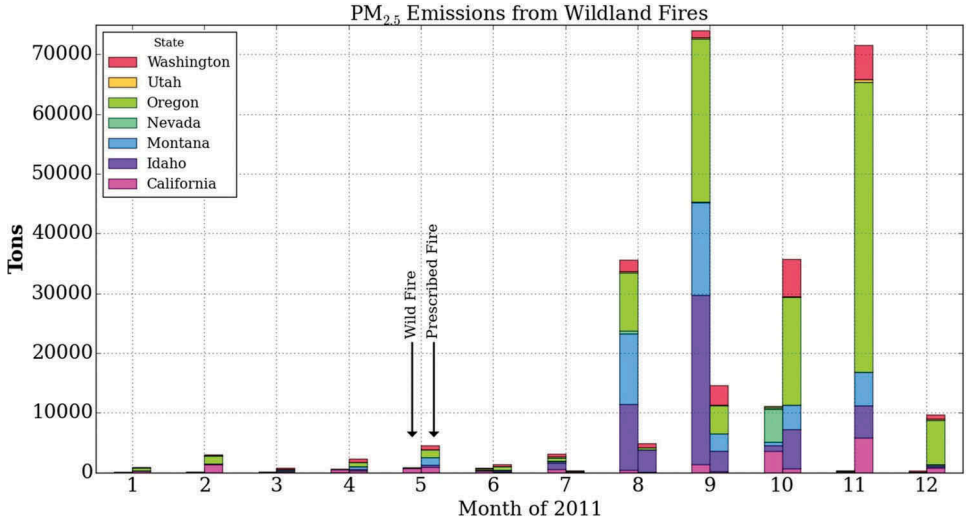

Figure 1. Emissions of $PM_{2.5}$ from wildfires and prescribed burns for 2011 based on NEI-2011. For each month, the stacked bar on the left is for wildfire and the bar on the right is for prescribed fire.

along with all other emissions for point, area, and mobile sources as used in daily AIRPACT forecasting.

30% Fire case: Includes all the prescribed burn sources as per NEI 2011, but all prescribed burn emissions and heat flux) are uniformly reduced by 70%, with other emissions the same as for the 100% fire case. This case reflects the harvesting of residue biomass for use as a potential feedstock for the aviation biofuel supply chain. This assumption is based on Perez-Garcia et al. (2012) and Pierobon, Eastin, and Ganguly (2018), which assume that of the total woody residue left in the forests, 65% is collected in the form of slash piles and 35% is left scattered in the forest. We assume that the biofuel production will reduce the need for burning, forming the basis of this case where we assume that 30% biomass is burned. While these numbers are for Washington, we uniformly apply these to the study domain. For calculating the emissions, a key assumption here is that a reduction in biomass will linearly reduce emissions and other related quantities. This is reasonable, considering that we are reducing the burn area.

No fire case: No prescribed burn emissions, and other emissions are kept same as the first case described. This case is the baseline against which the other two cases are compared.

Model evaluation methods

To address our first question on model performance, we evaluated the model for the with fire and no fire cases using conventional model performance measures as listed in Table 1. Model performance for the two scenarios is compared using mean fractional bias (MFB) and mean fractional error (MFE) and their comparison with the performance goals and performance criteria for $PM_{2.5}$. These performance goals (the best expected performance of a model) and criteria (acceptable level of model performance) were proposed by Boylan and Russell (2006) and were based on analysis of several modeling studies performed throughout the United States. We also compare the model performance using the normalized mean bias and error (NMB/E) goals and criteria established by (2017). Emery et al. We used two different observation datasets for the purpose of model evaluation: the Interagency Monitoring of Protected Visual Environments (IMPROVE) network (Malm et al. 1994) and USEPA's AQS dataset. AQS provides hourly $PM_{2.5}$ concentrations for urban areas across the United States. IMPROVE sites are usually located in or near class I areas and measure the concentration of $PM_{2.5}$ and other $PM_{2.5}$ species, such as organic carbon (OC), elemental carbon (EC), sulfate (SO_4), nitrate (NO_3), and ammonium (NH_4). IMPROVE sites report 24-hr average concentrations with a frequency of once every 3 days. For visibility investigation we use the deciview (dv) metric (defined in the following), a preferred metric in the regional haze rule because a unit change in deciviews is unbiased by the prevailing visibility being highly impaired or clean (Pitchford and Malm 1994).

For IMPROVE sites, the concentrations of various $PM_{2.5}$ species are used with their corresponding coefficients of extinction to get the total extinction coefficient (called the reconstructed extinction coefficient) using the following equation (Pitchford et al. 2007):

$$\begin{aligned}\beta_{ext} = {} & 2.2f_S(RH)[AMSUL_s] + 4.8f_L(RH)[AMSUL_L] \\ & + 2.4f_S(RH)[AMNIT_s] + 5.1f_L(RH)[AMNIT_L] \\ & + 2.8f_S(RH)_{OM}[OM_s] + 6.1f_L(RH)_{OM}[OM_L] \\ & + 1.7f(RH)_{SS}[PM_{2.5}SeaSalt] + 1.0[PM_{2.5}Soil] \\ & + 0.6[CoarseMass] + 10[EC] + 0.33[NO_2(ppb)] \\ & + \beta_{\mathrm{Ray}}\end{aligned} \tag{1}$$

where [*AMSUL*], [*AMNIT*], [*OM*], [*EC*], [*Soil*], [*Sea Salt*], [*Coarse Mass*], and [NO_2] are the concentrations (in μg/m^3) of ammonium sulfate, ammonium nitrate, organic mass, elemental carbon, soil, sea salt, coarse PM, and NO_2, respectively. The *f*(*RH*) is a dimensionless relative humidity adjustment factor, needed to account for effect of water uptake by aerosols on the dry extinction coefficient. Several components in the preceding equation are divided in small and large modes, each with a separate relative humidity (RH) adjustment factor. The Rayleigh extinction coefficient, β_{Ray}, accounts for scattering by air molecules. The revised IMPROVE equation assumes that organic matter is not hygroscopic (f_S (RH) = f_L(RH) = 1), and the OM/OC ratio is 1.8. Lowenthal and Kumar (2016) recently evaluated the revised IMPROVE equation based on data collected from field studies, and recommended that OM should be considered hygroscopic and the OM/OC ratio of 2.1 should be used. Based on their recommendations, we use the OM/OC ratio of 2.1 and assume OM to be hygroscopic. The relative humidity adjustment factors for our analysis were taken from Lowenthal and Kumar (2016). The calculated β_{ext} is converted to deciviews using eq 2:

$$dv = 10 * \ln(\beta_{ext}/10) \tag{2}$$

Estimating the health benefits

To obtain the health impact estimates from different scenarios, we utilize the Benefits Mapping and Analysis Program Community Edition version 1.1 (BenMAP CE; https://www.epa.gov/benmap, developed by the USEPA), which contains various $PM_{2.5}$ mortality and morbidity concentration–response (C-R) functions, population data sets, incidence rates, and population growth functions. BenMAP can thus be used to estimate the health effects of different scenarios for different health endpoints. The generic form of a health impacts function, which relates the changes in the incidence of a health endpoint to the change in a pollutants concentration, can be written as (Fann et al. 2012)

$$\Delta y = y_o[\exp(\beta * \Delta x) - 1] * population \tag{3}$$

where y_o is the baseline incidence rate, β is the mortality or morbidity effect estimate (i.e., an estimate of the percent change in mortality or morbidity caused by a unit change in ambient concentration of the pollutant), Δx is change in pollutant concentration, and *population* is the population affected by the changed concentration (Fann et al. 2012). Baseline incidence rates for this analysis use the incidence data set contained within BenMAP. For the current analysis, Δx is taken as the concentration difference between the 100% fire and no fire cases, and the concentration difference between the 100% fire and 30% fire cases. The Δy from these two scenarios will give the health effects of prescribed fires and benefits for different health endpoints considered for this study when emissions are reduced. We consider a wide spectrum of health endpoints for which C-R functions are available in BenMAP. We also derived the effect estimate for fire specific studies for two health endpoints: all cardiovascular hospital admissions, and all respiratory hospital admissions based on Delfino et al. (2009). While there are several fire specific health studies that can be used to derive the effect estimate for other health endpoints, we avoid doing so because most of these studies were conducted outside of the United States, caveats of which are also explained in Fann et al. (2018).

BenMAP requires a full year of data for pollutant concentrations at each of the grid cells, and our current simulations covered only two months. To overcome this, we used the model simulations from a different year (May–September 2009, December 2009–April 2010; no fire emissions were included in these simulations) and concatenated it with the current simulations (October–November 2011). While this is not the best approach, we consider this to be acceptable considering that we are investigating the health impacts only from prescribed fires, for which emissions (and hence population exposure) maximize during the October–November time-period.

Results and discussion

Model performance evaluation and impact of prescribed fire on model performance

We show the impact of prescribed fires on model performance by calculating various standard metrics (Table 1) for both the 100% fire case and the no fire case. Comparison of various statistical metrics for 100% fire and no fire cases is shown for both IMPROVE and AQS sites in Table 2. The mean concentration at urban sites is much larger (8.4 μg/m^3) compared to the nonurban

Table 2. Performance metrics for PM2.5 at the AQS and IMPROVE sites.

	AQS sites		IMPROVE sites	
Metric	No fire	Fire	No fire	Fire
Number of observed–modeled pairs	6561		537	
Mean observed (μg/m^3)	8.41		2.83	
Mean modeled (μg/m^3)	7.54	9.82	2.06	3.88
MB (μg/m^3)	−0.87	1.41	−0.74	1.11
ME (μg/m^3)	5.17	5.94	1.55	2.12
MFB (%)	−19	1	−21	19
MFE (%)	61	59	57	54
NMB (%)	−10	17	−26	39
NME (%)	62	71	55	75
RMSE	7.71	9.42	2.81	4.16

IMPROVE sites (2.8 μg/m^3). For the case when fire emissions are not included, the model underpredicts mean concentrations at both IMPROVE and AQS sites, whereas including fire emissions results in overprediction of mean concentration at both networks. While the fractional bias indicates improvement in performance when fire emissions are considered at the two networks, there is almost no change for the fractional error, indicating that large variability between modeled and observed concentrations still exists. Figure 2 shows how the model performance changes as a function of the observed concentration. The coefficient of correlation improves for both the networks for the 100% fire case, but the improvement is much more significant for IMPROVE sites. We also notice that, in general, the model overpredicts at lower observed concentration and underpredicts at higher observed concentration. Though including fire emissions improves the modeled concentration at higher observed concentrations, the underprediction is more severe at higher observed values, and more so at AQS sites; this could be attributed in part to the urban nature of sources, where the emissions from various sources such as vehicular emissions may not be captured completely. The bugle plots in Figure 3 show how the two different networks perform with respect to the performance goals and criteria. Most of the IMPROVE sites are within the performance criteria, with a few more sites within criteria with fire emissions included compared to the no fire case. While more sites are within criteria for the 100% fire case, including fire emissions results in overprediction at few sites where the average concentration is large. The new set of recommendations for model performance goals and criteria by Emery et al. (2017) uses NMB, NME, and the correlation coefficient (*r*). While recommended benchmarks for NMB and NME are specified for total $PM_{2.5}$ and for sulfate, ammonium, nitrate, organic carbon, and elemental carbon in the Emery et al. study, they only specify goals and criteria for *r* for total $PM_{2.5}$, sulfate, and ammonium. Our analysis does not use any concentration thresholds for total $PM_{2.5}$ or SO_4, NH_4, NO_3, OC, or EC. We find that NMB for $PM_{2.5}$ at AQS sites is −10% for the no fire simulations, which is within the NMB goal of ±10%, but increases to 17% for the fire simulations, which is still within the recommended criteria. With the $PM_{2.5}$ NME at AQS sites of 62% for the no fire case and 71% for the 100% fire case, the model doesn't meet either the NME goal or the criteria for any of the simulation scenarios. At the IMPROVE sites, $PM_{2.5}$ NMB is −26%, which is within the criteria of ±30% for the no fire case, but 39% for the 100% fire case.

The speciated data in Table 3 show that organic carbon (OC) is the most dominant species, with a mean observed concentration of 1.21 μg/m^3, and the performance is significantly improved with the bias changing from −0.74 μg/m^3 for no fire to 0.06 μg/m^3 for the 100% fire case and the corresponding fractional bias changing from −105% to only −17%. Compared to the no fire scenario when almost

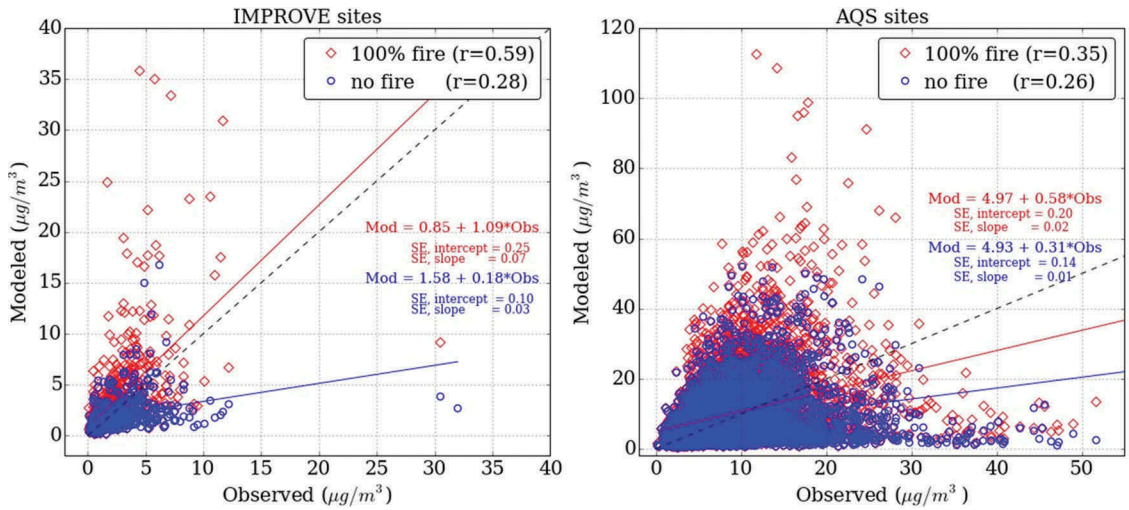

Figure 2. Modeled to observed ratio versus observed $PM_{2.5}$ concentrations for AQS and IMPROVE sites.

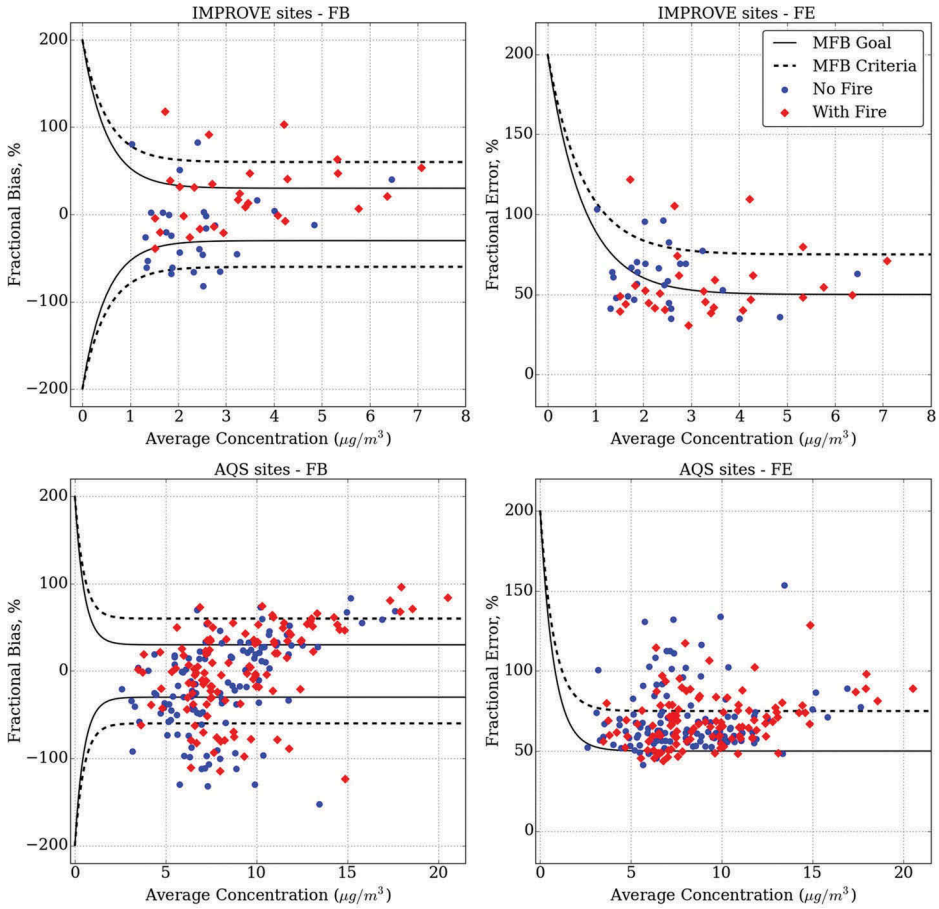

Figure 3. Bugle plots for comparison of MFB and MFE with goals and criteria for $PM_{2.5}$ during the period of simulation.

50% sites are outside MFB performance criteria for OC, including prescribed fire emissions results in MFB within performance criteria for all the sites. For EC, SO_4, and NO_3, species for both the 100% fire and no fire cases. However, the model performance improves significantly for OC, for which NMB is 5% for the 100% fire simulations, compared

Table 3. Performance metrics at the IMPROVE sites.

			With fire						No fire					
Species	Number observed	Mean observed (μg/m^3)	Bias (μg/m^3)	MFB (%)	MFE (%)	NMB (%)	NME (%)	RMSE (μg/m^3)	Bias (μg/m^3)	MFB (%)	MFE (%)	NMB (%)	NME (%)	RMSE (μg/m^3)
OC	490	1.21	0.06	−17	73	5	75	4.68	−0.74	−105	114	−26	55	4.46
EC	488	0.17	0.18	46	75	110	160	0.76	−0.98	−33	73	−43	55	0.49
SO_4^{2-}	502	0.33	0.20	55	66	61	78	0.33	−0.07	42	59	36	62	0.26
NH_4^+	502	0.16	0.02	−4	64	10	71	0.19	0.12	−35	69	−29	58	0.13
NO_3^-	502	0.14	0.19	48	100	141	183	0.49	−0.05	22	94	64	122	0.31

observed concentrations are much smaller compared to OC, and the model underpredicts in the no fire case and overpredicts in the 100% fire case. The concentrations for other species are small and remain within criteria in both the cases, since the MFB (MFE) approaches ±200% (200%) at very low concentrations. Table 3 also has NMB and NME for sulfate, nitrate, and elemental carbon, and we find that the model performance is relatively poor for these to −81% for the no fire simulations, though the NME for both simulation scenarios is much larger compared to NME goals/criteria. Model ability to predict ammonium also improves for the 100% fire simulations, where the NMB is 10%, compared to −29% for the no fire simulations, which barely meets the criteria. Improved performance for OC and NH_4 is important, because these combined together account for 48% of the observed

$PM_{2.5}$ mass concentration on average at the IMPROVE sites. The correlation coefficient, *r*, is shown in Figure 2 and is within the criteria for IMPROVE sites for total $PM_{2.5}$, but smaller than the recommended criteria at the AQS sites; nevertheless, there is an improvement is model performance during the 100% fire simulations compared to the no fire simulations.

The scatter diagram in Figure 4 shows the deciview comparison at various IMPROVE sites for all the days in October–November 2011, as well as for the 20% highest deciview days. The model does better when fire emissions are included, with a correlation coefficient (*r*) of 0.51. This value is comparable to evaluations from Mebust et al. (2003), who reported $r = 0.49$, although they used a different observational data set. The performance is relatively poorer for the 20% days with worst visibility when compared to all the days in the simulation period, with the coefficient of correlation equal to 0.27. The mean observed deciview and mean bias for all days were 8.22 dv and 2.72 dv, respectively. For the 20% worst and 20% best visibility days, the mean observed deciview (and mean bias) were 12.70 dv (−4.95 dv) and 2.80 dv (0.08 dv), respectively. These differences can in part be attributed to the model's ability to correctly reproduce the observed concentrations, as well as to the artifacts associated with the measurement of various $PM_{2.5}$ components (Mebust et al. 2003).

Overall, these results are comparable to previous studies using a 12-km version of AIRPACT conducted in the region during wildfire periods. Herron-Thorpe et al. (2014) reported that fractional bias for $PM_{2.5}$ at the AQS sites was ~−30% (FE of ~60%) for the simulations conducted for wildfire periods in 2007–08. While Herron-Thorpe et al. (2014) reported a mean bias of −0.72 μg/m^3 for AQS sites, Chen et al. (2008) reported an overprediction of 2.1 and 2.2 μg/m^3 at the AQS and IMPROVE sites, respectively. In general, we see that while some statistical metrics improve when fire emissions are considered, for some sites, fires cause large overprediction in the concentrations. This can be influenced by several factors, including the meteorological model failing to predict wind speed and direction correctly, application of a common temporal profile for all fires emissions, poorly simulated plume rise, and the complex topography in areas of prescribed fire. Additionally, it has been shown that the spatiotemporal allocation of the fires can also have a significant influence on the concentrations (Garcia-Menendez, Yongtao, and Odman 2014).

Regional and local impacts of prescribed fires

Figure 5a shows the concentration difference between the 100% fire and no fire cases for $PM_{2.5}$. Regional impacts of prescribed fires are most significant in the western part of the domain, especially in Oregon, where the average contribution from prescribed fires over the entire simulation period is ≥5 μg/m^3 $PM_{2.5}$. This is somewhat expected, since maximum emissions from prescribed fires occur in Oregon (Figure 1). The difference is even larger for some areas, and peak concentration differences are greater than 20–25 μg/m^3 in areas west of the Cascade mountain range. Emissions from prescribed fires contribute to $PM_{2.5}$ loading in excess of 5 μg/m^3 for parts of northern Idaho as well as western Montana. A comparison of the simulation period average $PM_{2.5}$ concentrations between the 100% fire and no fire cases showed that total $PM_{2.5}$ loading in northern Idaho during this period is almost entirely attributable to prescribed fire emissions. Figure 5b shows the modeled relative

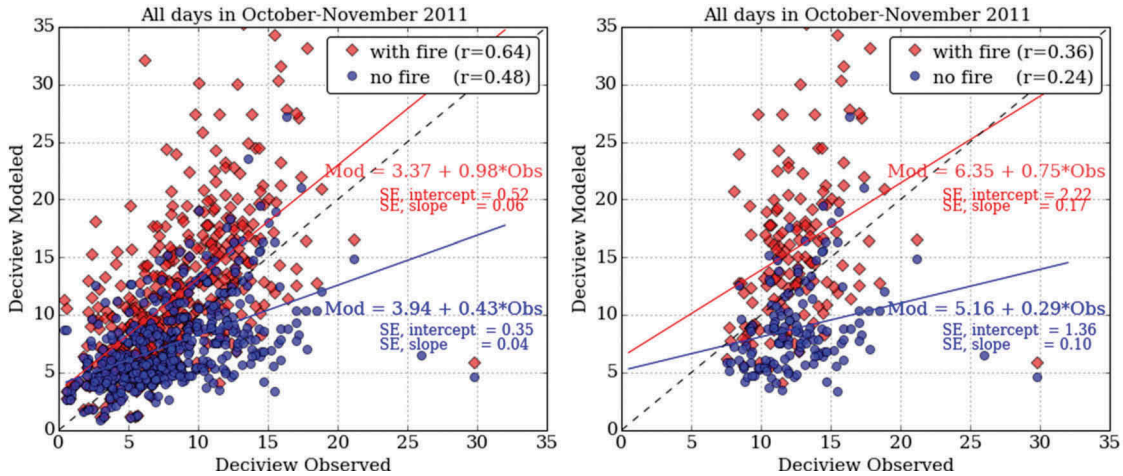

Figure 4. Modeled and observed deciview comparison for all days and 20% worst visibility days within the simulation period.

change between 100% fire and 30% fire cases, which shows the potential benefits of avoiding biomass burning, and hence depicts the air quality benefits if the biomass is harvested for subsequent biofuel production. Only those model grid cells where the simulation period average for the 100% fire case was greater than 12 μg/m^3 were considered. Large air quality benefits occur through $PM_{2.5}$ concentrations decreasing by more than 50–75% (6 μg/m^3 or more) for most of the region in western Oregon. Large population centers around the interstate highway 5 (I-5) corridor also see up to 20% changes (1.8–2.4 μg/m^3). Figure 5b is sensitive to the threshold concentration chosen for the 100% fire case, and changing this threshold to 8 or 10 μg/m^3 results in more areas in Washington as well as northern Idaho showing improvements. The simulated changes are not directly proportional to the changes in emissions since reduced heat fluxes for the 30% biomass burn scenario will result in lower plume rises and thereby in reduced dispersion, which will cause larger near-source impacts for the same amount of emissions. While the simulated effects on $PM_{2.5}$ were significant during the period of simulation, a similar analysis didn't show any contributions of prescribed fires to O_3, perhaps due to reduced photochemistry during the months of October–November. The 8-hr average O_3 concentration differences were very small (<1 ppb) at all the AQS sites considered, and so not of interest for further analysis.

To consider the local impacts of prescribed fires, we identified several monitoring locations within the domain where air quality impacts occurred (Figure 6) and apply the relative response factors to observed $PM_{2.5}$ concentration to calculate the no fire case and 30% fire case concentrations. At several sites, such as Pinehurst (located in West Silver Valley, part of Idaho's Shoshone County $PM_{2.5}$ non-attainment area) and Oakridge (a non-attainment region in Oregon), where prescribed fires are significant sources of $PM_{2.5}$, the 30% fire case causes the peak concentration to decrease significantly. Maximum hourly concentration changed from 111 μg/m^3 to 60 μg/m^3 for Pinehurst, and from 90 μg/m^3 to 66 μg/m^3 for Oakridge. For sites located near large populated areas, such as Portland, Eugene, and Vancouver, the average concentration is 10–11 μg/m^3, and the maximum hourly concentration is 50 μg/m^3, 71 μg/m^3, and 278 μg/m^3 in the 100% fire case, respectively. At all these places, the hourly peak concentration decreases by 5–10 μg/m^3 in the 30% fire case. At several other sites not shown in the figure, such as Chester (CA), St. Maries (ID), and Kootenai Tribe (ID), where the mean concentration is otherwise very small, prescribed fire causes the mean and maximum concentrations to increase by an order of magnitude. Time-series plots at the sites indicate that while some communities are affected by elevated $PM_{2.5}$ concentrations only for a few specific days, others are affected frequently during the burn season. The time series also indicate that at urban sites, the impacts from prescribed fires are small and only for specific hours, indicating that the smoke management agencies are indeed successful in avoiding exposure to these large and urban population centers. In general, results at several of these sites that are located in small communities show that prescribed fires can impact air quality significantly and can be detrimental to the health of specific age groups or sensitive populations, and thus, a decrease in burning will represent significant air quality benefits for these small communities.

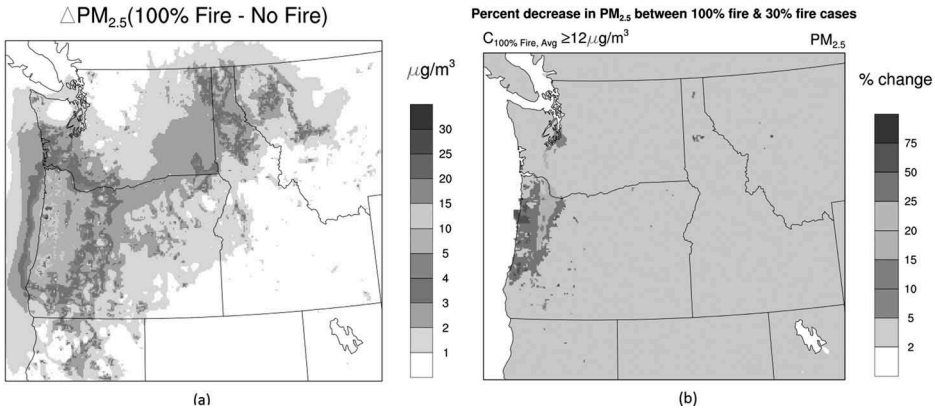

Figure 5. (a) $PM_{2.5}$ difference between the 100% fire case and the no fire case when averaged over all days in the simulation period. (b) Percent change in $PM_{2.5}$ concentrations when all the fire emissions are uniformly reduced by 70%. Results for only those model grid cells where $PM_{2.5}$ concentrations for 100% fire case is greater than 12 μg/m^3 are shown.

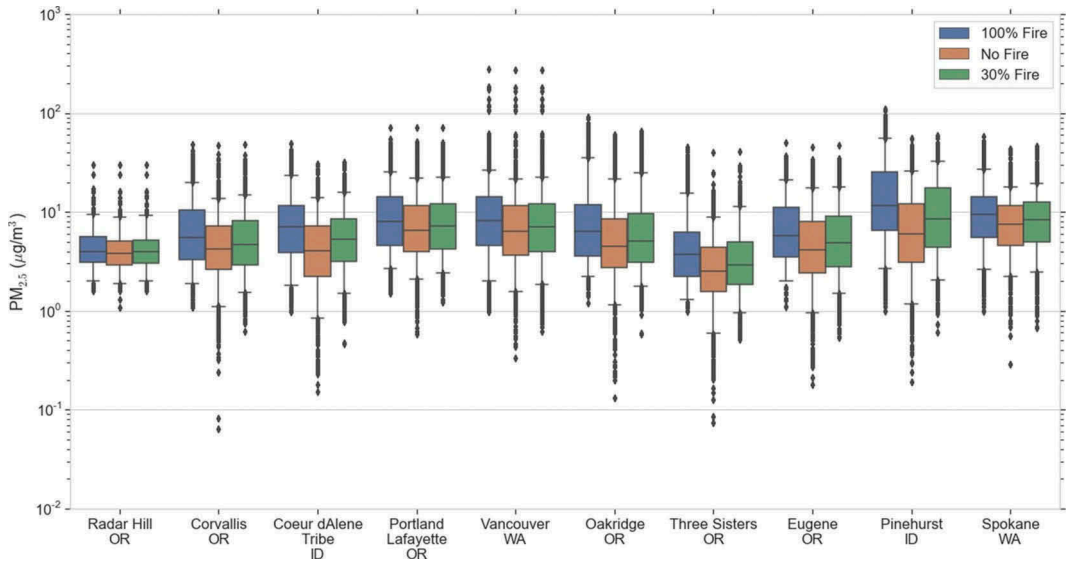

Figure 6. $PM_{2.5}$ distributions at selected sites for the three different scenarios. Whiskers represent 2nd and 98th percentiles.

Health effects of prescribed fires and benefits from avoided emissions

Using BenMAP, we calculated the health effects for two different control cases, the no fire and 30% fire cases, with respect to the base case (i.e., the 100% fire case). Table 4 shows the health impact estimates for these two cases. The mean number of additional mortalities caused by $PM_{2.5}$ from prescribed fire is ~280 for C-R functions from Krewski et al. (2009) and Pope et al. (2002), and ~710 deaths based on C-R functions from Laden et al. (2006), and these numbers decrease to ~200 and ~500, respectively, when prescribed fires are reduced by 70%. Additional mortality in both control cases is almost double for Laden et al. (2006) relative to Pope et al. (2002) or Krewski et al. (2009). This difference in the PM-caused mortality estimate is because of different effect estimates used in C-R functions. Estimates based on these studies are of long-term mortality. For quantifying short-term mortality due to smoke exposure from prescribed fires, we used the effect estimate from Zanobetti and Schwartz (2009). Based on Zanobetti and Schwartz (2009), an estimated 49 all-cause mortalities occur due to $PM_{2.5}$ from prescribed fires, and this number can be reduced to 34 for the scenario of reduced prescribed fires.

Impacts for a number of other health endpoints are also considered, such as acute bronchitis, acute myocardial infarction (nonfatal heart attack), asthma, chronic bronchitis (irritation or inflammation of lung airways), emergency-room visits, hospital admissions, lower respiratory symptoms (LRS; defined as two or more of cough, chest pain, phlegm, or wheeze), upper respiratory symptoms (URS; defined as one or more of the following symptoms: runny or stuffy nose, wet cough, and burning, aching, or red eyes), and two additional endpoints indicative of the overall loss in productivity: minor restricted activity days and lost workdays. Based on our results (Table 4), more than 100,000 asthma cases, 400 acute bronchitis cases, 100–200 chronic bronchitis cases, 65–70 emergency-room visits, and 20–40 hospital admissions can be attributed to additional $PM_{2.5}$ concentrations caused by prescribed fires. When effect estimates derived from smoke-specific studies are used in the C-R functions, an estimated 124 cases of hospital admissions due to respiratory issues and 47 of hospital admissions due to cardiovascular diseases can be attributed to prescribed fires. Prescribed fires are also expected to contribute to 7300 additional URS and 4400 LRS cases, which are reduced by 29% and 25% for 30% fire cases. Most significant is workday losses or days of restricted activity (35,000 and 200,000+, respectively). In a reduced prescribed burning scenario, we see an improvement for all health endpoints, but the improvement is not proportional to the reduction in emissions. While emissions are reduced by 70%, calculated decrease in health impacts for various endpoints is only 25–30%. We believe that this is partly due to our assumption where the larger burns that are more buoyant and dispersed to longer distances are uniformly reduced in size, and the resulting smaller burns are not dispersed as much because of lower plume buoyancy.

The state-wise distribution of two different health endpoints—all cause mortality and asthma exacerbation—is shown in Figure 7. The effects are maximum for Oregon and Washington, but a reduction in prescribed burn

Table 4. Impact estimates for various health endpoints for no fire and 30% fire case (control cases) with 100% fire case (base case).

Health endpoint	Author	Impact estimate $\Delta y = y_{100\%\ Fire} - y_{No\ Fire}$	Impact estimate $\Delta y = y_{100\%\ Fire} - y_{30\%\ Fire}$
Mortality (all cause)	Krewski et al. (2009)	277	196
	Laden et al. (2006)	707	501
	Pope et al. (2002)	277	196
	Zanobetti and Schwartz (2009)	49	34
Acute bronchitis	Dockery et al. (1996)	398	283
Acute myocardial infarction (nonfatal)	Sullivan et al. (2005)	19	14
	Zanobetti et al. (2009)	22	16
Asthma exacerbation (cough)	Mar et al. (2004)	79,878	59,311
Asthma exacerbation (shortness of breath)	Mar et al. (2004)	26,972	20,371
Chronic bronchitis	Abbey et al. (1995)	190	135
Emergency-room visits (asthma)	Mar, Koenig, and Primomo (2010)	125	90
	Slaughter et al. (2005)	67	48
Hospital admissions all cardiovascular (less myocardial infarctions)	Bell et al. (2008)	24	17
	Peng et al. (2009)	21	15
	Delfino et al. (2009)	47	33
Hospital admissions all respiratory	Zanobetti et al. (2009)	49	35
	Delfino et al. (2009)	124	87
Lower respiratory symptoms	Schwartz and Neas (2000)	4,386	3,294
Upper respiratory symptoms	Pope et al. (1991)	7,286	5,186
Minor restricted activity days	Ostro and Rothschild (1989)	207,588	150,042
Work loss days	Ostro (1987)	35,662	25,568

results in fewer deaths or a reduction of diseases across the domain. While the air quality benefits from fire reduction are most prominent for Lincoln and Benton counties in Oregon, BenMAP shows maximum health benefits in the counties of Multnomah, Lane, Clackamas, and Washington counties in Oregon and King and Clark counties in Washington. These larger benefits are due to larger populations in these counties.

Impacts on visibility in class I areas

To assess the visibility benefits for class I areas that can be attributed to the reduction in prescribed fire emissions, we extracted the deciview data for the grid cells having at least 50% of the grid cell area within a national park and/or wilderness area. CMAQ outputs hourly deciview, but the IMPROVE network uses 24-hr average concentration, along with the relative humidity factor, and reports one deciview metric per day. To get a daily deciview metric from CMAQ, we used the daily average concentrations for ammonium sulfate, ammonium nitrate, elemental carbon, organic aerosols, and fine soil concentrations, with the relative humidity factor calculated using the daily average relative humidity (RH).

The distribution of the daily average deciview for the three different modeling scenarios is shown in Figure 8. We can derive two important results from this: (1) Fires impact the deciview distribution at both low deciviews (i.e., best visibility) and high deciviews (i.e., worst visibility); and (2) as we move to higher deciviews, many more grid cells are influenced by fires and the highest deciviews solely occur because of fires. This is indicative of the poor visibility in partial areas of national parks/wilderness areas, which may not be captured by monitoring at specific locations through the IMPROVE network. Our analysis indicates that almost all national parks and/or wilderness areas are affected at deciview extremes, except the North Cascades National Park, the

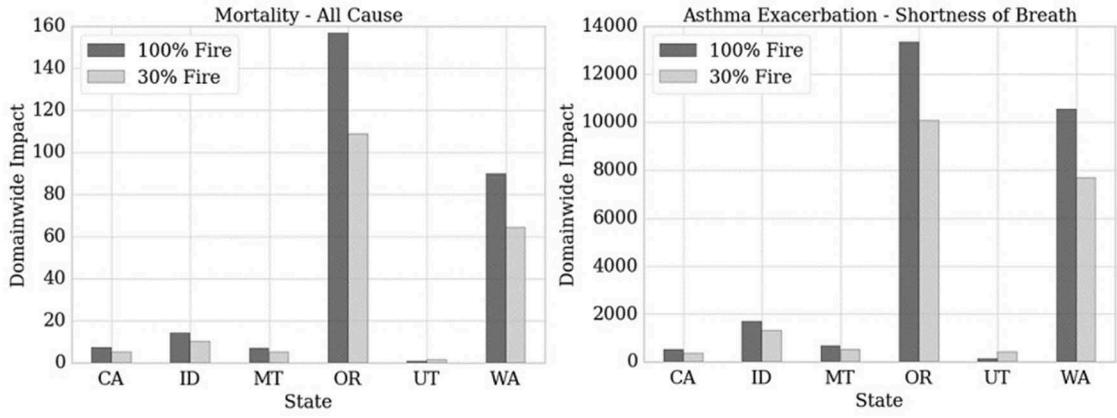

Figure 7. Prescribed fire attributed additional asthma attacks and mortality cases for various states in AIRPACT-4 model domain.

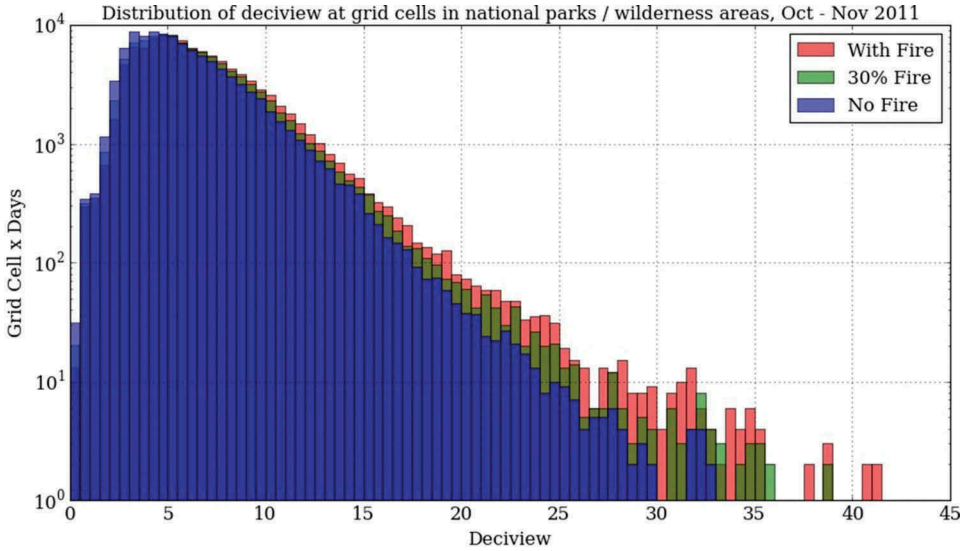

Figure 8. Deciview distribution for the grid cells in class I areas for the three emission scenarios.

Sawtooth National Forest, Olympic National Park, Mountain Rainier, and Sula Peak.

To analyze the change in visibility from fire emission reduction from a regulatory perspective, the regional haze rule approach of reasonable progress toward achieving the goal of natural visibility is followed here: improving the most impaired visibility days (i.e., 20% worst visibility days or haziest days) while not degrading the visibility on cleanest days during a year. The concentrations of various species required for calculated the reconstructed extinction coefficient were obtained from IMPROVE network for 2011. For each site in the AIRPACT domain (n = 26), the deciviews were calculated using eq 2, assuming hygroscopic organic aerosols. From this new deciview data, the cleanest and haziest 20% days were extracted. Using the deciviews during these cleanest and haziest 20% days, a relative response factor (RRF; USEPA 2014) was calculated for each of the components in eq 2, except for NO_2 and sea salt. However, when projecting the deciviews to the no fire and 30% fire cases, we applied the component specific RRFs only for the days in October–November 2011. We did this since a reduction will not affect the visibility during other times of the year when there are no prescribed fires. Thus, we created a deciviews time series at each IMPROVE network site in the following manner for the 20% cleanest and haziest days:

$$dv_{site,day} = \begin{cases} (dv_{RRF})_{site,day} \; if \; day \; in \; simulation \; period \\ (dv_{obs})_{site,day} \; if \; day \; not \; in \; simulation \; period \end{cases} \quad (4)$$

where dv_{RRF} is the RRF-based deciview and dv_{obs} is the observed deciview.

Average improvements in visibility for the 20% haziest days for each IMPROVE site are shown in Table 5. Sites where difference in deciview metric between the 100% fire and no fire or 30% fire case is comparable to the slope of Theil trend line (visibility trend at each site over the observed data set for 20% worst and best days; obtained from http://views.cira.colostate.edu/fed/siteBrowser/Default.aspx) are shown in bold. Our results show that at six sites (Columbia River Gorge, Monture, Craters of the Moon, Cabinet Mountains, Lava Beds, and Crater Lake), a no fire scenario will result in visibility improvements equal to or faster than current rate of improvements for the haziest 20% days given by the Theil trend. We also predict visibility improvements for the 30% fire scenario, with the largest rate of benefits for Monture, Glacier National Park, Crater Lake, Kalmiopsis, Cabinet Mountains, and Lava Beds. At Lava Beds and Crater Lake, the visibility improvements in the 30% fire scenario are also greater than the current rate of improvements given by the Theil trend. While we did see benefits from prescribed fires for the haziest 20% days, we did not see much difference for the cleanest 20% of the days and hence those are not reported here. These results show that even though prescribed fires are events planned to minimize smoke exposure, they contribute to impaired visibility in the protected natural environments, and their reduction via feedstock harvesting can improve visibility.

Table 5. Changes in average deciviews for the haziest 20% days during October–November 2011 at IMPROVE sites. Sites where the deciview improvements for the recued fire case are comparable with the Theil trend are shown in bold.

Site name	Site code	Deciview for 100% fire case	delta dv 100 % fire – No fire	delta dv 100% fire – 30% fire	Theil trend (dv/year)
Columbia River Gorge	**CORI1**	**20.87**	**−0.13**	**−0.05**	**−0.10**
Columbia Gorge #1	COGO1	19.42	−0.01	−0.02	−0.50
Three Sisters wilderness	**THSI1**	**18.74**	**−0.09**	**−0.04**	**−0.10**
Glacier NP	**GLAC1**	**18.17**	**−0.17**	**−0.07**	**−0.18**
Redwood NP	REDW1	17.93	−0.08	−0.03	−0.17
Mount Rainier NP	MORA1	16.97	0.00	0.00	−0.34
Starkey	STAR1	16.85	−0.16	−0.05	−0.44
Kalmiopsis	**KALM1**	**16.85**	**−0.13**	**−0.06**	**−0.14**
Hells Canyon	**HECA1**	**16.49**	**−0.11**	**−0.05**	**−0.16**
Mount Hood	**MOHO1**	**16.26**	**−0.09**	**−0.04**	**−0.16**
Trinity	TRIN1	16.15	−0.04	−0.02	−0.16
Snoqualmie Pass	SNPA1	15.96	−0.07	−0.03	−0.28
Monture	**MONT1**	**15.94**	**−0.19**	**−0.09**	**−0.10**
Flathead	FLAT1	15.77	−0.13	−0.06	−0.32
Olympic	OLYM1	15.77	−0.02	−0.01	−0.28
Craters of the Moon NM	**CRMO1**	**15.01**	**−0.04**	**−0.02**	**−0.04**
Lassen Volcanic NP	LAVO1	14.92	−0.07	−0.03	0.02
Cabinet Mountains	**CABI1**	**14.65**	**−0.13**	**−0.06**	**−0.08**
Sula Peak	SULA1	14.49	−0.01	0.00	0.00
Lava Beds NM	**LABE1**	**13.59**	**−0.13**	**−0.06**	**−0.05**
Crater Lake NP	**CRLA1**	**13.48**	**−0.16**	**−0.07**	**−0.06**
North Cascades	NOCA1	13.38	−0.01	0.00	0.00
Pasayten	PASA1	13.21	−0.06	−0.03	−0.29
Sawtooth NF	SAWT1	12.75	−0.01	0.00	−0.13
Gates of the Mountains	GAMO1	11.51	−0.03	−0.01	−0.01
White Pass	**WHPA1**	**11.38**	**−0.10**	**−0.04**	**−0.11**

Conclusion

In this study, we have considered the role of emissions from prescribed fires used for fuel management on model performance, regional and local air quality, visibility, and associated health impacts. We find that for the period of simulation, including the prescribed fire emissions improves the model performance both at urban AQS sites and at the rural IMPROVE network for several performance metrics. The mean observed $PM_{2.5}$ concentrations for the AQS and IMPROVE site were 8.41 μg/m^3 and 2.83 μg/m^3. When fire emissions are included in modeling we see an improvement in MFB and correlation coefficient; however, mean bias changes from −0.74 μg/m^3 to 1.11 μg/m^3 for IMPROVE sites (−0.87 μg/m^3 to 1.41 μg/m^3 for AQS sites) for the no fire and with fire cases, respectively, showing an overprediction in the mean concentration. However, when individual sites are considered, a small number of additional sites meet the MFB and MFE goals and criteria for $PM_{2.5}$ for the 100% fire scenario. Among PM species, maximum improvement occurs for OC, with performance significantly improving when simulating with all fire emissions. All sites were within criteria for EC, nitrate, sulfate, and ammonium aerosols in both the cases. When using the newer set of goals and criteria suggested by Emery et al. (2017) and based on NMB/NME, we see mixed results for the model performance. The model meets performance goals for $PM_{2.5}$ at AQS sites for the no fire case, while meeting criteria for the case where all fires are included. Similarly, at IMPROVE sites, the model starts overpredicting in the case where fire emissions are considered. Among speciated PM2.5, using the newer NMB/NME based goals and criteria, we find that model performance improves for organic carbon and ammonium, which form the bulk of $PM_{2.5}$ mass.

Our results show that a large part of the domain is affected by emissions from prescribed fires, with the most affected areas being western Oregon, northern Idaho, and western Montana. Under a scenario of 70% decrease in fire emissions (i.e., 70% avoided emissions when biomass is harvested for biofuels), we show that most of western Oregon and small areas in northern Idaho will see large decreases in average $PM_{2.5}$ concentrations. We have shown that while the impact from prescribed burning is small in more populated urban centers, for some small and tribal communities mean and peak concentrations can increase significantly, and these are the sites where a decrease in emissions can result in significant improvements in air quality. While our simulation period covers only a part of the year, results show that this reduction can also help some non-attainment and maintenance areas such as Pinehurst and Sandpoint in Idaho, where contributions from prescribed fires can be large. These indicate potential regional, as well as local, air quality benefits of avoided biomass burning and harvesting for a biofuel industry. Prescribed fires alone are expected to cause health impacts across an array of endpoints: 280–700 additional deaths, 4400 lower respiratory symptom cases, 7300 upper respiratory symptom cases, around 400 acute bronchitis cases, and several thousand workday

losses, among others. A reduction of 70% in the prescribed fire emissions will benefit by reducing mortality and morbidity by 25–30% for most of the endpoints.

Prescribed fires also contribute to impaired visibility in the protected class I areas. Following the concept of regional haze rule, we derived the RRF for different modeling scenarios, and analyzed the effects of reduced fire emissions on the visibility in protected natural environments in the model domain. We have shown that a reduction in fire emissions will improve visibility at number of IMPROVE sites during the 20% worst visibility days, whereas the improvement is minimal for the cleanest 20% days.

We have seen that the model performance during the period of simulation is not very good. Also, in the absence of detailed data on slash pile burning across the region, we have used the prescribed burning emission inventory available from the USEPA for our analysis. The imperfect modeling results and other assumptions embedded in our analysis make the results uncertain. Despite these uncertainties, based on our analysis we can conclude that a reduction in prescribed burning emissions due to feedstock harvesting can have significant local and regional air quality benefits, especially at small rural and tribal communities, and can also be beneficial for current non-attainment parts in the domain and trying to meet the national ambient air quality standard. From a policy perspective, the benefits from avoided management fires can be significant for public health and present an opportunity toward accelerated improvement for visibility in protected natural environments.

Funding

Funding for this work was provided by the Northwest Advanced Renewables Alliance (NARA) program, which is led by Washington State University and supported by the Agriculture and Food Research Initiative Competitive Grant number 2011-68005-30416 from the USDA National Institute of Food and Agriculture. The AIRPACT system used in this study is operated with support from NW-AIRQUEST (http://lar.wsu.edu/nw-airquest) and the Agriculture and Food Research Initiative, USDA National Institute of Food and Agriculture [2011-68005-30416].

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Environmental Research 150 (2016) 227–235

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

Differential respiratory health effects from the 2008 northern California wildfires: A spatiotemporal approach

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ARTICLE INFO

Article history:
Received 6 January 2016
Received in revised form
5 June 2016
Accepted 6 June 2016
Available online 15 June 2016

Keywords:
Wildland fires
Climate change
Vulnerable populations
Asthma
Air pollution

ABSTRACT

We investigated health effects associated with fine particulate matter during a long-lived, large wildfire complex in northern California in the summer of 2008. We estimated exposure to $PM_{2.5}$ for each day using an exposure prediction model created through data-adaptive machine learning methods from a large set of spatiotemporal data sets. We then used Poisson generalized estimating equations to calculate the effect of exposure to 24-hour average $PM_{2.5}$ on cardiovascular and respiratory hospitalizations and ED visits. We further assessed effect modification by sex, age, and area-level socioeconomic status (SES). We observed a linear increase in risk for asthma hospitalizations (RR=1.07, 95% CI=(1.05, 1.10) per 5 $\mu g/m^3$ increase) and asthma ED visits (RR=1.06, 95% CI=(1.05, 1.07) per 5 $\mu g/m^3$ increase) with increasing $PM_{2.5}$ during the wildfires. ED visits for chronic obstructive pulmonary disease (COPD) were associated with $PM_{2.5}$ during the fires (RR=1.02 (95% CI=(1.01, 1.04) per 5 $\mu g/m^3$ increase) and this effect was significantly different from that found before the fires but not after. We did not find consistent effects of wildfire smoke on other health outcomes. The effect of $PM_{2.5}$ during the wildfire period was more pronounced in women compared to men and in adults, ages 20–64, compared to children and adults 65 or older. We also found some effect modification by area-level median income for respiratory ED visits during the wildfires, with the highest effects observed in the ZIP codes with the lowest median income. Using a novel spatiotemporal exposure model, we found some evidence of differential susceptibility to exposure to wildfire smoke.

1. Introduction

Wildfires have been increasing in frequency and severity in western North America, and this increase has been associated with earlier spring snowmelt and higher temperatures (Westerling et al., 2006). The risk of wildfires is projected to increase in California (Westerling and Bryant, 2008; Westerling et al., 2011) and in many parts of the world (Liu et al., 2010; Moritz et al., 2012) under probable future climate change scenarios.

Smoke from wildfires contains many pollutants of concern for public health including nitrogen dioxide, ozone, carbon monoxide, polycyclic aromatic hydrocarbons, aldehydes, and particulate matter less than 2.5 μm in aerodynamic diameter ($PM_{2.5}$) (Naeher et al., 2007). Previous epidemiological studies of wildfire smoke exposure have found consistent evidence of respiratory health effects in general and most specifically for exacerbations of asthma and chronic obstructive pulmonary disease (COPD). Findings for other health outcomes have been inconsistent across studies, and insufficient research has investigated whether particular

Abbreviations: AOD, aerosol optical depth; BRFSS, Behavioral Risk Factor Surveillance System; CI, confidence interval; COPD, chronic obstructive pulmonary disease; CV, cross-validated; ED, emergency department; GBM, generalized boosting method; GEE, generalized estimating equations; GOES, Geostationary Operational Environmental Satellite; IHD, ischemic heart disease; IQR, interquartile range; MODIS, MODerate resolution Imaging Spectroradiometer; PM, particulate matter; $PM_{2.5}$, particulate matter less than or equal to 2.5 μm in aerodynamic diameter; QICu, Quasi Information Criterion for GEE models; RH, relative humidity; RMSE, root mean squared error; RR, relative risk; RUC, Rapid Update Cycle model; SES, socio-economic status; WRF-Chem, Weather Research and Forecasting model with Chemistry; ZCTA, ZIP code tabulation area

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http://dx.doi.org/10.1016/j.envres.2016.06.012

population subgroups are more susceptible to wildfire smoke exposure (Reid et al., 2016). Additionally, the existing literature lacks information on the shape of the exposure-response curve for wildfire smoke. Such information could be useful to decision-makers issuing health advisories during wildfire events.

This study investigates a particularly long-lived, large wildfire complex that occurred in northern California in the summer of 2008. A combination of meteorological conditions and difficulty with fire suppression contributed to very high air pollution levels throughout northern California (Reid et al., 2009). Smoke from the fires covered a large region with large population centers for almost six weeks, making this an important fire episode for analysis of public health effects.

We examined the effects of this relatively long exposure on cardiovascular and respiratory hospital admissions and emergency department (ED) visits within the population of northern and central California using a novel spatiotemporal exposure model. We aimed to assess if there were differential health effects of $PM_{2.5}$ during the wildfire compared to reference periods before and after the fires, to assess at what level of $PM_{2.5}$ the risk of adverse health effects starts to increase, and to identify population subgroups that were more susceptible to wildfire smoke during this event.

2. Methods

2.1. Study setting

The 2008 northern California wildfire complex consisted of thousands of wildfires ignited by a large lightning storm the weekend of June 20–21, 2008. Most of these fires were contained by the end of July 2008. We defined the pre-fire period as May 6 to June 19 (days=43), the fire period as June 20 to July 31 (days=42), and the post-fire period as August 1 to September 15 (days=46). These cut points were determined based on having similar numbers of days in the three time periods, the timing of the onset of the fires ignited by the lightning storm, and the designation that most of the fires had been contained by the end of July. The spatial confines of our analysis were the ZIP codes that fall within the following air basins: the Sacramento Valley, the San Francisco Bay Area, the Mountain Counties, Lake County, the North Central Coast, and the northern part of the San Joaquin Valley (Fig. 1). Most of the fires were located in mountainous regions that ringed the northern Central Valley: in the Trinity Alps west of Redding, the Sierra Nevada in the Mountain Counties to the east of Redding and Chico, and some fires near Big Sur, which is along the coast west of Fresno.

2.2. Exposure data

We estimated exposure to $PM_{2.5}$ for each day in each ZIP code using an exposure prediction model that was created from a large set of spatiotemporal data sets through data-adaptive machine learning methods. This method used 10-fold cross validation (CV) to select from within a large number of predictor variables and across many different statistical algorithms to optimize prediction of $PM_{2.5}$. The 24-hour average $PM_{2.5}$ values at 112 monitoring stations (Fig. 1) were used as the dependent variable. The predictor variables included aerosol optical depth (AOD) from the Geostationary Operational Environmental Satellite (GOES), output from the Weather Research and Forecasting coupled with Chemistry (WRF-Chem) model, various meteorological variables from the Rapid Update Cycle model, Julian date, weekend, amount of land use types within 1 km, the X-coordinate, the Y-coordinate, elevation, and traffic counts. In a previous paper (Reid et al., 2015), the generalized boosting model (GBM) predicted 24-hour average $PM_{2.5}$ better than the 10 other algorithms with a CV-R^2 of 0.80 using all of the predictor variables. In this analysis, we re-ran the GBM model and expanded the time period to include time periods before and after the fires. Accordingly, we removed predictor variables that were not available for the before and after time periods (e.g., local aerosol optical depth (AOD) and distance to the nearest fire cluster). In this modeling run, a GBM model containing 24 out of 25 possible predictor variables had an out-of-sample CV-R^2 of 0.79 and a CV root mean squared error (RMSE) of 1.44 $\mu g/m^3$, but including only the six most predictive variables resulted in almost equally good performance with a CV-R^2 of 0.78 and a CV-RMSE of 1.46 $\mu g/m^3$. The six most predictive variables were AOD from the GOES satellite, WRF-Chem output, Julian date, surface pressure, the X-coordinate and the Y-coordinate. The model predicted observed values better during the fires than before or after (Supplement Fig. S1).

We used this more parsimonious model to estimate exposures at the population-weighted centroid of each of 781 ZIP code tabulation areas (ZCTA), spatial constructs used by the US Census Bureau to create ZIP codes from census-area designations, using ArcGIS 10.1 (ESRI, 2012). The predictor variables in the exposure model are assigned to each ZCTA as the value of that input variable closest to the population-weighted centroid for that ZIP code. Predicted values for all ZIP codes in the study area over time are presented in Fig. 2.

2.3. Health data

We obtained daily counts of hospital admission visits (OSHPD, 2008b) and ED visits (OSHPD, 2008a) for each ZIP code in the study area for the following causes (ICD-9 code): asthma (493), COPD (496, 491–492), pneumonia (480–486), ischemic heart disease (IHD) (410–414), cardiac dysrhythmias and conduction disorders (426–427), heart failure (428), cerebrovascular disease (430–435, 437), and hypertension (401–405). The total population based on the 2010 US Census for all ZIP codes in the study area was 12.7 million.

2.4. Covariate data

Temperature and RH data are 24-hour averages taken from the Rapid Update Cycle (RUC) model from the National Climatic Data Center (http://ruc.noaa.gov/). We assigned the value from the grid cell that overlaid the population-weighted centroid of each ZIP code. We obtained estimates by ZIP code of population, median income, percent of the population over 65, percent of the population living in owner-occupied housing, and percent of the population with less than a high school diploma from the 2000 US Census. We used smoking prevalence estimates derived from Behavioral Risk Factor Surveillance System (BRFSS) data by ZIP code for the 2006–2010 time period based on the 2000 census ZIP codes (Ortega Hinojosa et al., 2014). For the ZIP codes (N=66, 8.5%) in our analysis that were created after 2000, we used county-level estimates. Daily 8-hour maximum ozone concentrations come from WRF-Chem.

2.5. Statistical analysis

We used Poisson generalized estimating equations (GEE) to calculate the population-averaged effect of exposure to $PM_{2.5}$ on cardiovascular and respiratory hospitalizations and ED visits during the summer of 2008 in northern California. We hypothesized that the effect of an increase in $PM_{2.5}$ during the wildfire period would be different than that in non-fire periods, and therefore included an interaction term indicating the time periods before, during, and after

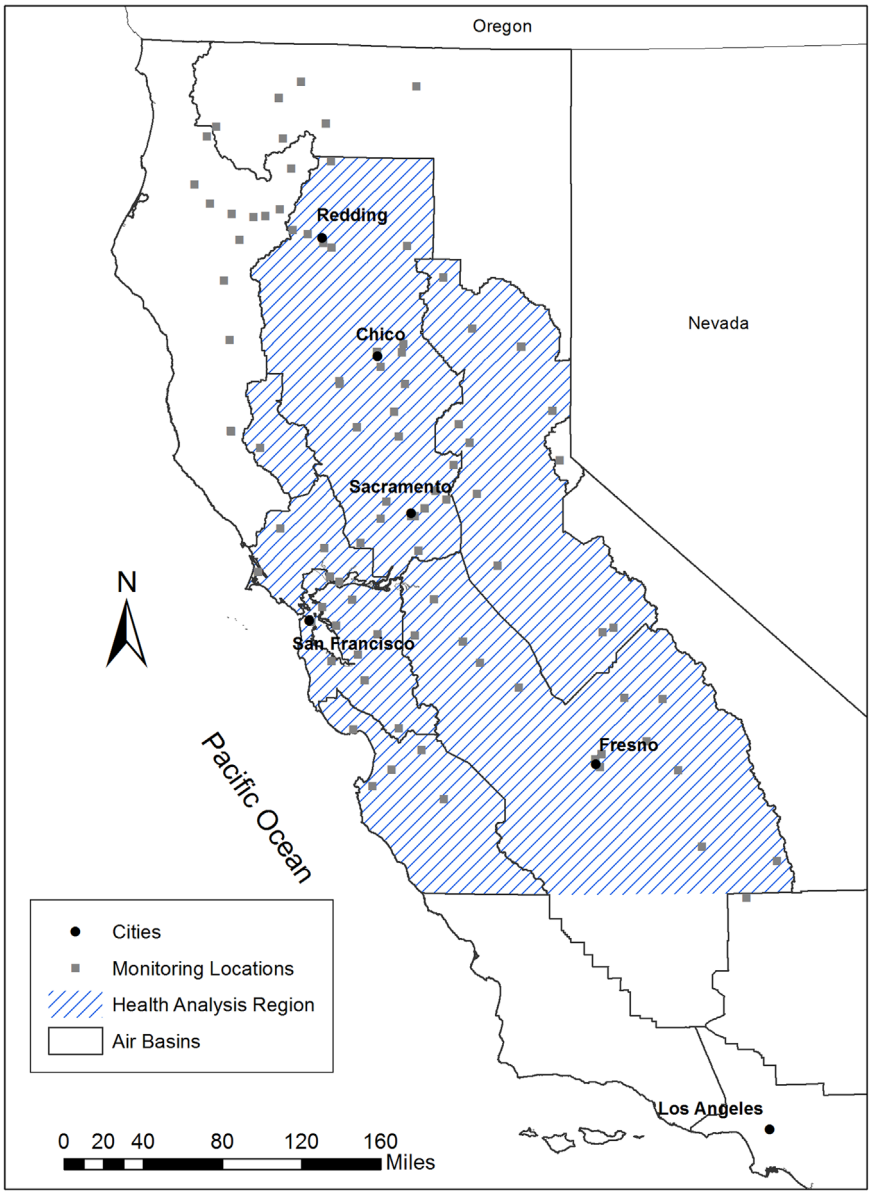

Fig. 1. Study region.

the fires. We used indicator variables to control for holiday and day of week effects. We assessed a variety of ways to control for temporal trend and found that a natural cubic spline on Julian date with 3 degrees of freedom (df) had the smallest Quasi Information Criterion for GEE models (QICu) (Hardin and Hilbe, 2003). To control for potential spatial confounding, we adjusted for smoking prevalence, median income as a measure of socio-economic status (SES), and percent of the population over 65 years of age (because elderly people may have increased susceptibility to wildfire health effects Reid et al., 2016) at the ZIP code level. Temperature and ozone are both spatiotemporal variables that could confound the $PM_{2.5}$-health relationship particularly during wildfires. We controlled for ozone and the heat index, a measure of apparent temperature that combines both temperature and RH, based on an algorithm used by the US National Weather Service that has been evaluated as the best of various apparent temperature metrics (Anderson et al., 2013). We found little difference between temperature and the heat index in our study domain (Pearson's r=0.995), but used heat index because other studies consider both temperature and RH to be confounders of the wildfire $PM_{2.5}$-health relationship (Delfino et al., 2009; Johnston et al., 2007). We also assumed *a priori* that the relationship between temperature and health would be linear, as the fires occurred only during the warm months. We used an exchangeable correlation structure with the sandwich estimator of the variance, which provides standard error estimates that are robust to misspecification of the covariance structure and also adjusts for any over-dispersion in the count data. We included the log of the size of the ZIP code population as an offset term.

Previous studies of the effects of $PM_{2.5}$ from wildfires on health have used various lags, mostly same-day, one-day, or two-day moving average. We initially investigated lags up to 28 days but did not find sustained effects (data not shown). We therefore applied a moving average of the two days prior to the date of hospital admission based on minimizing the QICu values. For the main analysis, we chose not to include same-day $PM_{2.5}$ in the moving average as we did not have access to the time of day of the hospitalization or ED visit and did not want to include counts of health outcomes that could have occurred before the exposure; however, we did a sensitivity analysis with the same-day data included (a three-day moving average).

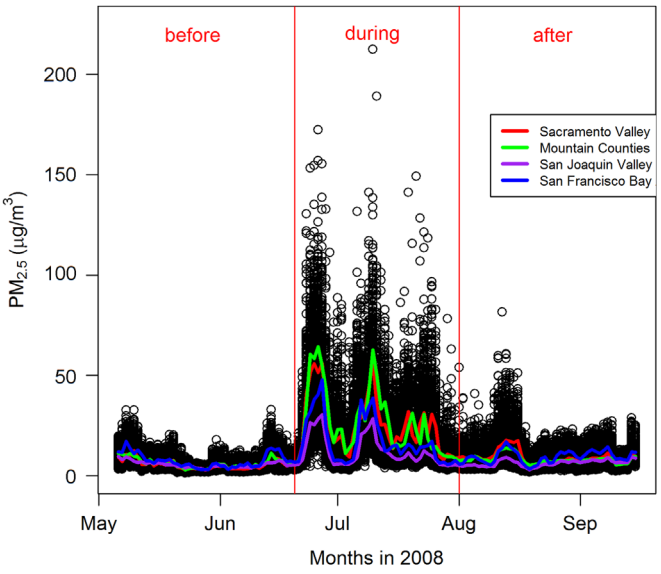

Fig. 2. $PM_{2.5}$ predictions by ZIP code for the before, during, and after fire periods with mean daily values for selected air basins. Open circles are predicted $PM_{2.5}$ values for each ZIP code for each day and colored lines represent the average value for all ZIP codes in that air basin.

Many epidemiological analyses of air pollution display their results as the effect for a change of 10 units or the interquartile range (IQR) in the pollutant of the exposures in the study. In an analysis over time, the effect estimates should represent the effect due to day-to-day differences in exposures, which may be much smaller (Snowden et al., 2015). We present our main findings as per 5 $\mu g/m^3$ $PM_{2.5}$ because our data had very few day-to-day changes of the IQR (6.7 $\mu g/m^3$) or higher, but 32% of ZIP code-days during the fire period and 11% overall experienced a day-to-day change of 5 $\mu g/m^3$or greater.

2.6. Exposure-response estimation

We evaluated the shape of the exposure-response function for wildfire smoke exposure by categorizing the continuously predicted 24-hour average $PM_{2.5}$ values to represent levels of the Air Quality Index (http://airnow.gov/index.cfm? action=aqibasics.aqi) updated for 2012. The categories we used were those considered to be good (< 12 $\mu g/m^3$), moderate (12.1–35.4 $\mu g/m^3$), unhealthy for sensitive groups (35.5–55.4 $\mu g/m^3$), and unhealthy, very unhealthy and hazardous ($> =55.5$ $\mu g/m^3$). We chose these cut-points because public health officials are given guidance on issuing advisories based on AQI levels as they get reported in the media (Lipsett et al., 2008). At this point, however, it is not clear that health effects increase with increasing values of $PM_{2.5}$ during wildfire episodes in the same way as they do for other forms of $PM_{2.5}$.

2.7. Identification of sensitive and vulnerable sub-populations

We evaluated the effect of $PM_{2.5}$ exposure during the fire period on hospitalizations and ED visits stratified by sex and age group (under 20 years old, 20–64 years old, and 65 and over). We also assessed effect modification by tertiles of ZIP code-level median income, percent of the population with less than a high school diploma, and percent of owner-occupied housing units. Counts of hospitalizations and ED visits by specific outcome and by these groups are presented in Supplemental Tables S1 and S2.

2.8. Sensitivity analyses

We performed the following sensitivity analyses: (1) using an exposure model that excluded variables that were highly correlated with those in the epidemiological models (i.e., Julian date, temperature, and RH), (2) including same day exposures along with the lag 1 and lag 2 exposures, thus a three-day moving average, (3) adjusting for temperature and relative humidity separately rather than combined in the heat index, and (4) including additional spatial covariates.

All statistical analyses were performed in R v. 2.15.3 Vienna, Austria (R Core Team, 2013). The Center for Protection of Human Subjects at the University of California, Berkeley deemed this work to be not human subjects research because the health data were administrative and not identifiable.

3. Results

3.1. Descriptive statistics

Daily $PM_{2.5}$ exposures were much higher during the fire period than in the periods before or after (Table 1 and Fig. 2). The heat index was much lower before the fires compared to during or after, likely due to seasonally lower temperatures in May and June compared to July, August, and September. Hospital and ED visits were highest in the before fire period.

Clear spatial differences in covariates existed by air basin (Table 2) demonstrating the need to control for purely spatial covariates that could confound the $PM_{2.5}$-health relationship. $PM_{2.5}$ from the wildfire was highest in the Sacramento Valley air basin, which was surrounded by fires, and the smoke funneled into the valley regardless of the wind direction.

3.2. Analyses by time period

During the fires, $PM_{2.5}$ was associated with both asthma hospitalizations (RR=1.07, 95% CI =(1.05, 1.10) per 5 $\mu g/m^3$ increase) and ED visits (RR=1.06, 95% CI =(1.05, 1.07) per 5 $\mu g/m^3$ increase); the association for ED visits was larger during than after the fires based on p-values of the interaction terms between $PM_{2.5}$ and time period (Tables 3 and 4). We also found a significant relationship between $PM_{2.5}$ and asthma ED visits and asthma hospitalizations before the wildfire period. ED visits for COPD were also associated with $PM_{2.5}$ during the fires (RR=1.02 (95% CI= (1.01, 1.04)) per 5 $\mu g/m^3$ increase); this was significantly different from effects found before but not after the fires. All-cause respiratory hospitalizations and ED visits were also associated with $PM_{2.5}$ during the fires, likely driven by asthma visits.

We found largely null results for cardiovascular disease outcomes related to $PM_{2.5}$ during the wildfires (Tables 3 and 4 and Supplement Figs. S2 and S3). Hypertension ED visits were associated with $PM_{2.5}$ after the fires but not before or during the fires. We also found an unanticipated protective association between $PM_{2.5}$ and congestive heart failure during the fire period. In both cases, rates were not significantly different from rates before the fire period.

3.3. Exposure-response analysis

For asthma hospitalizations and ED visits, the RR across exposure categories was not linear (Figs. 3 and 4). ED visits for COPD increased abruptly in the highest exposure category. We did not find differences in the shape of the exposure-response curves for the whole season compared to only the fire period (data not shown).

Table 1
Temporal descriptive statistics by time period.

	Full Season[a] N=102,311	Before Fires[a] N=33,583	During Fires N=32,802	After Fires N=35,926
Days (count)	131	43	42	46
Spatiotemporal Data – mean (SD)				
$PM_{2.5}$ (moving average of 24-hour average on lag days 1 and 2) ($\mu g/m^3$)	11.21 (10.78)	6.40 (3.17)	19.14 (15.48)	8.46 (3.99)
Temperature (°C)	21.29 (5.31)	18.77 (5.31)	22.48 (5.07)	22.57 (4.65)
RH (%)	52.75 (19.23)	52.50 (18.94)	54.67 (18.51)	51.24 (20.00)
Heat index moving average (°C)	21.78 (5.29)	18.83 (5.16)	23.25 (5.02)	23.18 (4.44)
Ozone (ppb)	54.40 (21.55)	47.63 (15.64)	59.69 (25.53)	55.92 (20.64)
Hospitalization Counts for whole area averaged by day: mean (min, max)				
All Respiratory	145.2 (92, 227)	174.7 (134, 227)	132.9 (92, 170)	128.9 (95, 165)
Asthma	26.6 (13, 49)	31.5 (17, 49)	23.4 (16, 38)	25.0 (13, 44)
COPD	34.6 (17, 52)	39.4 (24, 52)	32.2 (19, 46)	32.3 (13, 44)
Pneumonia	70.9 (33, 117)	86.8 (69, 117)	66.4 (33, 89)	60.1 (39, 83)
All CVD	390.9 (238, 509)	407.7 (254, 509)	387.7 (250, 480)	378.3 (238, 498)
Ischemic heart disease	125.1 (67, 176)	129.5 (71, 176)	124.4 (67, 169)	121.6 (67, 172)
Congestive heart failure	68.3 (39, 102)	73.9 (39, 102)	67.9 (45, 94)	63.6 (43, 85)
Dysrhythmias	63.3 (25, 98)	65.3 (39, 98)	63.0 (35, 86)	61.7 (25, 86)
Hypertension	19.6 (7, 33)	21.0 (9, 33)	18.4 (7, 27)	19.4 (7, 30)
Cerebrovascular disease	75.8 (45, 109)	78.2 (52, 109)	75.4 (51, 102)	78.8 (45, 100)
Emergency Department Visit Counts for whole area averaged by day: mean (min, max)				
All respiratory	752.0 (516, 1083)	883.6 (704, 1067)	665.6 (534, 852)	698.7 (516, 951)
Asthma	142.5 (83, 244)	169.1 (111, 244)	124.9 (85, 182)	133.7 (83, 211)
COPD	65.2 (41, 96)	71.3 (51, 94)	62.2 (41, 80)	61.3 (45, 78)
Pneumonia	113.6 (71, 176)	138.9 (112, 175)	103.9 (71, 134)	97.0 (73, 121)
All CVD	421.8 (342, 497)	430.6 (356, 493)	415.0 (364, 492)	414.5 (342, 490)
Ischemic heart disease	82.5 (60, 104)	84.3 (62, 103)	81.6 (62, 103)	81.1 (60, 101)
Congestive heart failure	77.2 (49, 110)	81.7 (48, 109)	76.5 (56, 101)	72.3 (55, 93)
Dysrhythmias	99.0 (63, 131)	99.6 (78, 128)	97.7 (63, 119)	98.2 (78, 128)
Hypertension	61.7 (39, 96)	61.7 (42, 81)	58.7 (39, 81)	63.5 (42, 96)
Cerebrovascular disease	78.5 (53, 103)	80.3 (63, 99)	78.0 (62, 95)	76.8 (53, 103)

N is the number of ZIP code-days, the unit of analysis.

[a] data as analyzed with two lagged days removed.

3.4. Sensitive and vulnerable populations

ED visits for asthma were significantly associated with $PM_{2.5}$ during the fire period for all age groups, with a nonsignificant increase in effect with increasing age (Supplement Table S3 and Fig. 5). Associations between $PM_{2.5}$ and hospitalizations for asthma were only present for ages 20–64 and ages ≥ 65 (Supplement Table S4). During the wildfires, individuals aged 20–64 had a significantly higher RR for COPD ED visits associated with $PM_{2.5}$ compared to those ≥ 65 (Fig. 5).

We also found some differences by sex. The association between $PM_{2.5}$ and ED visits for asthma and hypertension were significantly higher for females compared to males (Fig. 6 and Supplement Tables S5 and S6).

Asthma ED visits were significantly associated with $PM_{2.5}$ during the wildfires for all levels of SES (Supplement Fig. S4). The only consistent differential effects across tertiles of SES metrics were for ZIP-code level median income and respiratory ED visits. For asthma, COPD, pneumonia, and all-cause respiratory ED visits, there was a clear declining RR with increasing ZIP-code level median income (Fig. 7), but this was not observed for other respiratory outcomes (Supplement Tables S7 and S8).

3.5. Sensitivity analyses

Effect estimates were generally consistent across sensitivity analyses (Supplement Tables S9–S16) compared to our main model. Other formulations of the exposure model, inclusion of same-day hospitalizations and ED visits, and use of the heat index compared to temperature and relative humidity separately did not appreciably change the associations found with the main model.

4. Discussion

We found a significant relationship between $PM_{2.5}$ from wildfires and respiratory hospitalizations and ED visits. We used a sophisticated spatiotemporal exposure model with excellent performance in predicting $PM_{2.5}$ concentrations measured at air quality monitoring stations (out of sample CV-R^2 of 0.79), which may have enhanced our ability to detect subtle health effects. The most consistent effects were for asthma, with significant increases in hospitalizations and ED visits with a clear linear exposure-response relationship in categorical exposure models for ED visits. Regardless of level of SES for three measures of SES, there was a clear indication that increasing $PM_{2.5}$ levels during the wildfire events was associated with increased ED visits for asthma. We also observed some evidence that women were more susceptible than men to the effects of $PM_{2.5}$ during a wildfire on asthma. The finding of significant effects of asthma hospitalizations and ED visits before the fires as well as during and the lack of consistent interaction terms between time periods could imply that the effects of $PM_{2.5}$ on respiratory health outcomes are from $PM_{2.5}$ in general and not different by source of $PM_{2.5}$. This would imply that the risk associated with wildfires is due mainly to the heightened levels of exposure.

Our results are comparable to previous studies, particularly larger studies with spatiotemporal exposure assessments (Delfino et al., 2009; Henderson et al., 2011). The study most similar to ours found significant increases in respiratory hospitalizations associated with $PM_{2.5}$ during wildfire periods and also found few significant differences between the effects observed during the wildfires compared to after the wildfires (Delfino et al., 2009). Growing evidence suggests that wildfire smoke exposure is associated with exacerbation of COPD (Reid et al., 2016). Although we

Table 2
Spatial descriptive statistics by air basin.

	Full Area	Sacramento	San Joaquin	Mountain	Lake Tahoe	San Francisco	Lake County	North Coast	North Central Coast
Spatial Data – mean (SD)									
ZIP codes	781	173	168	108	8	248	13	17	46
Median income ($)	55,630 (26,510)	47,092 (18,956)	39,010 (13,359)	46,528 (17,057)	49,693 (21,897)	76,808 (28,923)	39,246 (10,525)	59,490 (27,546)	59,870 (18,446)
% less than HS education	12.88 (6.68)	13.80 (5.26)	10.65 (4.93)	17.59 (8.86)	6.71 (3.32)	11.87 (5.89)	20.95 (6.04)	13.12 (8.09)	10.69 (7.00)
% owner-occupied housing	63.90 (16.97)	67.74 (15.03)	60.18 (15.07)	74.76 (11.33)	64.58 (11.99)	59.57 (19.74)	72.22 (7.16)	63.06 (8.26)	58.7 (14.65)
Total Population	16,316 (18,150)	13,485 (16,471)	15,712 (16,997)	3716 (6121)	5825 (10,401)	26,254 (19,293)	4479 (4353)	2944 (4480)	15,293 (18,152)
Smoking prevalence	0.17 (0.04)	0.20 (0.03)	0.17 (0.04)	0.20 (0.02)	0.16 (0.03)	0.15 (0.04)	0.23 (0.01)	0.17 (0.03)	0.15 (0.03)
Spatiotemporal Data – mean (SD), [min, max]									
$PM_{2.5}$ moving average ($\mu g/m^3$)	11.21 (10.78) [1.62, 200.86]	13.16 (13.81) [1.82, 144.79]	7.92 (5.95) [2.91, 63.81]	13.29 (14.59) [1.62, 200.86]	15.18 (14.82) [1.96, 106.52]	11.27 (8.31) [2.39, 106.17]	14.72 (15.75) [2.42, 99.01]	13.54 (11.10) [2.22, 82.44]	8.29 (4.61) [2.08, 38.73]
Temperature (°C)	21.29 (5.31)	23.76 (4.23)	24.73 (4.09)	22.49 (5.14)	15.29 (4.59)	17.68 (4.22)	21.99 (3.76)	17.91 (4.11)	18.23 (4.45)
RH (%)	52.75 (19.23)	43.73 (12.77)	43.45 (11.12)	38.44 (14.48)	42.09 (16.37)	68.59 (16.3)	46.41 (14.43)	63.65 (17.28)	68.51 (17.24)
Heat index moving average (°C)	21.78 (5.29)	24.22 (4.2)	25.3 (4.18)	22.6 (5.13)	14.97 (4.58)	18.29 (4.17)	22.38 (3.7)	18.42 (4.06)	18.9 (4.51)
Ozone (ppb)	54.40 (21.55)	60.78 (18.16)	69.10 (16.96)	65.41 (17.50)	60.80 (15.64)	38.44 (17.33)	47.94 (16.95)	36.03 (11.76)	44.53 (15.60)

did not find significant increases in hospitalizations for COPD associated with $PM_{2.5}$ during the fire period, other studies have observed such effects (Delfino et al., 2009; Morgan et al., 2010). We did find significantly elevated ED visits for COPD during the fire period, which has been found in one other study (Rappold et al., 2011).

Our study, similar to many wildfire epidemiological studies, did not find evidence of significant effects of $PM_{2.5}$ from wildfires on hospitalizations or ED visits for cardiovascular disease (Hanigan et al., 2008; Henderson et al., 2011; Martin et al., 2013; Morgan et al., 2010). A few recent papers, however, have found significant effects for out-of-hospital cardiac arrests (Dennekamp et al., 2015; Haikerwal et al., 2015), hospitalizations for hypertension (Arbex et al., 2010), cardiovascular clinic visits (Lee et al., 2009), ED visits for congestive heart failure (Rappold et al., 2011), and hospitalizations for IHD (Johnston et al., 2007).

One important contribution to the literature on wildfire-health effects is our analysis of the exposure-response function for $PM_{2.5}$ during wildfires, as very few other studies have investigated this. Our findings are in line with Johnston et al. (2002) and Thelen et al. (2013) in finding increasingly significant respiratory health impacts with increasing concentrations of PM during wildfire periods.

Another important finding from our study relates to differential effects on certain populations. We found that women were more likely to be hospitalized and visit the ED for asthma and visit the ED for hypertension than men when exposed to high levels of $PM_{2.5}$ during wildfires. The only other study that investigated differential effects by sex on asthma hospitalization also found higher rates of asthma hospitalizations for women than men during the 2003 southern California wildfires (Delfino et al., 2009). To our knowledge, no other study has investigated differential gender effects of wildfire $PM_{2.5}$ on hypertension outcomes. It is not clear if women with asthma have greater biological susceptibility to wildfire smoke, if women are more likely to seek medical care for asthma exacerbations, if women are more likely to have uncontrolled asthma that would lead to heightened susceptibility, if it is some combination of these reasons, or due to chance.

We also found greater impact of wildfire air pollution on hospitalizations and ED visits for asthma among people aged 20–64 than those younger and older. Although this has been found in other previous wildfire studies in which middle-aged adults had higher odds of physician visits for asthma associated with PM_{10} during a wildfire than younger or older groups (Henderson et al., 2011), another study found the highest relative rates of asthma hospitalizations associated with $PM_{2.5}$ during a wildfire among people aged 65 and older (Delfino et al., 2009). In general, results on differential age effects have been inconsistent, and therefore this potential susceptibility factor should be further studied.

Interestingly, regardless of area-level SES - as measured by median income, high school graduation prevalence, prevalence of owner-occupied housing, or race - ED visits for asthma were significantly associated with $PM_{2.5}$ during the wildfire period (Supplement Fig. S4). Although we found consistent effects across tertiles of all measures of SES (Supplement Table S6), we did find some evidence of effect modification by ZIP code median income, with higher RRs with decreasing median income. Henderson et al. (2011) found no clear differences by neighborhood SES in associations between physician visits and various exposure metrics of wildfire smoke. Among counties affected by smoke from a peat fire in North Carolina, counties with lower SES had higher rates of ED visits for asthma and congestive heart failure compared to counties with higher SES (Rappold et al., 2012). Further research is needed to understand differential vulnerability to wildfire smoke exposure by SES.

This study made many comparisons to further understand population health effects and vulnerability to wildfire smoke, an

Table 3
Relative risks of hospitalization associated with $PM_{2.5}$ before, during, and after the 2008 northern California wildfires.

	RR for a 5 μg/m^3 change in $PM_{2.5}$			p-value comparing during to before	p-value comparing after to during
	Before Fires RR (95% CI)	During Fires RR (95% CI)	After Fires RR (95% CI)		
All respiratory	0.987 (0.946, 1.030)	1.018 (1.007, 1.029)	1.002 (0.959, 1.047)	0.165	0.473
Asthma	1.143 (1.042, 1.253)	1.073 (1.045, 1.101)	1.015 (0.928, 1.110)	0.185	0.227
COPD	0.890 (0.815, 0.971)	1.014 (0.992, 1.036)	1.048 (0.964, 1.140)	0.004	0.441
Pneumonia	0.966 (0.912, 1.024)	1.008 (0.991, 1.024)	1.001 (0.944, 1.062)	0.176	0.830
Cardiovascular disease	0.994 (0.968, 1.021)	0.995 (0.988, 1.002)	0.988 (0.965, 1.012)	0.969	0.577
Congestive disease	0.984 (0.925, 1.048)	0.987 (0.971, 1.003)	0.991 (0.935, 1.051)	0.943	0.878
Ischemic heart disease	1.003 (0.953, 1.055)	0.997 (0.984, 1.010)	0.986 (0.943, 1.030)	0.821	0.615
Dysrhythmias	1.013 (0.951, 1.079)	1.000 (0.984, 1.017)	1.022 (0.966, 1.082)	0.702	0.455
Cerebrovascular disease	0.980 (0.919, 1.046)	0.985 (0.970, 1.000)	0.974 (0.917, 1.033)	0.901	0.716
Hypertension	0.940 (0.840, 1.053)	1.002 (0.968, 1.037)	1.015 (0.905, 1.140)	0.290	0.825

All models are for the two-day moving average controlling for time trend, day of week, heat index, median income, percent of the population over 65, smoking prevalence, and ozone.

area that has not been sufficiently studied. We did not apply multiple testing corrections and thus p-values and confidence intervals should be interpreted accordingly. We do not claim that these results are definitive; rather they should be taken as part of a larger body of work on wildfire smoke exposure and health effects. This study used a novel spatiotemporal exposure model and the findings are generally in alignment with other studies of wildfire smoke exposure. Comparison of our results with those from future studies with spatiotemporal exposure modeling should provide better insight into the value of this approach.

We only investigated one air pollutant ($PM_{2.5}$) from these wildfires. Wildfires cause increases in other air pollutants of concern for public health. In ongoing research, we are modeling health effects of ozone from wildfires, which has been only minimally studied (Azevedo et al., 2011; Jalaludin et al., 2000). Our spatiotemporal modeling of ozone and $PM_{2.5}$ will allow assessment of effect modification and effect decomposition in meaningful ways.

Although exceptions exist (Szpiro et al., 2011), better exposure assessment can improve health effect estimation by decreasing exposure misclassification. Our exposure model predicted better during the fire period than in the before and after periods of the fires (Supplement Fig. S1). The health effects observed during the fires could be stronger than those before or after the fires because of better prediction by the exposure model, even though very few of the findings were significantly different between time periods in the main analysis. Indeed, the standard errors during the fire period are much smaller than those in the other two time periods. One of the likely reasons for better prediction during the fires is because satellite AOD, the strongest predictor in our exposure model, better predicts PM in the western US during high pollution events such as wildfires (Gupta et al., 2007).

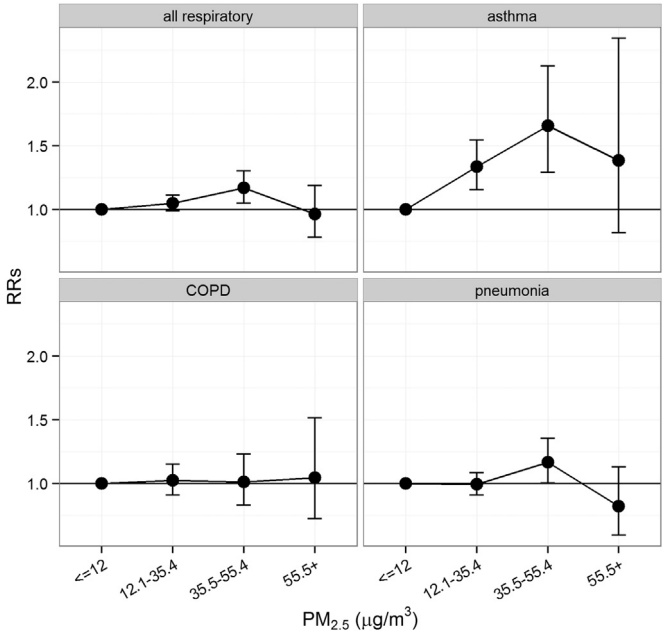

Fig. 3. Exposure-response for respiratory hospitalizations during the wildfire period.

Table 4
Relative risks of ED visits associated with $PM_{2.5}$ before, during, and after the 2008 northern California wildfires.

	RR for a 5 μg/m^3 change in $PM_{2.5}$			p-value comparing during to before	p-value comparing after to during
	Before Fires RR (95% CI)	During Fires RR (95% CI)	After Fires RR (95% CI)		
All respiratory	0.987 (0.968, 1.007)	1.015 (1.009, 1.020)	0.988 (0.967, 1.010)	0.008	0.019
Asthma	1.046 (1.000, 1.095)	1.056 (1.045, 1.068)	0.965 (0.925, 1.008)	0.682	0.000
COPD	0.959 (0.896, 1.027)	1.022 (1.006, 1.039)	1.043 (0.987, 1.102)	0.072	0.482
Pneumonia	0.939 (0.899, 0.980)	1.001 (0.989, 1.014)	0.99 (0.945, 1.036)	0.006	0.621
Cardiovascular Disease	1.003 (0.979, 1.028)	0.993 (0.987, 0.999)	1.000 (0.975, 1.026)	0.444	0.577
Congestive Heart Failure	0.980 (0.924, 1.040)	0.982 (0.967, 0.998)	1.033 (0.976, 1.092)	0.947	0.074
Ischemic Heart Disease	0.998 (0.946, 1.053)	0.997 (0.983, 1.011)	0.985 (0.931, 1.041)	0.965	0.654
Dysrhythmias	1.007 (0.961, 1.056)	0.995 (0.981, 1.010)	0.992 (0.939, 1.049)	0.649	0.916
Cerebrovascular Disease	0.988 (0.930, 1.051)	0.987 (0.973, 1.002)	0.979 (0.925, 1.037)	0.972	0.784
Hypertension	1.021 (0.953, 1.092)	1.012 (0.995, 1.029)	1.066 (1.008, 1.127)	0.818	0.080

All models are for the two-day moving average controlling for time trend, day of week, heat index, median income, percent of the population over 65, smoking prevalence, and ozone.

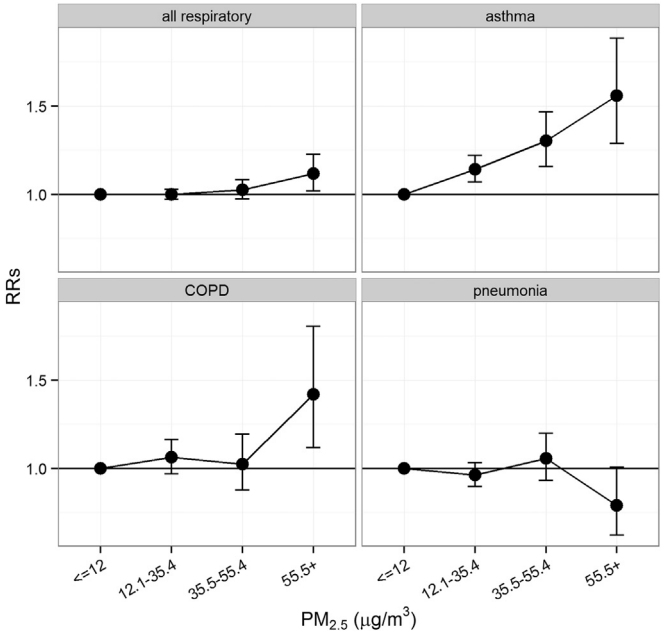

Fig. 4. Exposure-response for respiratory ED visits during wildfire period.

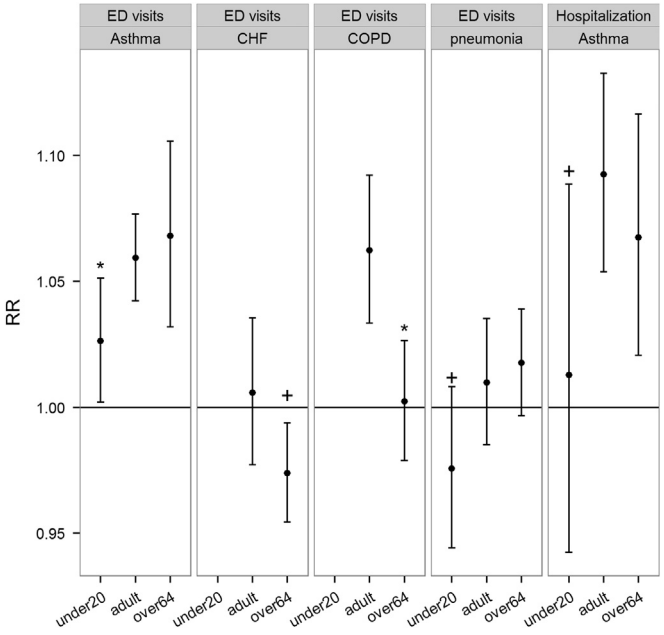

Fig. 5. Relative risks for a 5 μg/m^3 increase in $PM_{2.5}$ during the fire period by age group for respiratory hospitalizations. *denotes $p < 0.05$ level and + denotes $p < 0.10$ level for that age group compared to the adult (reference) age group during the fire period. No effect estimate is presented for the under20 age group for hospitalization for COPD because of so few observations of this health outcome in that group.

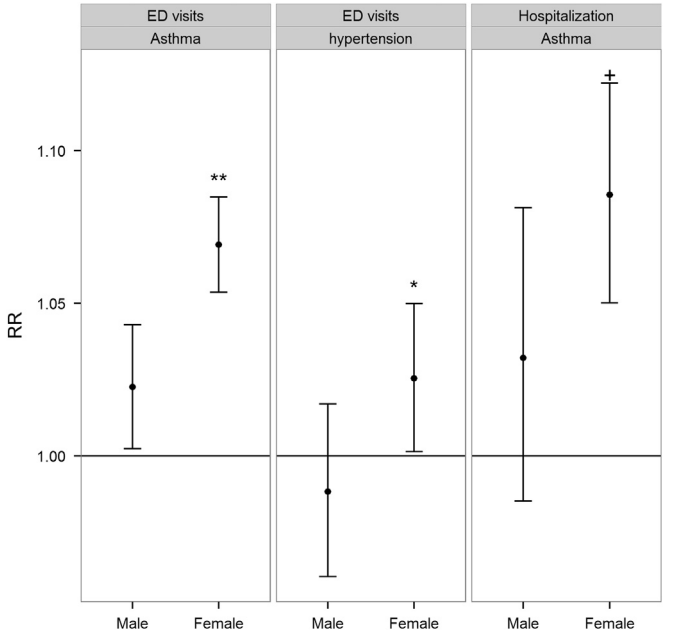

Fig. 6. Relative risks for a 5 μg/m^3 increase in $PM_{2.5}$ during the fire period by sex. **denotes $p < 0.01$, * denotes $p < 0.05$, and + denotes $p < 0.10$ for females compared to males during the fire period.

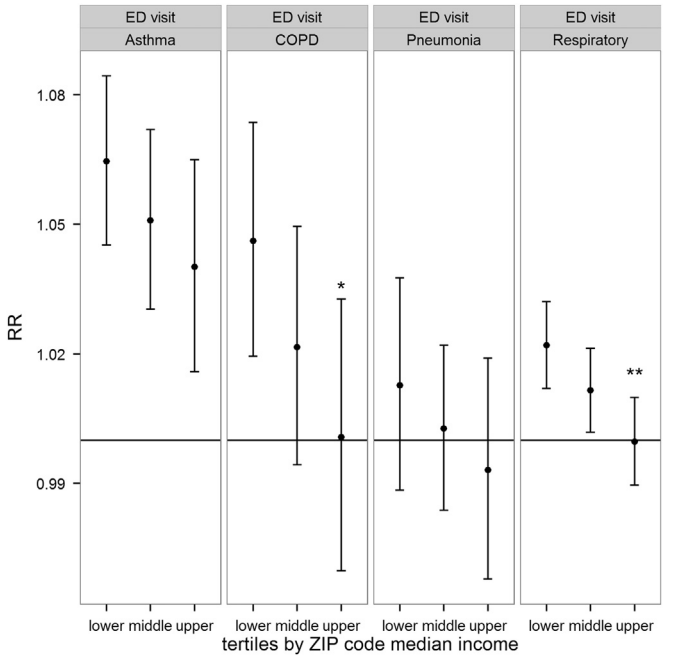

Fig. 7. Relative risks for a 5 μg/m^3 increase in $PM_{2.5}$ during the fire period by tertile of owner-occupied housing. **denotes $p < 0.01$, * denotes $p < 0.05$, and + denotes $p < 0.10$ compared to the lower tertile.

5. Conclusions

Using a novel spatiotemporal exposure model, we found that hospitalizations and ED visits for asthma were significantly associated with $PM_{2.5}$ during the 2008 northern California wildfires and that these effects increased with increasing $PM_{2.5}$ levels. Our results align with other studies that have used spatiotemporal exposure models (Delfino et al., 2009; Henderson et al., 2011) as well as more traditional exposure assignment methods (Johnston et al., 2007). We identified some differential effects by sex, age, and SES that should be further studied to determine if these groups are more vulnerable to wildfire smoke exposure. Our results add to the growing understanding of health risks associated with wildfire smoke, an exposure of increasing importance globally.

Funding sources

This research was supported under a cooperative agreement from the Centers for Disease Control and Prevention through the Association of Schools of Public Health [Grant Number CD300430],

an EPA STAR Fellowship Assistance Agreement [no. FP-91720001-0] awarded by the U.S. Environmental Protection Agency (EPA) and a grant from the Bureau of Land Management [grant number L14AC00173]. The views expressed in this article are solely those of the authors and EPA does not endorse any products or commercial services mentioned in this article.

The Center for Protection of Human Subjects at the University of California, Berkeley deemed this work to be not human subjects research because the health data were administrative and not identifiable.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.envres.2016.06.012.

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RESIDUES OF
FIRE ACCELERANT CHEMICALS

VOLUME I:
RISK ASSESSMENT

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Abstract

This report summarizes the results of quantitative human health and ecological risk assessments of chemical residues in the environment from the use of a variety of accelerants to ignite prescribed burns. On a per-unit basis for each ignition method, no risks were identified for human health, nor for general wildlife species. However, consideration should be given at the planning stage to protecting sensitive aquatic species in small watersheds that have limited potential for diluting residue chemicals that may run off or erode to surface water.

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RISK ASSESSMENT: RESIDUES OF FIRE ACCELERANT CHEMICALS

1.0 INTRODUCTION

The U.S. Forest Service and other natural resource agencies use a variety of chemical products to ignite prescribed burns in grass, shrub, and forest vegetation. The potential health and environmental impacts of these products has not been previously evaluated in a systematic manner, within the framework of their use as fire accelerants. This report presents an assessment of the potential impacts of these products.

This document is organized into nine major sections. Section 1.0 provides an introduction, background information, and an overview of the analysis approach. Sections 2.0 through 4.0 address the human health risks, including a hazard assessment (Section 2.0), exposure assessment (Section 3.0) and human health risk characterization (Section 4.0). Sections 5.0 through 7.0 present the ecological risk assessment, consisting of problem formulation (Section 5.0), analysis (Section 6.0) and ecological risk characterization (Section 7.0). Section 8.0 lists the references cited throughout this report.

1.1 BACKGROUND: FIRE ACCELERANTS

There are several types of equipment and associated chemical products that may be used to ignite prescribed burns, as summarized in this section.

Fusees (J. Schalau photo).

Fusees. Essentially the same as railroad flares, fusees can be attached together or put on a stick, are ignited, and then the employee will walk through the target area using it as a hand ignition device. A lit fusee may also be tossed to the target area. They contain strontium nitrate and potassium perchlorate.

Drip torch. A gasoline/diesel fuel mixture is dispensed by hand from a cylindrically shaped aluminum container with a handle. When inverted, fuel drips out of the container and a wick-like device on the top of the cylinder ignites the fuel. Authorized mixtures are one gallon of gasoline to three, four, or five gallons of diesel fuel.

Drip torch in use (BLM photo).

Use of gelled gasoline (Forest Service photo).

Helitorch/terra-torch or gelled gasoline in hand-thrown plastic bags. Gasoline can be mixed with a gelling agent, generally aluminum carboxylates ("alumagel"), which solidifies the gasoline into a jelly-like form, sold under the trade names FireGel®, Surefire®, and Petrol Gel®. The two substances are mixed in a mixing and storage tank, to

which is attached a pump and firing wand. Gelled gasoline is sent through the pump and ignited by a propane lighter. The gelled fuel is projected from 20 to 150 feet, coating the vegetation. When this type of system is mounted on a helicopter, it is called a helitorch. When installed in a ground-based vehicle, it is a terra-torch. Gelled gasoline can also be hand-tossed to the target area in plastic bags with a section of igniter cord attached and lit.

Aerial ignition device system ("ping-pong balls"). Polystyrene spheres, about the size of ping-pong balls and containing potassium permanganate crystals, are dispensed from a machine mounted in a helicopter. Just prior to release, a small amount of ethylene glycol is automatically injected into each sphere by the dispensing machine. Within 20 to 30 seconds, the sphere ignites.

"Ping-pong ball" aerial ignition system (Forest Service Research photo).

Incendiary devices (flares) propelled by launcher pistols. A launcher pistol can be used to propel a flare-like device to the target area. A typical flare may contain aluminum, calcium sulfate, iron oxide, and other minor ingredients.

Launcher pistol firing flare (Quoin International, Inc.)

Propane dispensed from backpack tanks. A propane tank is carried on the back, supplying propane to a wand with an ignition device, which is used to directly apply flame to target fuels.

Propane-fueled wand (J. Wolf).

The chemicals that form the basis for the target fuel ignition process in each of these methods are collectively referred to as *accelerants*. When accelerants, or any other substances, are oxidized during the burning process, new chemicals may be formed. Many of these are gaseous or particulate chemicals that are quickly dispersed in the open air. However, it is possible that some solid or liquid residues may remain on the soil after these accelerants are used to start a prescribed burn. These accelerant residues are the focus of this risk assessment.

1.2 IDENTIFICATION OF ACCELERANT RESIDUES

The purpose of this assessment is to estimate the risks to human health and the environment from residues remaining after the use of fire accelerants. The chemicals addressed in this risk assessment were selected based on the residues identified in Table 1-1.

Based on the combustion products identified in Table 1-1 for each accelerant, the solid or liquid residues that may potentially be present are as follows:

- Aluminum
- Aluminum oxide

Table 1-1. Chemicals Evaluated in Risk Assessment

Accelerant	Components	Residues[1]
Fusee[2]	Strontium nitrate + Potassium perchlorate + Sulfur + Sawdust/oil binder	Strontium sulfate Strontium oxide Strontium sulfide *Nitrogen oxides* Potassium chloride Potassium hydroxide *Carbon dioxide* *Water vapor* *Sulfur dioxide*
Gasoline	Gasoline mixture + MTBE (additive)	Gasoline as a mixture MTBE
Diesel fuel	Diesel fuel mixture	Diesel fuel as a mixture
Firegel/Alumagel/Surefire/ Petrol Jel	Aluminum carboxylates	Aluminum oxide *Water vapor*
Ping-pong balls	Potassium permanganate + Ethylene glycol + Polystyrene ball	Manganese dioxide Potassium hydroxide *Carbon dioxide* *Water vapor* *Styrene* Uncombusted polystyrene
Flares propelled by launcher pistols	Aluminum + Calcium sulfate + Iron oxide + Copper oxide + Silicon + Potassium perchlorate + Lead oxide + Black powder: (Potassium nitrate + Sulfur + Charcoal)	Aluminum Aluminum oxide Calcium sulfate Iron oxide Copper oxide Silicon dioxide Potassium chloride *Carbon dioxide* *Water vapor* Lead Potassium hydroxide *Nitrogen oxides* *Sulfur dioxide*
Propane	Propane	*Carbon dioxide* *Water vapor*

[1]Gaseous compounds are presented in italics; they are not analyzed in this assessment.
[2]Fusees may not be completely used during fire ignition: an end piece remains that may contain some unburned fuel. This endpiece is generally tossed into the fire, where it would be consumed.
Sources: Etiumsoft 2002, Lewis 1994a, Lewis 1994b, Sumi and Tsuchiya 1971.

- Calcium sulfate
- Copper oxide
- Diesel fuel
- Gasoline
- Iron oxide

- Lead
- Manganese dioxide
- MTBE
- Polystyrene
- Potassium chloride
- Potassium hydroxide
- Silicon dioxide
- Strontium oxide
- Strontium sulfate
- Strontium sulfide

1.3 OVERVIEW OF THE HUMAN HEALTH RISK ASSESSMENT

To assess the risk of human health effects from residues of fire accelerant chemicals, it was necessary to estimate the human exposures that could occur as a result of their application and associated activities, and to estimate the probability and extent of adverse health effects that could occur as a result of those exposures. This risk assessment employs the three principal analytical elements that the National Research Council (1983) described and EPA (1989, 2000a) affirmed as necessary for characterizing the potential adverse health effects of human exposures to existing or introduced hazards in the environment: hazard assessment, exposure assessment, and risk characterization.

Hazard assessment requires gathering information to determine the toxic properties of each chemical and its dose-response relationship. Human hazard levels are derived primarily from the results of laboratory studies on animals. The goal of the hazard assessment is to identify acceptable doses for noncarcinogens, and identify the cancer potency of potential carcinogens.

Exposure assessment involves estimating doses to persons potentially exposed to the accelerant residues. In the exposure assessment, dose estimates were made for members of the public from exposure to water, fish, or soil containing accelerant residues.

Risk characterization requires comparing the hazard information with the dose estimates to predict the potential for health effects to individuals under the conditions of exposure. The risk characterization also identifies uncertainties (such as data gaps where scientific studies are unavailable) that may affect the magnitude of the estimated risks.

1.4 OVERVIEW OF THE ECOLOGICAL RISK ASSESSMENT

The ecological risk assessment follows the steps of problem formulation, analysis, and risk characterization, as described in the U.S. Environmental Protection Agency's *Guidelines for Ecological Risk Assessment* (EPA 1998). This risk assessment also identifies uncertainties that are associated with the conclusions of the risk characterization. The discussion that follows briefly describes these elements. A detailed description of ecological risk assessment methodology is contained in these guidelines (EPA 1998).

In *problem formulation*, the purpose of the assessment is provided, the problem is defined, and a plan for analyzing and characterizing risk is determined. The potential stressors (in this case, accelerant residues), the ecological effects expected or observed, the receptors, and ecosystem(s) potentially affected are identified and characterized. Using this information, the three products of problem formulation are developed: (1) assessment endpoints that adequately reflect management goals and the ecosystem they represent, (2) conceptual models that describe key relationships between a stressor and assessment endpoint, and (3) an analysis plan that includes the design of the assessment, data needs, measures that will be used to evaluate risk hypotheses, and methods for conducting the analysis phase of the assessment.

Analysis is a process that examines the two primary components of risk–exposure and effects– and the relationships between each other and ecosystem characteristics. The assessment endpoints and conceptual models developed during problem formulation provide the focus and structure for the analysis. Exposure characterization describes potential or actual contact or co-occurrence of stressors with receptors, to produce a summary exposure profile that identifies the receptor, describes the exposure pathway, and describes the intensity and extent of contact or co-occurrence. Ecological effects characterization consists of evaluating ecological effects (e.g., ecotoxicity) data on the stressor of interest, as related to the assessment endpoints and the conceptual models, and preparing a stressor-response profile.

Risk characterization uses the results of the analysis phase to develop an estimate of the risks to ecological entities, describes the significance and likelihood of any predicted adverse effects, and identifies uncertainties, assumptions, and qualifiers in the risk assessment.

2.0 HUMAN HEALTH HAZARD ASSESSMENT

This section presents the results of the hazard assessment—a review of available toxicological information on the potential human health hazards associated with the accelerant residues. Section 2.1 provides background information to familiarize the reader with the terminology and technical information in this hazard assessment. Section 2.2 describes the hazard assessment methodology. Section 2.3 summarizes the toxicity data and identifies the toxicity values used in this risk assessment. Section 2.4 lists hazard assessment data gaps that affect the ability to quantify risks from these chemicals.

2.1 Background Information

Because of the obvious limitations on testing in humans, information on effects in non-human test systems usually provides the basis for an informed judgment as to whether an adverse impact is correlated with a particular exposure. These animal toxicity test results may be supplemented by information on a chemical's effects on humans, such as the results of dermatologic or exposure testing in humans, and occasional studies of low-level dosing of human volunteers by oral or other routes.

Toxicity tests in laboratory animals are designed to identify specific toxic endpoints (effects of concern), such as lethality or cancer, and the doses associated with such effects. Studies vary according to the test species used, the endpoint, test duration, route of administration, and dose levels. The dosing schedule, number of test groups, and number of animals per group also vary from one test to another, but the tests are generally designed to demonstrate whether a causal relationship exists between administered doses and any observed effects.

2.1.1 Duration of Tests

The duration of toxicity tests ranges from single-dose (acute) or short-term (subacute) tests, through longer subchronic studies, to chronic studies that may last up to the lifetime of an animal. Acute toxicity studies involve administering a chemical to each member of a test group, either in a single dose or in a series of doses over a period less than 24 hours. Subacute, subchronic, and chronic studies are used to determine the effects of multiple doses. Subacute toxicity studies involve repeated exposure to a chemical for one month or less. Subchronic toxicity studies generally last from one to three months, and chronic studies last for more than three months.

Acute studies are used primarily to determine doses that are immediately lethal, which results in limited utility in an assessment of long-term or repeated low-level human exposures. Acute and subacute toxicity studies include dermal irritation tests, dermal sensitization tests, eye irritation tests, and inhalation exposure or daily oral dosing of laboratory animals for up to one month to further define effects from limited exposures.

Longer term studies are designed to characterize the dose-response relationship resulting from repeated exposure to a compound. All other things being equal, the greater the duration of the study, the more reliable will be the resulting value for estimating the effects of subchronic or

chronic exposures in humans. Adverse effects in laboratory tests may include overt clinical signs of toxicity, reduced food consumption, abnormal body weight change, abnormal clinical hematology or chemistry, or visible or microscopic abnormalities in the tissue of the test organism. Chronic studies in rats or mice that continue for longer periods of time, usually about two years, may also be used to assess the carcinogenic potential of a chemical.

2.1.2 Routes of Exposure

For assessing hazards from the accelerant residues, the routes of administration in laboratory tests that reflect the likely types of exposures to humans are oral by dietary (in food or water) or gavage (forced into the stomach through tubing). Selection of the route of administration of a particular test material is based on the probable route of human exposure.

2.1.3 Units

A dose is expressed as milligrams of a chemical per kilogram of body weight of the test animal (mg/kg), in parts per million (ppm) in the animal's diet, or in milligrams per liter in the water that it drinks. In chronic studies, the test substance is generally administered in the diet at specified amounts in parts per million (mg of chemical per kg of food). The known weight of the animal over the test period and its food intake rate are used to convert parts per million in the diet to milligrams of a chemical per kilogram of body weight per day (mg/kg/day) for extrapolation to humans. In most chronic toxicity studies, at least two dosing levels are used, in addition to a zero-dose, or control group. In general, the control group receives only the vehicle (for example, water or saline) used in administering the test material. In a dietary study, the animal's feed would serve as the vehicle.

2.1.4 Toxicity Endpoints

In acute toxicity studies, the endpoint of interest is often the median lethal dose (LD_{50}), which is the single dose that is calculated to be lethal to 50 percent of the test animals.

For examination of non-lethal, noncarcinogenic endpoints, toxicity testing can be used to estimate threshold exposure levels. The threshold level is the dose level at which a significant proportion of the test animals first exhibit the toxic effect. The threshold dose will vary among tested species and among individuals within species. Examples of toxic effects include pathologic injury to body tissue; a body dysfunction, such as respiratory failure; or another toxic endpoint, such as developmental defects in an embryo. It is not possible to determine threshold dose levels precisely; however, the no-observed-adverse-effect level (NOAEL) indicates the dose at which there is no statistically or biologically significant increase in the frequency or severity of an adverse effect in individuals in an exposed group, when compared with individuals in an appropriate control group. The next higher dose level in the study is the lowest-observed-adverse-effect level (LOAEL), at which adverse effects are observed. The true threshold dose level for the particular animal species in a study lies between the NOAEL and the LOAEL. If a chemical produces effects at the lowest dose tested in a study, the NOAEL must be at some lower dose. If the chemical produces no effects, even at the highest dose tested, the NOAEL is equal to or greater than the highest dose.

Carcinogenicity studies are used to determine the potential for a compound to cause malignant (cancerous) or benign (noncancerous) tumors when administered over an animal's lifetime. Several dose levels are used, with the highest set at the maximum tolerated dose, as established from preliminary studies. A control group is administered the vehicle (the liquid or food with which the test chemical is given) alone. Because tumors may arise in test animals for reasons unrelated to administration of the test compound, statistical analyses are applied to the tumor incidence results to determine the significance of observed results. Amdur et al. (1991) listed four types of responses that have generally been accepted as evidence of compound-induced tumors:

- The presence of types of tumors not seen in controls
- An increase in the incidence of the tumor types occurring in controls
- The development of tumors earlier than in controls
- An increased multiplicity of tumors

Some chemicals that elicit one or more of these responses may not be primary carcinogens (that is, tumor-inducers on their own), but may be enhancers or promoters. However, a carcinogenicity evaluation remains appropriate, because they may contribute to an increase in cancer incidence.

In a carcinogenicity assay, the dose-specific tumor incidence data are used to calculate a cancer slope factor, which represents the probability that a 1-mg/kg/day chronic dose of the agent will result in formation of a tumor, and is expressed as a probability, in units of "per mg/kg/day" or $(mg/kg/day)^{-1}$.

2.2 Hazard Assessment Methodology

The goal of the hazard analysis is to determine toxicity levels for quantification of risk. There are two types of toxicity endpoints: noncarcinogenic effects and carcinogenic effects.

For noncarcinogenic effects, it is generally assumed that there is a threshold level, and that doses lower than this threshold can be tolerated with little potential for adverse health effects. The U.S. EPA has determined threshold doses for many chemicals, and refers to these as reference doses (RfDs). The RfD is an estimate of the highest possible daily dose of a chemical that will pose no appreciable risk of deleterious effects to a human during his or her lifetime. The uncertainty of the estimate usually spans about one order of magnitude. The RfD is calculated using the lowest NOAEL from the species and study most relevant to humans, or the most sensitive species (the species that exhibited the lowest NOAEL overall). This NOAEL is divided by an uncertainty factor (usually 100) consisting of a factor of 10 to allow for the variation of response within the human population and a factor of 10 to allow for extrapolation to humans. Additional uncertainty factors may be applied to account for extrapolation from a shorter term study, overall inadequacy of data, or failure to determine a no-effect level. RfDs are expressed in units of mg/kg/day. EPA lists RfDs in its Integrated Risk Information System, a chemical risk database (EPA 2002a). RfDs can also be calculated using EPA's methodology. RfDs are

analagous to the acceptable daily intake levels identified by groups such as the World Health Organization.

For compounds that are known, probable, or possible human carcinogens, cancer slope factors that have been calculated by EPA or other appropriate sources are identified for use in this risk assessment.

2.3 Toxicity Data

2.3.1 Aluminum and Aluminum Oxide

Free aluminum (Al) is reactive. Following combustion, aluminum oxide (Al_2O_3) will be the dominant form in the environment. ATSDR (1999a) estimated a minimal risk level of 2.0 mg Al /kg/day for intermediate (15 to 364 days) oral exposure, based on the most sensitive toxicity endpoint (neurotoxicity) identified in studies in laboratory animals. This minimal risk level is equivalent to 3.78 mg Al_2O_3/kg/day. Long-term feeding studies in mice and rats using aluminum potassium sulfate or aluminum phosphide led reviewers to conclude that aluminum has not demonstrated carcinogenicity in laboratory animals (ATSDR 1999a).

2.3.2 Calcium Sulfate

Calcium sulfate is widely present in the environment as gypsum; it is also known as plaster of Paris. The U.S. Food and Drug Administration stated that calcium sulfate is "generally recognized as safe", or GRAS, when directly added to human food (21 CFR 184.1230). No specific acceptable ingestion intake level or limit was identified for calcium sulfate.

2.3.3 Copper Oxide

Copper oxide is a relatively insoluble form of the metal copper, which is found naturally in the environmental and is a necessary component of the human diet. Excessive intake of copper can cause dizziness, headaches, diarrhea, and liver and kidney damage (ATSDR 1999b). The mean dietary intake of copper in adults ranges from 0.9 to 2.2 mg (HSDB 2002). This corresponds to levels of 0.016 to 0.038 mg CuO/kg/day for an average-weight adult. EPA (40 CFR 141.51) has set a maximum contaminant level goal (MCLG) of 1.3 mg/L for copper in drinking water, which corresponds to 1.6 mg CuO/L. An oral RfD for CuO of 0.034 mg/kg/day was calculated, based on the MCLG.

No tumors were reported in rats injected with copper oxide; however, EPA has not classified copper as to its carcinogenic potential (EPA 1991a).

2.3.4 Diesel Fuel

Diesel fuel is a complex and variable mixture of petroleum hydrocarbons. It has an oral LD_{50} in rats of 7,400 mg/kg (API 1980a, as cited in CONCAWE 1996). No subchronic or chronic oral toxicity data are available. Therefore, an RfD was calculated based on a study by Layton et al. (1987) in which data for chemicals having known RfDs and LD_{50}s were compared. A

conversion factor of 0.00005 was used to generate an RfD that corresponds to subchronic exposure. The conversion factor represents the median value of the reported ratios of reference doses to LD_{50} values. The estimated RfD is 0.37 mg/kg/day.

Although diesel fuel is not classifiable as to its carcinogenicity in humans according to IARC (1989), it contains many polycyclic aromatic hydrocarbons, some of which have been associated with cancer in laboratory animals (NPS 1997).

2.3.5 Gasoline

Like diesel fuel, gasoline is a petroleum hydrocarbon mixture. No NOAEL was identified for gasoline. The lowest LOAEL for toxicity endpoints relevant to human toxicity (body weight, gastrointestinal effects) was 2,000 mg/kg in a 28-day study in rats (ATSDR 1995). Based on this LOAEL, an RfD of 0.6 mg/kg/day was estimated, incorporating uncertainty factors of 0.1 to account for lack of a NOAEL, 0.3 for the absence of a longer-term study, and 0.01 for extrapolation from laboratory animals to humans and inter-individual variation among humans.

No studies were located regarding cancer in humans or animals after oral exposure to gasoline. However, gasoline is considered to be carcinogenic, since it contains benzene, a known human carcinogen (EPA 2000b). Two percent benzene content is representative of unleaded gasoline (Caprino and Togna 1998). Using the upper end of the oral cancer slope factor range for benzene of 0.015 to 0.055 per mg/kg/day, a cancer slope factor of 0.0011 was estimated for unleaded gasoline, based on its benzene content.

2.3.6 Iron Oxide

Iron oxide is the chemical name for the substance commonly observed as rust on items made of iron. It is regulated by the Food and Drug Administration for use as a food coloring and in food packaging, and is generally recognized as safe (21 CFR 186).

Iron is a required nutrient. The current recommended daily allowance (RDA) for iron varies by age, gender, and, for females, whether they are pregnant or breastfeeding. For children ages four to eight years old, the RDA is 10 mg/day. For adults, the RDA ranges up to 27 mg/day for pregnant women. Upper limits representing levels that are likely to pose no adverse effects are 40 mg/day for children through 13 years old, and 45 mg/day for adults (IOM 2002). These upper limits correspond to 1.8 mg/kg/day for a 6-year-old 22.6-kg child, and 0.63 mg/kg/day for an average 71.8-kg adult.

Severe toxicity may result in children following ingestion of more than 0.5 g of iron. In adults, chronic excessive ingestion may lead to toxicity, manifested by hemosiderosis, disturbances in liver function, diabetes mellitus, and possible endocrine disturbances and cardiovascular effects (Amdur et al. 1991). It is not classifiable as to its carcinogenicity in humans (IARC 1987).

EPA has established a secondary drinking water regulation of 0.3 mg/L for iron, based on aesthetic endpoints (EPA 1992).

2.3.7 Lead

EPA's reference dose workgroup concluded it was inappropriate to develop an RfD for lead because some of lead's adverse effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold (EPA 1993). Lead is a probable human carcinogen, but a quantitative estimate of risk is not appropriate given current data (EPA 1993).

EPA has recommended a screening level of 400 parts per million (ppm) lead in soil for residential land uses (the most restrictive category of exposure) (EPA 1994). This value was used in the risk assessment to assess soil exposures to residual lead. EPA's maximum contaminant level for lead in drinking water is zero; if more than 10% of tap water samples exceed an action level of 0.015 mg/L, the water system must take additional steps, including corrosion control treatment, source water treatment, lead service line replacement, and public education (40 CFR 141.80). This level is used in this risk assessment to identify risks from drinking water.

2.3.8 Manganese Dioxide

The mean manganese intake in the U.S. from foodstuffs is estimated to be about 1.5 mg/child/day for a two-year-old child, and ranges from 2 to 9 mg/person/day for adults (HSDB 2002).

ATSDR (2000) adopted the National Research Council's upper range of the estimated safe and adequate daily dietary intake of 5 mg/day as a provisional guidance value for oral exposure to manganese; this is equivalent to 0.07 mg/kg/day. EPA (1996a) has set an oral reference dose of 0.14 mg/kg/day for manganese intake; this is equivalent to 0.22 mg manganese dioxide/kg/day. It is not classifiable as to its carcinogenicity in humans (EPA 1996a).

2.3.9 MTBE

ATSDR (1996) derived an intermediate-duration minimal risk level of 0.3 mg/kg/day, based on a 90-day study in rats in which decreased blood urea nitrogen levels were observed at the lowest dose tested of 100 mg/kg/day. This value is used as the RfD for MTBE in this risk assessment.

MTBE is considered a possible human carcinogen at high doses, with a cancer slope factor of 0.004 per mg/kg/day calculated for the oral route of exposure based on a study in rats, in which females exhibited a dose-related increase in lymphoma and leukemia, and male rats developed testicular tumors at the highest dose level (EPA 1997). However, EPA does not recommend calculating cancer risks for low doses to MTBE, due to uncertainties regarding this study's dosing method, possible effects of the vehicle used, and lack of histopathological diagnoses and individual animal data.

2.3.10 Polystyrene

Polystyrene is formed by the polymerization of styrene to form a rigid, odorless, tasteless plastic. It is widely used in consumer products, including video and audio cassettes, cosmetic containers, toys, computer housings, and packaging and insulating materials for food, including the air-blown form of polystyrene known as Styrofoam® (EPA 1995).

Monte (1983) concluded that polystyrene was not absorbed when administered orally to laboratory rats. IARC (1979) reported that implantation of polystyrene materials under the skin in rats caused sarcomas. No quantitative toxicity data were available for the routes of exposure evaluated in this risk assessment; therefore, risk from polystyrene could not be quantified.

2.3.11 Potassium Chloride and Potassium Hydroxide

Potassium chloride is a commercial dietary salt substitute (HSDB 2002). The oral toxic dose ranges from 200 to 1,000 mg/kg, depending on kidney efficiency (HSDB 2002). Potassium chloride dissolves to potassium and chloride ions. Potassium compounds are ubiquitous in the earth's crust, and the element is naturally found in the human bloodstream. Acute oral potassium poisoning is rare, since large doses usually induce vomiting (HSDB 2002). EPA has set a secondary (non-enforceable) drinking water standard for chloride of 250 mg/L.

Potassium hydroxide is one of the strongest alkalies—it is extremely corrosive. Swallowing caustic alkalies causes immediate burning pain in the mouth, throat, and stomach, and the lining membranes become swollen and detached. However, it is readily soluble in water, producing potassium and hydroxide ions. A significant amount of hydroxide ions in a water body *could* change the pH of the water, but that would not be the case from the amounts potentially present as a result of potassium hydroxide formation from use of a fire accelerant.

Since only very small amounts of the precursors to these chemicals would be present in fire accelerants that are widely dispersed over the terrain (fusees—6% potassium perchlorate; ping-pong balls—3 g potassium permanganate; and launcher pistol flares—0.1% potassium perchlorate), and the toxicity review does not associate health concerns with environmentally mediated contact with these residues and their dissolution products, these compounds are not further analyzed in the quantitative risk assessment.

2.3.12 Silicon Dioxide

Silicon dioxide occurs naturally as sand and quartz. It is chemically and biologically inert when ingested (HSDB 2002). No signs of toxicity or histologic changes were observed in dogs or rats that were fed 800 mg silicon/kg/day as the dioxide, equivalent to 1,700 mg silicon dioxide/kg/day (HSDB 2002). It is approved for use in food products at levels up to 2% when used for specific purposes (21 CFR 172.480). Crystalline silica is carcinogenic by the inhalation route of exposure (IARC 1997).

Since any silicon dioxide formed as a result of the combustion of the accelerants would be indistinguishable from sand in the soils on which it forms, it is not appropriate to analyze this compound further in this risk assessment.

2.3.13 Strontium Oxide, Strontium Sulfate, and Strontium Sulfide

The strontium ion has a low order of toxicity. It is chemically and biologically similar to calcium. The oxides are moderately caustic materials (Lewis 1994a). The human daily intake of strontium has been determined to be 2 mg (HSDB 2002). EPA (1996b) set an oral RfD of 0.6 mg/kg/day for strontium. There are no data on its potential for carcinogenicity.

3.0 HUMAN HEALTH EXPOSURE ASSESSMENT

3.1 Introduction

This section describes the pathways by which human populations could be potentially exposed to fire accelerant residues. The possible routes of exposure were identified as follows:

- drinking water
- ingestion of fish
- incidental soil ingestion

In this analysis, it was assumed that an adult weighs 71.8 kg (158 lb) and a six-year old child weighs 22.6 kg (49.8 lb) (EPA 1999a).

3.2 Exposure and Dose

Two primary conditions are necessary for a human to receive a chemical dose that may result in a toxic effect. First, the chemical must be present in the person's immediate environment—such as in food or water—so that it is available for intake. The amount of the chemical present in the person's immediate environment is the exposure level. Second, the chemical must enter the person's body by some route. Chemicals in food or water may be ingested. The amount of a chemical that moves into the body by an exposure route constitutes the dose. Exposure, then, is the amount of a chemical available for intake into the body; dose is the amount of the substance that actually enters the body.

3.3 Potential Exposures

This subsection describes the representative human health exposure scenarios analyzed in this risk assessment.

3.3.1 Ingestion of Surface Water

This scenario investigates the risk from drinking water contaminated by accelerant residues on soil that dissolve or are eroded in runoff to a stream or river. For this scenario, it was assumed that a 71.8-kg adult drinks 1.51 L (0.4 gal) of water per day, and a six-year-old 22.6-kg child drinks 0.74 L (0.2 gal) per day, based on statistics presented in EPA (1999a). The following equation was used to calculate the dose to adults and children:

$$DOSE = CONC \times AMT / BW$$

where:

DOSE = dose from drinking contaminated water (mg/kg)
CONC = concentration of chemical in water (mg/L)
AMT = water consumption amount (L)
BW = body weight (kg)

Since estimation of residue concentrations in groundwater would require knowledge of site specific soil, climate, and hydrology, this route of exposure was not analyzed quantitatively in this assessment. However, it is expected that the risks would be less than those estimated for drinking surface water from the small stream, given the simplified, conservative approach used in this scenario.

3.3.2 Ingestion of Fish

In this scenario, it was assumed that an adult or child ingests fish caught in a stream or river after it receives storm water containing runoff from an area where accelerant residues are present. It was assumed that 0.113 kg of fish per day is ingested by an adult and 0.072 kg/day by a six-year-old child (EPA 1999a). This dose was calculated using the following equation:

$$DOSE = CONC \times BCF \times AMT / BW$$

where:

DOSE = dose from ingesting fish (mg/kg)
CONC = concentration of chemical in river (mg/L)
BCF = bioconcentration factor (mg/kg per mg/L)
AMT = fish consumption amount (kg)
BW = body weight (kg)

3.3.3 Incidental Soil Ingestion

In this scenario, it was assumed that an adult or child ingests soil incidental to recreational or occupational activities undertaken on areas where accelerant residues may be present in soil. It was assumed that a child ingests 400 mg/day, the upper percentile recommended by EPA (1999a). It was assumed that an adult conducting outdoor work ingests 480 mg/day, as reported in EPA (1999a). Further, it was assumed that at most 1% of an individual's daily soil contact is in the specific location where the accelerant residue remains. This dose was calculated using the following equation:

$$DOSE = RATE \times CONC \times FRAC \times CF / BW$$

where:

DOSE = dose from incidental soil ingestion (mg/kg)
RATE = soil ingestion rate (mg/day)
CONC = estimated concentration of chemical in soil (ppm, equal to mg/kg)
FRAC = fraction of soil exposure in area with accelerant residue (unitless)
CF = conversion factor (kg soil / $1x10^6$ mg soil)
BW = body weight (kg)

3.3.4 Lifetime Doses

Lifetime doses were calculated for the potential carcinogens evaluated in this risk assessment: the benzene component of gasoline and the gasoline additive MTBE. The lifetime dose was estimated by assuming that an individual has exposure from three pathways (drinking water, consuming fish, incidental soil ingestion) for a one-week period. The estimated total dose from these exposures was averaged over a typical 75-year lifetime (EPA 1999a).

3.4 Estimated Environmental Concentrations

To estimate doses using the quantitative approaches listed in the previous section, it is necessary to predict the environmental concentrations of the accelerant residues. Since the amount per area (for example, gallons per acre or mg per square meter) of accelerant used to ignite a prescribed burn will vary based on the site-specific requirements, environmental concentrations were estimated based on a per-unit basis for the various accelerants. That is, concentrations of accelerant residues in two representative environments were estimated for the following amounts of accelerant:

- Fusee: one 0.52-lb fusee
- Drip torch: one gallon of 1:3 gasoline:diesel fuel mix
- Helitorch, terra-torch, or hand-thrown gelled gasoline: one gallon of gelled gasoline
- Ping-pong balls: one ball
- Flares from launcher pistol: one flare
- Propane-fueled wand: no residues (or associated risks) are expected

Environmental characteristics of ecosystems where prescribed burns may occur can vary widely. Some of the potential areas of variation, and the corresponding effect on potential exposures, are as follows:

- *Soil type*. Some soils, such as clays or soils high in organic matter, are less permeable to water drainage through the soil column or tend to bind dissolved chemicals to the soil particles. Other soils, such as those with a higher sand content or lower organic matter, could have the opposite tendency. In addition to these examples, many additional soil characteristics factor into runoff or leaching potential.

- *Slope.* On relatively flat terrain, precipitation has a longer time frame in which to pool and potentially permeate the soil and enter groundwater, whereas areas with increasing slopes will be associated with a greater potential for runoff to surface water.

- *Precipitation.* If rain or snow (and subsequent snowmelt) occurs, accelerant residues could be mobilized from their location on the soils where they were applied, leaching to groundwater or running off to surface water. If little or no precipitation is present, the chemicals may persist longer in the soil, subject to transformations by sunlight or microorganisms in the soil.

- *Revegetation.* Once an area where accelerants were applied is covered with new vegetation, the soil itself (and any remaining accelerant residues) will be more shielded from direct contact by humans using the area.

- *Surface water.* The type of surface water present in an area, if any, can vary greatly. Some streams and ponds may be present only at certain times of year or after significant rainfall events. Others may be continually present. Larger lakes and rivers can contain volumes of water that would so dilute any accelerant residues entering them as to make the water concentration non-detectable. Lakes that are slowly recharged or slow-flowing rivers and streams may allow chemical concentrations to persist locally within them for longer periods of time, whereas fast-flowing, turbulent rivers would more quickly mix and dilute any entering chemical.

- *Watershed.* The size of a watershed will be associated with the amount of noncontaminated runoff potentially entering a river or stream, to further dilute any accelerant residue in the runoff itself and in the stream. In addition, vegetated areas between the location of residues and surface water can act as buffers, slowing or stopping overland transport.

These characteristics provide just a few examples of the complexities in estimating exposures from runoff or leaching of a chemical when no specific location in which the exposure occurs has been identified. To offer a wide range of applicability for the results of this risk assessment, the following approach was taken to estimated the potential exposures:

- Two representative watersheds were assumed. One is a small, one-acre drainage area with a perennial stream that has an average flow rate of 12 cubic feet per second (cfs); and the other is a 100-acre basin with a river that flows at 350 cfs. Ingestion of fish and drinking surface water were evaluated for each chemical in both watersheds, assuming that the residue enters the water either dissolved in runoff or sorbed to eroded soil over a 15-minute period following a 0.5-inch rainfall event. For gasoline and diesel fuel, for which the primary chemical components contributing toxicity to the fuels are also significantly volatile and would largely evaporate during the burn, it was assumed that 1% of the applied volume remains as a residue.

- In the soil ingestion scenario, it is assumed that no precipitation or other weathering occurs, allowing all of the residue to remain in the top two cm of soil, the evaluation depth recommended by EPA (1996c). It was further assumed that physical disturbance and wind distributed the residue from a single fusee, flare, or ping-pong ball, or 0.1 gallon of liquid or gelled fuel, over a one-square-meter area.

Table 3-1 lists the estimated soil and water concentrations for the fire accelerants and their associated residues.

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4.0 HUMAN HEALTH RISK CHARACTERIZATION

4.1 Introduction

This section characterizes the estimated risks to human health that may result from accelerant residues. In the risk characterization, the human doses estimated in the exposure assessment (Section 3.0) are compared with the toxicity characteristics described in the hazard assessment (Section 2.0), to arrive at estimates of risk.

Section 4.2 describes the methods used to evaluate human health risks, including both noncarcinogenic and carcinogenic risks. Section 4.3 contains the results of the quantitative risk characterization for the accelerant residues. Section 4.4 discusses the uncertainties in this risk assessment.

4.2 Methodology for Assessing Risks

Several of the fire accelerants may leave residues that are mixtures of several chemicals. Risks from these mixtures were evaluated following the recommendations of EPA (2000c): *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures*. Specifically, the following approaches were applied:

- EPA states that "whenever possible, the preferred approach to the health risk evaluation of chemical mixtures is to perform the assessment using health effects and exposure data on the whole mixture." In the case of gasoline and diesel fuel, this approach was taken, since toxicity data on the mixture were available.

- EPA also stated that "even if a risk assessment can be made using whole-mixture data, it may be desirable to also conduct a risk assessment based on toxicity data on the components in the mixture . . . When a mixture contains component chemicals whose critical effects are of major concern, e.g., cancer or developmental toxicity, an approach based on the mixture data alone may not be sufficiently protective in all cases." This analytical approach was applied for the multiple-chemical residues from flares, and from gasoline plus its additive MTBE.

The assessment of risks for the residues and residue mixtures was conducted following the standard risk assessment methodology described in NRC (1983) and EPA (1989), summarized in the following paragraphs.

4.2.1 Noncarcinogenic Risk Estimation

In this risk assessment, the potential risks were evaluated by comparing the representative doses (estimated in the exposure assessment) with the RfDs (identified in the hazard assessment). All the RfDs used in this risk analysis take into account the possibility of multiple exposures and represent acceptable dose levels. The comparison of dose to RfD consists of a simple ratio, called the Hazard Quotient:

$$Hazard\ Quotient = \frac{Estimated\ Dose\ (mg / kg / day)}{RfD\ (mg / kg / day)}$$

If the estimated dose does not exceed the RfD, the hazard quotient will be one or less, indicating a negligible risk of noncarcinogenic human health effects. It is important to note two characteristics of the hazard quotient: (1) the greater the value of the hazard above one, the greater the level of concern; but (2) the level of concern does not increase linearly as the hazard quotient increases, because RfDs do not have equal accuracy or precision and are not based on the same severity of toxic effects. Thus, the interpretation of the potential toxic response associated with a particular hazard quotient can range widely depending on the chemical (EPA 1989).

A dose estimate that exceeds the RfD, although not necessarily leading to the conclusion that there will be toxic effects, clearly indicates a potential risk for adverse health effects. Risk is presumed to exist if the hazard quotient is greater than one. However, comparing doses from short-term exposures (such as those from fire accelerant residues) to RfDs that are designed to represent long-term exposures with repeated daily doses tends to exaggerate the risk from those limited events.

Following the guidance presented in EPA (2000c), the additive approach was used to sum the hazard quotients when more than one residue chemical from a particular accelerant was quantified in the analysis. In these cases, a hazard index for the residue mixture, representing the sum of the hazard quotients, was calculated. The hazard index is interpreted in the same manner as the hazard quotient; that is, risk is presumed to exist if the accelerant hazard index exceeds one.

4.2.2 Cancer Risk Estimation

As a result of the review of cancer studies presented in the Human Health Hazard Assessment (Section 2.0), a risk analysis for cancer was two components of the gasoline mixture: benzene, and the additive MTBE. Although lead is a potential accelerant residue that is a probable carcinogen, no quantitative cancer slope factor is available on which to base a cancer risk estimate.

The mechanism for cancer dose-response can be complex, and EPA is currently developing updated guidance for deriving cancer slope factors that are applicable to human health risk assessment from the results of studies in laboratory animals. In laboratory studies, high doses are used to elicit an observable cancer incidence in a finite group of test animals. Historically, carcinogenic effects were assumed to have no threshold, requiring extrapolation to compare exposures from the much lower doses associated with environmental exposure to chemicals. EPA's current guidance in force, the 1986 *Guidelines for Carcinogen Risk Assessmen*t, provided a basic rationale for linear dose-response assumptions in cancer risk assessment (EPA 1986). However, new perspectives on methods to assess risks of cancer are gaining wider acceptance, such as consideration of mode of action, thresholds for carcinogenicity, and incorporating other types of biological data. In 1996, EPA proposed revised guidelines for carcinogen risk assessment which address these (and other) issues, but they have not yet been finalized.

Estimation of cancer slope factors using updated methods is occurring on a chemical-by-chemical basis, as new laboratory studies are completed and new risk assessments are conducted. For the chemicals identified as known or possible human carcinogens in this risk assessment, a linear (no-threshold) approach was used in calculating the cancer slope factors, in accordance with the guidance that has been in effect.

Cancer risk from a chemical is expressed as the probability that cancer will occur over the course of a person's lifetime, as a result of the stated exposure. This risk probability is calculated as follows:

$$RISK = DOSE \times CSF \times OCC / LIFE$$

where:

RISK	=	the lifetime probability of cancer as a result of the specified exposure
DOSE	=	estimated dose (mg/kg/day)
CSF	=	cancer slope factor (per mg/kg/day)
OCC	=	number of occurrences of the daily dose during an individual's lifetime
LIFE	=	the number of days in a 75-year lifetime (27,375 days)

The resulting cancer probability is compared to a benchmark value of $1x10^{-6}$ (or 1 in 1 million), a value commonly accepted in the scientific community as representing a cancer risk that would result in a negligible addition to the background cancer risk of approximately one in four in the United States. In some occupational health risk assessments, cancer risks as high as $1x10^{-4}$ (1 in 10,000) can be considered acceptable. However, the benchmark of 1 in 1 million is used in this risk assessment.

4.3 Estimated Risks from Accelerant Residues

Tables 4-1 and 4-2 present the residues' estimated human health risks to adults and children, respectively, from the residue of one "unit" of fire accelerant.

The risk tables in this section use scientific notation, since many of the values are very small. For example, the notation 3.63E-001 represents 3.63×10^{-1}, or 0.363. Similarly, 4.65E-009 represents 4.65×10^{-9}, or 0.00000000465.

All accelerant residues resulted in hazard quotients less than one and cancer risks less than 1 in 1 million, indicating negligible risk from each unit of accelerant used to ignite prescribed burns.

To develop this information into a form that will be useful for planners, decisionmakers, and those who may conduct site-specific environmental impact assessments, Table 4-3 displays the maximum number of units per watershed (small or large) that would be associated with a conclusion of "negligible risk," based on the methodology of this risk assessment.

Table 4-1. Unit Risks to Human Health from Accelerant Residues—Adults

Accelerant	Residue	Incidental Soil Ingestion	Small Stream		River	
			Drinking Water	Eating Fish	Drinking Water	Eating Fish
		--Hazard Index--				
Fusee	Strontium compounds	2.24E-03	2.37E-03	1.88E-01	6.02E-05	4.78E-03
Gasoline	Gasoline	7.47E-05	2.37E-03	ND**	6.02E-05	ND**
	MTBE	2.64E-03	8.36E-02	1.07E-02	2.12E-03	2.72E-04
	Additive Hazard Quotient	*2.71E-03*	*8.59E-02*	*1.07E-02*	*2.18E-03*	*2.72E-04*
Diesel fuel	Diesel fuel	1.71E-04	5.42E-03	ND**	1.38E-04	ND**
Gasoline + diesel fuel (Driptorch 1:3 mix)	Gasoline, MTBE, diesel fuel	8.06E-04	2.56E-02	ND**	6.49E-04	ND**
Gelling agent	Aluminum oxide	1.79E-06	5.67E-05	1.45E-03	1.44E-06	3.69E-05
Gelled gasoline	Gasoline, MTBE, aluminum oxide	2.71E-03	8.60E-02	1.22E-02	2.19E-03	3.09E-04
Ping-pong balls	Manganese dioxide	1.39E-04	4.42E-04	1.89E-02	1.12E-05	4.79E-04
Launcher pistol flares	Aluminum oxide	3.95E-04	1.25E-03	3.21E-02	3.18E-05	8.15E-04
	Calcium sulfate	ND†	ND†	ND†	ND†	ND†
	Iron oxide	8.77E-05	2.78E-04	2.38E-05	7.07E-06	6.04E-07
	Copper oxide	2.32E-03	7.36E-03	6.29E-02	1.87E-04	1.60E-03
	Lead*	2.74E-03	7.37E-03	ND‡	1.87E-04	ND‡
	Additive Hazard Quotient	*5.55E-03*	*1.63E-02*	*9.50E-02*	*4.13E-04*	*2.41E-03*
Propane	-none-					
		--Cancer Risk--				
Gasoline	Gasoline	1.26E-11	4.00E-10	ND**	1.02E-11	ND**
	MTBE	8.09E-10	2.56E-08	3.29E-09	6.52E-10	8.35E-11
	Total Cancer Risk	*8.21E-10*	*2.60E-08*	*3.29E-09*	*6.62E-10*	*8.35E-11*

*Hazard Index calculated as soil or water concentration relative to criteria listed in Section 2.3.7.

**No BCF was available for these chemical mixtures.

†No reference dose was found for calcium sulfate. However, the substance is "generally regarded as safe" in small amounts in food.

‡No reference dose or medium-specific criterions was identified for lead in ingested fish.

Table 4-2. Unit Risks to Human Health from Accelerant Residues—Children

Accelerant	Residue	Incidental Soil Ingestion	Small Stream		River	
			Drinking Water	Eating Fish	Drinking Water	Eating Fish
		--Hazard Index--				
Fusee	Strontium compounds	5.93E-03	3.69E-03	3.34E-01	9.37E-05	8.48E-03
Gasoline	Gasoline	1.98E-04	3.69E-03	ND**	9.37E-05	ND**
	MTBE	6.98E-03	1.30E-01	1.90E-02	3.31E-03	4.83E-04
	Additive Hazard Quotient	*7.18E-03*	*1.34E-01*	*1.90E-02*	*3.40E-03*	*4.83E-04*
Diesel fuel	Diesel fuel	4.53E-04	8.44E-03	ND**	2.14E-04	ND**
Gasoline + diesel fuel (Driptorch 1:3 mix)	Gasoline, MTBE, diesel fuel	2.13E-03	3.98E-02	ND**	1.01E-03	ND**
Gelling agent	Aluminum oxide	4.74E-06	8.83E-05	2.58E-03	2.24E-06	6.55E-05
Gelled gasoline	Gasoline, MTBE, aluminum oxide	7.18E-03	1.34E-01	2.16E-02	3.40E-03	5.48E-04
Ping-pong balls	Manganese dioxide	3.69E-04	6.88E-04	3.35E-02	1.75E-05	8.50E-04
Launcher pistol flares	Aluminum oxide	1.04E-03	1.95E-03	5.69E-02	4.95E-05	1.45E-03
	Calcium sulfate	ND†	ND†	ND†	ND†	ND†
	Iron oxide	2.32E-04	4.33E-04	4.21E-05	1.10E-05	1.07E-06
	Copper oxide	6.15E-03	1.15E-02	1.12E-01	2.91E-04	2.83E-03
	Lead*	2.74E-03	7.37E-03	ND‡	1.87E-04	ND‡
	Additive Hazard Quotient	*1.02E-02*	*2.12E-02*	*1.68E-01*	*5.39E-04*	*4.28E-03*
Propane	-none-					
		--Cancer Risk--				
Gasoline	Gasoline	3.34E-11	6.22E-10	ND**	1.58E-11	ND**
	MTBE	2.14E-09	3.99E-08	5.83E-09	1.01E-09	1.48E-10
	Total Cancer Risk	*2.17E-09*	*4.06E-08*	*5.83E-09*	*1.03E-09*	*1.48E-10*

*Hazard Index calculated as soil or water concentration relative to criteria listed in Section 2.3.7.

**No BCF was available for these chemical mixtures.

†No reference dose was found for calcium sulfate. However, the substance is "generally regarded as safe" in small amounts in food.

‡No reference dose or medium-specific criterions was identified for lead in ingested fish.

4.4 Discussion and Uncertainties

On a per-unit basis in the representative small and large watersheds, and from incidental soil ingestion, no health risks were predicted from potential residues remaining after the use of accelerants to ignite a prescribed burn. Table 4-3 provides estimates of the maximum amount of each accelerant that could be used and still be associated with an expectation of no risks to the health of adults or children. It is important to note that if a prescribed burn was conducted in a dry section of a watershed, the drinking water and fish consumption pathways would not exist, resulting in hazard indices and cancer risks of zero from these exposure routes.

In a supplemental information report prepared by the Forest Service, ranges of estimates were included of the quantity used of several ignition sources for a proposed prescribed burn. These estimates are provided in Table 4-4, along with a comparison to the risk threshold values summarized in Table 4-3.

Risks from dermal exposure to the accelerant residuals was not quantified; however, based on the conclusions of the soil ingestion scenario, in which residues are transferred to the mouth via soil contamination of the hands, no risks would be expected from these levels of exposure. The skin is less permeable to chemical substances than are mucous membranes such as those lining the gastrointestinal system; therefore, dermal absorption would be expected to be minimal compared to the estimated oral doses.

Risks from inhalation exposures were outside the scope of this assessment, requiring a complex analysis of simultaneous exposure to the products of burning vegetation to accurately depict the overall risk from inhalation at a prescribed burn. The use of the accelerant chemicals outdoors would be expected to be associated with exposures of short duration, as any inhalable gases or particles would be quickly dispersed throughout the atmosphere.

It is important to note that several conservative assumptions were made in conducting this analysis, necessitated by the generic nature of this assessment; the primary source of likely overestimation of exposure is the assumption that all of the accelerant residue is mobilized into surface water. For inorganic chemicals, the nature of the soil, its pH, oxidation-reduction environment, temperature, and presence of other chemicals can all affect the mobility of the chemical (Maidment 1993). For the organic chemicals in this analysis, the presence of moisture, sunlight, and microorganisms can contribute to their degradation on the soil surface; some of the components of the gasoline and diesel fuel chemical mixtures are also likely to volatilize during or after their application, decreasing the toxicity of the mixture as a whole by their removal. However, complete mobility and no degradation were assumed, so that the analysis would provide an upper bound on the potential exposures from any accelerant residues, and so that it would be highly unlikely that any actual risks would exceed those presented in this report.

The risks summarized in this assessment are not probabilistic estimates of risk, but are conditional estimates. That is, these risks are likely only if all exposure scenario assumptions that were described are met. The primary areas of uncertainty in this analysis include the precise chemical composition of accelerant residues; their actual environmental concentrations in soil and water; the predicted RfDs and cancer slope factors, which are based on studies in laboratory

Table 4-3. Maximum Units Associated with Negligible Human Health Risk

Accelerant	Unit	Maximum Number of Units Associated with Negligible Risk	
		Small Watershed*	Large Watershed**
Fusee	One 0.53-lb fusee	3	118
Gasoline + diesel fuel (Driptorch 1:3 mix)	One gallon	25	989
Gelled gasoline	One gallon†	7	294
Ping-pong balls	One ball	30	1176
Launcher pistol flares	One flare	6	234
Propane	Tank	NR‡	NR‡

*One acre with 12-cfs stream.

**100 acres with 350-cfs river.

†Assumed mix rate of 4 lb gelling agent/55 gallons gasoline.

‡No residues expected.

Table 4-4. Comparison of Ignition Source Quantities Used to Estimated Risks

Ignition Source	Estimated Range of Quantity Used (per acre)*	Maximum Units Associated with No Risk		Comments
		Small Watershed	Large Watershed	
Drip torch	1 - 3 gal	25	989	No risk from estimated range.
Helitorch	1 -3 gal	7	294	No risk from estimated range.
Fusee	10 - 40	3	118	Small drainage areas should be evaluated for human water use patterns (particularly potential fish consumption)
Flare	10 - 50	6	234	Small drainage areas should be evaluated for human water use patterns (particularly potential fish consumption)
Plastic sphere	10	30	1,176	No risk from estimated range.

*Source: USDA undated.

animals using standard extrapolation methods; and the quantity of a chemical to which an individual may actually be exposed, compared to the standard exposure assumptions recommended by EPA.

These uncertainties could be addressed by conducting laboratory and field studies and monitoring sites where prescribed burns have occurred. However, the use history of these chemicals and the highly site-specific and variable nature of prescribed burn characteristics would require application of a large margin of error, limiting the utility of any studies in providing greater confidence in the risk conclusions.

5.0 ECOLOGICAL PROBLEM FORMULATION

This section presents the results of the ecological risk assessment problem formulation, in which the purpose of the ecological risk assessment is provided, the problem is defined, and a plan for analyzing and characterizing risk is determined. Section 5.1, integrating available information, identifies and characterizes the stressors, the ecological effects expected or observed, the receptors, and ecosystem potentially affected. Section 5.2 describes the assessment endpoints for the ecological risk assessment. Section 5.3 presents the conceptual model describing key relationships between the stressors and assessment endpoints. Section 5.4 summarizes the analysis plan that includes the design of the assessment, data needs, measures that will be used to evaluate risk hypotheses, and methods for conducting the analysis phase of the assessment.

5.1 Integration of Available Information

In this ecological risk assessment, the potential stressors are the accelerant residues on the site of a prescribed burn. The information presented in Sections 1.1 and 1.2 of this report provides detail on the types of fire accelerants that may be used to ignite a prescribed burn, their chemical nature, and the potential residues that may be left on the soil after the project. The amount and type of accelerant used in a particular prescribed burn will vary. Therefore, risks from all accelerant methods were evaluated, and the estimated environmental concentrations of the potential residues were associated with the accelerants on a per-unit basis (Section 3.4), to facilitate the utility of the conclusions of this risk assessment.

The ecological effects that may be associated with the accelerant residues are those associated with direct toxicity to wildlife species that encounter the chemicals. The impacts of the prescribed burn itself on wildlife are not within the scope of this analysis.

The receptors in this ecological risk assessment were selected to represent a range of wildlife species. These receptors include mammals, birds, reptiles, amphibians, fish, and aquatic invertebrates. In addition, exposures to these representative species were also compared to risk criteria relevant to endangered, threatened, and sensitive species that could occupy the same ecological “niche”.

There is wide variation in the type of habitat on which accelerants could be used to ignite a prescribed burn, including grass, shrubs, and forested areas.

5.2 Assessment Endpoints

Assessment endpoints are selected based on three criteria: ecological relevance, susceptibility to stressors, and relevance to management goals (EPA 1998). For species that are endangered, threatened, or sensitive, the assessment endpoint selected is individual survival, growth, and reproduction. For non-sensitive species, the assessment endpoint selected is the survival of populations.

Scenarios describing the potential impacts of accelerant residues on the assessment endpoints are developed in the conceptual model described in the next section.

5.3 Conceptual Model

A conceptual model consists of a risk hypothesis that describes relationships between the stressor, exposure, and assessment endpoint response; and a diagram illustrating these relationships. For the accelerant residues, the risk hypothesis is as follows.

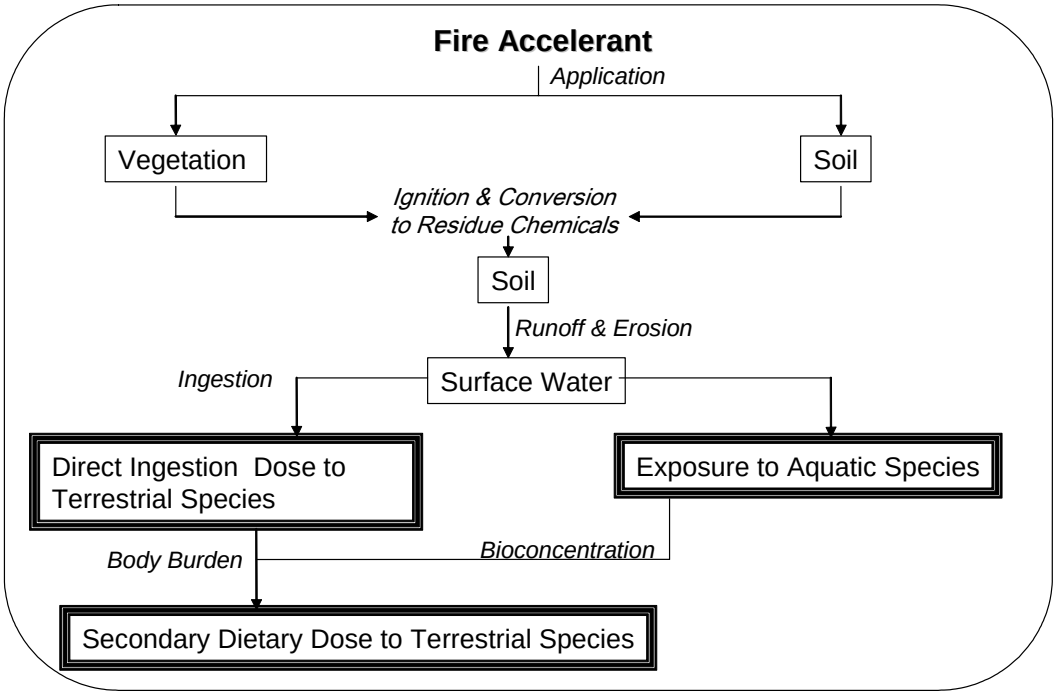
Risk Hypothesis
Residual chemicals from fire accelerants are toxic to wildlife species at various levels of exposure, based on laboratory and field tests that have characterized exposure-response relationships. The associated hypothesis is that use of accelerants to ignite prescribed burns will cause chemical toxicity from the accelerant residues, resulting in adverse effects to the individual's survival, growth, and reproduction for sensitive species, or to the survival of populations of non-sensitive species.

To test this hypothesis, a conceptual model was developed to illustrate the relationships between stressors, exposure routes, and receptors. The conceptual model is presented in Figure 5-1.

5.4 Analysis Plan

Based on the conceptual model, terrestrial and aquatic species exposure scenarios were selected to evaluate risks to ecological resources.

Figure 5-1. Conceptual Model



Representative terrestrial and aquatic species and their characteristics were identified, illustrating the various types of residue exposure for wildlife species. Using the results of the environmental fate assessment described in Section 3.4, environmental exposures were estimated, in terms of dose (mg/kg) for terrestrial species or concentration (mg/L) for aquatic species.

The toxic properties of each residual chemical to wildlife species were researched and summarized, using data available in scientific journals, reference books, and government sources. Endpoints were identified, consisting of median lethal doses (LD_{50}s) for terrestrial species and median lethal concentrations (LC_{50}s) for aquatic species.

The doses and concentrations identified in the exposure characterization were compared to the toxic properties identified in the effects characterization, using the guidelines specified by EPA for interpreting risk estimates to general wildlife and to endangered, threatened, or sensitive species.

6.0 ECOLOGICAL ANALYSIS

6.1 Data and Models for Analysis

A combination of laboratory study data, field study data, and modeling outputs were used in the ecological risk assessment.

A literature search was conducted to identify LD_{50}s (median lethal doses) and LC_{50}s (median lethal concentrations) for the accelerant residues. These values were used to represent the toxicity of the chemicals to the representative wildlife species.

The estimated environmental concentrations listed in Table 3-1 were used in the ecological risk assessment.

6.2 Characterization of Exposure

6.2.1 Terrestrial Species

The terrestrial species exposure scenario postulates that a variety of terrestrial wildlife species may be present at or near a site at which a prescribed burn has occurred. The scenario further postulates that these terrestrial species may be exposed to any accelerant residues through ingestion of surface water that has received runoff and, where appropriate, ingestion of terrestrial or aquatic species containing a body burden of accelerant residues.

The list of representative species is as follows:

Mammals
- Deer mouse (herbivore)
- Mink (carnivore/piscivore)

Birds
- Northern bobwhite (herbivore)
- Red-tailed hawk (raptor)
- Great blue heron (piscivore)

Reptiles/Amphibians
- Painted turtle (herbivore/piscivore)
- Racer (carnivore)

These particular wildlife species were selected because they represent a range of foraging habitat and diets relevant to the environmental fate of the residue chemicals, and for which parameters are generally available.

For each species, characteristics were identified that were used in estimating doses of the residue chemicals, including body weight, water intake, dietary intake, composition of diet, and home range/foraging area.

For terrestrial wildlife, exposures were assumed to occur through ingestion of food with body burden, and ingestion of water from small streams. Predators that feed on other animals were assumed to receive the total body burden that each of the prey species received. Wildlife that feed on aquatic species were assumed to receive residue levels based on the chemical concentrations in water and the associated bioconcentration factors. Chemical concentrations in drinking water sources for wildlife were assumed to be those predicted for a small stream in a one-acre watershed, as presented in Section 3.4.

To calculate doses for terrestrial wildlife, the doses from the exposure routes described in the preceding paragraph were summed, as follows:

$$DOSE = \left[\left(FRAC_t \times DIET_t \times BB\right) + FRAC_w \times CONC \times \left(DIET_w \times BCF + H2O\right)\right] \div BW$$

where:

DOSE	=	dose to wildlife species (mg/kg)
$FRAC_t$	=	fraction of terrestrial diet assumed to be contaminated*
$DIET_t$	=	daily dietary intake of other terrestrial animal species (kg)
BB	=	body burden of terrestrial prey items (mg/kg)
$FRAC_w$	=	fraction of aquatic diet assumed to be contaminated**
CONC	=	concentration of chemical in small stream (mg/L)
$DIET_w$	=	daily dietary intake of aquatic species (kg)
BCF	=	bioconcentration factor (mg/kg per mg/L)
H2O	=	daily water intake (L)
BW	=	body weight (kg)

*equivalent to 1 m^2, the soil area assumed to contain residues from one unit of accelerant, divided by the species' foraging area (m^2)

**assumed to be 1/3, since water flow will move stream concentration of chemical out of local area

6.2.2 Aquatic Species

The aquatic species exposure scenario postulates that fish, tadpoles, and aquatic invertebrates in streams or rivers may be exposed to accelerant residues as a result of chemicals in runoff coming from areas to which the accelerants were applied.

For each chemical, risks were estimated for general fish species, for the water flea (daphnid species) as a representative aquatic invertebrate, and for tadpoles, representing the aquatic stage of amphibians.

The concentrations of the accelerant residues were estimated using the approach described in Section 3.4 and listed in Table 3-1.

6.3 Characterization of Ecological Effects: Ecological Response Analysis and Stressor-Response Profiles

The most commonly used measurement of terrestrial species toxicity in ecological risk assessments is the acute toxicity test. Acute toxicity studies are used primarily to determine the toxicity reference level known as the median lethal dose (LD_{50}), which is the dose that kills 50 percent of the test animals within 14 days of administering a substance. The lower the LD_{50}, the greater the toxicity of the chemical. Toxic symptoms displayed by the animals are recorded throughout the study, and tissues and organs may be examined for abnormalities at the end of the test. In most cases, toxicity studies with laboratory animals such as rats and mice have been used because of the lack of specific wildlife studies. The results of laboratory animal studies are considered to be representative of the effects that would occur in similar species in the wild. Acute toxicity studies are also sometimes available for common avian species, such as bobwhite quail. The toxicity values identified in the following section include oral LD_{50}s for laboratory and field species, as available.

For aquatic species, the LC_{50} is the water concentration that is lethal to half the test population, and is presented in terms of milligrams per liter (mg/L).

The available ecotoxicity data for the accelerant residues are summarized in Tables 6-1 and 6-2 for terrestrial and aquatic species, respectively. If no data are displayed for a particular animal group (e.g., amphibians) for a chemical, it is because no studies were identified with that endpoint during the literature search.

Table 6-1. Ecotoxicity to Terrestrial Species

Accelerant Residue	Test Species	LD_{50} (mg/kg)	Reference
Aluminum oxide	Rat	162	ATSDR 1999a
	Mouse	164	ATSDR 1999a
	Northern bobwhite	>2,303	EPA 2002b
	Japanese quail	1,439	EPA 2002b
Calcium sulfate	No data		
Copper oxide	Rat	376 (lowest lethal dose)	ATSDR 1999b
	Domestic chicken	626	Eisler 1998
Diesel fuel	Rat	7,400	CONCAWE 1996
	Mallard	20	NPS 1997
Gasoline	Rat	14,063	ATSDR 1995
Iron oxide	Mice	5,400 (intraperitoneal)	DHHS 1987
Lead	Dog	1,307 (lowest lethal dose, estimated)	ATSDR 1999c
	Japanese quail	875	HSDB 2002
Manganese dioxide	Rat	17,803	ATSDR 2000
MTBE	Rat	2,962	HSDB 2002
Potassium chloride, potassium hydroxide	Not quantified for terrestrial species; see Section 2.3.10		
Silicon dioxide	Chemically unreactive in the environment, occurs naturally in various forms and is practically non-toxic to non-target organisms.	NA	EPA 1991b
Strontium compounds	Rat	1,139	Oxford 2002

Table 6-2. Ecotoxicity to Aquatic Species

Accelerant Residue	Test Species	LC_{50} (mg/kg)	Reference
Aluminum oxide	Rainbow trout	1.17	EPA 2002b
	Daphnia spp.	2.6	EPA 2002b
	Jefferson salamander	1.4	Pauli et al. 2000
Calcium sulfate	Bluegill sunfish	>2,980	EPA 2002b
	Daphnia magna	>1,970	EPA 2002b
Copper oxide	Rainbow trout	25.4	EPA 2002b
	Ceriodaphnia dubia	0.035	Eisler 1988
Diesel fuel	Rainbow trout	21	Chevron 2001a
	Daphnia magna	20	Chevron 2001a
	Wood frog	4.2	CONCAWE 1996
Gasoline	Rainbow trout	2.7	Chevron 2001b
	Daphnia magna	3.0	Chevron 2001b
Iron oxide	Iron oxide equivalent of iron ambient water quality criterion for protection of freshwater aquatic life	2.9	EPA 1999b
Lead	Rainbow trout	1.17	EPA 2002b
	Daphnia magna	4.4	EPA 2002b
	Northern leopard frog	105	Eisler 1988
Manganese dioxide	Rainbow trout	7.64	Reimer 1988
	Daphnia magna	7.4	Reimer 1988
MTBE	Rainbow trout	880	Johnson 1998
	Ceriodaphnia dubia	340	Johnson 1998
	European common frog	2,000*	Pauli et al. 2000
Potassium chloride, potassium hydroxide	Potassium chloride equivalent of chloride ambient water quality criterion for protection of freshwater aquatic life	230	EPA 1999b
Silicon dioxide	Chemical unreactive in the environment, occurs naturally in various forms and is practically non-toxic to non-target organisms.	NA	EPA 1991b
Strontium compounds	Rainbow trout	0.049**	EPA 2002b
	Eastern narrowmouth toad	0.16†	Pauli et al. 2000

*No lethality observed at this concentration, no LC_{50} available.
**LC_{10}, no LC_{50} available.
†Seven-day LC_{50}, not comparable to exposure durations in this risk assessment.

7.0 ECOLOGICAL RISK CHARACTERIZATION

Risk characterization is the last step in the ecological risk assessment process. The exposure profile is compared to the stressor-response profile, to estimate the likelihood of adverse effects.

7.1 Risk Estimation

By comparing the exposure profile data (estimated dose or water concentration) to the stressor-response profile data (LD_{50}s, LC_{50}s), an estimate of the possibility of adverse effects can be made. The levels of concern are determined following the quotient methodology used by EPA's Office of Pesticide Programs. The quotient is the ratio of the exposure level to the hazard level. For acute exposures, the levels of concern at which a quotient is concluded to reflect risk to non-target species are as follows:

- Terrestrial species (general): 0.5, where dose equals one-half the LD_{50}.
- Terrestrial species (endangered, threatened, sensitive): 0.1, where dose equals one-tenth the LD_{50}.
- Aquatic species (general): 0.5, where water concentration equals one-half the LC_{50}.
- Aquatic species (endangered, threatened, sensitive): 0.05, where water concentration equals one-twentieth the LC_{50}.

Tables 7-1 through 7-3 summarize the estimated risks to terrestrial species, and Table 7-4 summarizes the estimated risks to aquatic species. Ecological risks for each accelerant residue are presented on a per-unit basis, as previously described in the assessment of human health risks.

The risk tables in this section use scientific notation, since many of the values are very small. For example, the notation 3.63E-001 represents 3.63×10^{-1}, or 0.363. Similarly, 4.65E-009 represents 4.65×10^{-9}, or 0.00000000465.

7.2 Estimated Risks to Terrestrial Wildlife

Risks to General Species

On a per-unit basis, no risks from the fire accelerant residues are predicted for terrestrial wildlife species.

Although some terrestrial invertebrates in an area with accelerant residues in the soil may be exposed to accelerant residues and may constitute a portion of the dose to insectivorous wildlife species, populations of beneficial insects as a whole are not expected to suffer adverse impacts because the accelerant applications are quite dispersed over the landscape within a defined burn area.

Table 7-1. Unit Risks to Mammalian Wildlife

		Risk Quotient	
		Deer	
Accelerant	**Residue**	**Mouse**	**Mink**
Fusee	Strontium compounds	1.13E-05	1.77E-03
Gasoline	Gasoline	9.13E-07	1.60E-07
	MTBE	7.65E-05	3.28E-05
	Additive Hazard Quotient	*7.74E-05*	*3.29E-05*
Diesel fuel	Diesel fuel	2.45E-06	4.30E-07
Gasoline + diesel fuel (Driptorch 1:3 mix)	Gasoline, MTBE, diesel fuel	2.12E-05	8.56E-06
Gelling agent	Aluminum oxide	1.18E-05	6.07E-04
Gelled gasoline	Gasoline, MTBE, aluminum oxide	8.92E-05	6.40E-04
Ping-pong balls	Manganese dioxide	4.93E-08	4.17E-06
Launcher pistol flares	Aluminum oxide	2.61E-04	1.34E-02
	Calcium sulfate	ND	ND
	Iron oxide	2.93E-07	1.01E-07
	Copper oxide	6.02E-06	1.03E-04
	Lead	1.61E-08	1.17E-07
	Additive Hazard Quotient	*2.67E-04*	*1.35E-02*
Propane	-none-		

Table 7-2. Unit Risks to Birds

Accelerant	Residue	Risk Quotient: Northern Bobwhite	Risk Quotient: Great Blue Heron	Risk Quotient: Red-Tailed Hawk
Fusee	Strontium compounds	2.27E-06	3.28E-03	5.00E-05
Gasoline	Gasoline	1.84E-07	7.21E-08	4.05E-06
	MTBE	1.54E-05	4.19E-05	1.92E-05
	Additive Hazard Quotient	*1.56E-05*	*4.20E-05*	*2.33E-05*
Diesel fuel	Diesel fuel	1.83E-04	7.15E-05	2.85E-03
Gasoline + diesel fuel (Driptorch 1:3 mix)	Gasoline, MTBE, diesel fuel	1.41E-04	6.41E-05	2.14E-03
Gelling agent	Aluminum oxide	1.70E-07	1.26E-04	3.96E-05
Gelled gasoline	Gasoline, MTBE, aluminum oxide	1.58E-05	1.68E-04	6.29E-05
Ping-pong balls	Manganese dioxide	9.95E-09	7.71E-06	3.20E-06
Launcher pistol flares	Aluminum oxide	3.74E-06	2.79E-03	3.96E-05
	Calcium sulfate	ND	ND	ND
	Iron oxide	5.92E-08	1.15E-07	1.06E-05
	Copper oxide	7.29E-07	1.13E-04	9.11E-05
	Lead	4.84E-09	3.17E-07	6.51E-05
	Additive Hazard Quotient	*4.54E-06*	*2.90E-03*	*2.06E-04*
Propane	-none-			

Table 7-3. Unit Risks to Reptiles and Amphibians

		Risk Quotient	
		Painted	
Accelerant	**Residue**	**Turtle**	**Racer**
Fusee	Strontium compounds	2.06E-03	9.80E-12
Gasoline	Gasoline	3.20E-08	7.94E-13
	MTBE	2.53E-05	6.65E-11
	Additive Hazard Quotient	*2.53E-05*	*6.73E-11*
Diesel fuel	Diesel fuel	3.18E-05	7.88E-10
Gasoline + diesel fuel (Driptorch 1:3 mix)	Gasoline, MTBE, diesel fuel	3.02E-05	6.08E-10
Gelling agent	Aluminum oxide	7.07E-04	1.04E-11
Gelled gasoline	Gasoline, MTBE, aluminum oxide	7.32E-04	7.77E-11
Ping-pong balls	Manganese dioxide	4.86E-06	4.29E-14
Launcher pistol flares	Aluminum oxide	1.56E-02	2.29E-10
	Calcium sulfate	ND	ND
	Iron oxide	6.80E-08	2.55E-13
	Copper oxide	1.19E-04	5.23E-12
	Lead	1.99E-07	2.09E-14
	Additive Hazard Quotient	*1.57E-02*	*2.35E-10*
Propane	-none-		

Table 7-4. Unit Risks to Aquatic Species

		Hazard Quotient					
		Small Stream			Large River		
Accelerant	Residue	Fish	Aquatic Invertebrate	Amphibian	Fish	Aquatic Invertebrate	Amphibian
Fusee	Strontium compounds	ND	ND	ND	ND	ND	ND
	Potassium chloride	9.41E-05	9.41E-05	9.41E-05	2.39E-06	2.39E-06	2.39E-06
	Additive Hazard Quotient	*9.41E-05*	*9.41E-05*	*9.41E-05*	*2.39E-06*	*2.39E-06*	*2.39E-06*
Gasoline	Gasoline*	9.51E-03	8.56E-03	ND	2.42E-04	2.17E-04	ND
	MTBE	1.35E-03	3.51E-03	ND	3.44E-05	8.91E-05	ND
	Additive Hazard Quotient	*1.09E-02*	*1.21E-02*	*ND*	*2.76E-04*	*3.07E-04*	*ND*
Diesel fuel	Diesel fuel	8.22E-04	8.29E-04	2.27E-02	2.09E-05	2.11E-05	5.77E-04
Gasoline + diesel fuel (Driptorch 1:3 mix)	Gasoline, MTBE, diesel fuel	1.17E-02	1.29E-02	2.27E-02	2.97E-04	3.28E-04	5.77E-04
Gelling agent	Aluminum oxide	8.71E-03	3.92E-03	7.28E-03	2.21E-04	9.96E-05	1.85E-04
Gelled gasoline	Gasoline, MTBE, aluminum oxide	1.96E-02	1.60E-02	7.28E-03	4.97E-04	4.06E-04	1.85E-04
Ping-pong balls	Manganese dioxide	6.05E-04	6.24E-04	ND	1.54E-05	1.59E-05	ND
Launcher pistol flares	Aluminum oxide	1.92E-01	8.65E-02	1.61E-01	4.89E-03	2.20E-03	4.08E-03
	Calcium sulfate	1.60E-05	2.42E-05	ND	4.06E-07	6.14E-07	ND
	Iron oxide	2.87E-03	2.87E-03	2.87E-03	7.30E-05	7.30E-05	7.30E-05
	Copper oxide	4.69E-04	3.40E-01	ND	1.19E-05	8.64E-03	ND
	Potassium chloride	2.79E-07	2.79E-07	2.79E-07	7.08E-09	7.08E-09	7.08E-09
	Lead	9.45E-05	2.74E-07	1.05E-06	2.40E-06	6.95E-09	2.67E-08
	Additive Hazard Quotient	*1.96E-01*	*4.30E-01*	*1.64E-01*	*4.97E-03*	*1.09E-02*	*4.16E-03*
Propane	-none-						

*Concentration adjusted to reflect only benzene, toluene, ethylbenzene, and xylene (BTEX), to correspond to LC50 data.

No excess risks to terrestrial plants existing at the time of the burn from the accelerant residues are expected, since the prescribed burn's objective is to remove vegetation from the area. Some of the residues may have localized effects on regrowth vegetation, in the specific locations where any residues remain in the soil. Efroymson et al. (1997) evaluated the effects of soil concentrations of chemicals on plants. Adsorption of chemicals to soil can greatly decrease their availability for plant uptake. The organic carbon partition coefficient (K_{oc}) is a measure of soil adsorption. K_{oc}s for diesel fuel and gasoline, as mixtures, were identified in the literature search conducted to support this risk assessment. Diesel fuel is more strongly held to soil particles, while gasoline is more mobile. These mixtures are also subject to degradation by chemical and biological processes, which would steadily reduce any residue remaining in the soil during and

after vegetative regrowth. Also, for metallic compounds, Efroymson and colleagues stated that, in addition to the metal species' characteristics, the potential for uptake is greatly determined by site-specific parameters: "Soil characteristics (e.g., pH, clay and organic matter content and type, and moisture content) also determine availability to plants by controlling speciation of the element, temporary immobilization by particle surfaces (adsorption-desorption processes), precipitation reactions, and availability in soil solution . . . Although particulate soil organic matter serves to immobilize metals, soluble organic matter may act to keep metals in solution in a form absorbed and translocated by plants." The authors established soil screening benchmarks for several chemicals in soil based on their effects on plants. The estimated localized concentrations of aluminum from a flare could exceed this level; however, the residue (and any effects on vegetation) would be so localized in the area of flare residues that no significant effect to vegetation or regrowth within a landscape would be expected.

Risks to Endangered, Threatened, and Sensitive Species

The hazard quotients estimated in Tables 7-1 through 7-3 were reviewed to determine if any exceeded the sensitive species threshold of 0.1. No quotients greater than 0.1 were identified. Therefore, on a per-unit basis, no risks to endangered, threatened, or sensitive terrestrial species are expected.

7.3 Estimated Risks to Aquatic Wildlife

Stream concentrations, summarized in Table 3-1, are compared to the LC_{50}s presented in Table 6-2, to calculate the risk quotients for aquatic species.

Risks to General Species

Per-unit hazard quotients for fish, aquatic invertebrates, or aquatic stages of amphibians are all less than the risk criterion of 0.5, indicating no risks to general aquatic species are expected.

Risks to Endangered, Threatened, and Sensitive Species

In the small representative watershed, the hazard quotients for fish and aquatic stages of amphibians from aluminum oxide residues from launcher pistol flares slightly exceeds the sensitive species risk criterion of 0.05. No risks were predicted for sensitive species in larger rivers.

7.4 Risk Discussion and Uncertainties

As in the human health risk assessment, the per-unit ecological risk conclusions were translated into the maximum number of units per watershed that would be associated with a conclusion of "negligible risk," based on the methodology of the ecological risk assessment. These results are presented in Table 7-5, and represent only the chemicals for which ecotoxicity data were available with which to quantify risks.

Table 7-5. Maximum Units Associated with Negligible Ecological Risk

Accelerant	Unit	Maximum Number of Units Associated with Negligible Risk	
		Small Watershed*	Large Watershed**
Fusee	One 0.53-lb fusee	153	209084
Gasoline + diesel fuel (Driptorch 1:3 mix)	One gallon	22	866
Gelled gasoline	One gallon†	26	1005
Ping-pong balls	One ball	801	31509
Launcher pistol flares	One flare	1	46
Propane	Tank	NR‡	NR‡

*One acre with 12-cfs stream; accounts for risks to terrestrial and aquatic species.

**100 acres with 350-cfs river; reflects only risks to aquatic species (all terrestrial species exposures assumed small stream).

†Assumed mix rate of 4 lb gelling agent/55 gallons gasoline.

‡No residues expected.

In a supplemental information report prepared by the Forest Service, ranges of estimates were included of the quantity used of several ignition sources for a proposed prescribed burn. These estimates are provided in Table 7-6, along with a comparison to the risk threshold values summarized in Table 7-5.

Widespread exposures to entire populations of wildlife species through environmental pathways are not expected, since the accelerants are used in small amounts in "scattered" patterns over defined areas.

No per-unit risks were predicted for general or sensitive terrestrial species, nor for general aquatic species. In the small representative watershed, the hazard quotients for fish and aquatic

Table 7-6. Comparison of Ignition Source Quantities Used to Estimated Risks

Ignition Source	Estimated Range of Quantity Used (per acre)*	Maximum Units Associated with No Risk		Comments
		Small Watershed	Large Watershed	
Drip torch	1 - 3 gal	22	866	No risk from estimated range.
Helitorch	1 -3 gal	26	1,005	No risk from estimated range.
Fusee	10 - 40	153	209,084	Small drainage areas should be evaluated for the presence of sensitive aquatic species
Flare	10 - 50	1	46	Areas to be burned should be evaluated for the presence of sensitive aquatic species
Plastic sphere	10	801	31,509	No risk from estimated range.

*Source: USDA undated.

stages of amphibians from aluminum oxide residues from launcher pistol flares slightly exceed the sensitive species risk criterion of 0.05. This screening-level conclusion indicates that impacts to small streams containing endangered, threatened, or sensitive aquatic species in the proximity of a proposed prescribed burn should be evaluated based on site-specific characteristics, to ensure that no toxicity from fire accelerant residues poses a risk of adverse effects to such species. This review should be conducted in concert with the pre-project evaluation of the potential effects of the prescribed burn itself on any adjacent aquatic habitat containing protected species. No risks were predicted for sensitive species in larger rivers, which offer greater dilution potential as a result of the river's water volume plus a greater amount of uncontaminated runoff from the larger drainage basin.

It is important to note that there are many conservative assumptions in this analysis that are prompted by the nature of the available information. In particular, a significant source of potential overestimation of risk to aquatic species is the use of 48- or 96-hour LC_{50}s, which are compared to stream and river concentrations at their initial peak loading value, prior to any longitudinal dispersion, hydrolysis, volatilization, degradation, or sorption to sediment, which would rapidly decrease chemical concentrations in the water column below the starting levels. That is, toxicity data from two- to four-day laboratory studies are being compared to stream concentrations that would be expected to be significantly reduced, likely to negligible levels, within 12 to 24 hours at the most. Also, the analytical approach did not reduce the initial residue levels on the soil to account for degradation, evaporation, binding, or other mechanisms that can reduce or remove a chemical from a given environmental transport and exposure route; and also assumed that all the mass of the identified residue chemicals remained as a solid or liquid on the soil, while a significant amount is actually likely to be dispersed in the air during the burning process. The environmental fate processes are dependent on site-specific parameters, including soil types; temperature; soil moisture; other chemicals present in the environment; pH, hardness, and turbulence of surface water; and many others. As a result of simplifying the environmental fate calculations to be independent of these site-specific variables, the results presented in this assessment provide a reasonable upper bound on the potential risks from accelerant residues.

8.0 REFERENCES

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ATTACHMENT 1:
SUMMARY OF LITERATURE SEARCH

Aluminum Oxide, CAS #1344-28-1 (Al_2O_3, aluminum trioxide, alumina)

Data Point	Data Summary	Reference
Water solubility	Practically insoluble 0.000098 g/100 cc = 0.0000098 mg/L	Budavari et al. 1989 ATSDR 1999
K_{oc}		
Soil half-life	No degradation.	ATSDR 1999
BCF	BCFs are less than 300 in fish, since aluminum is highly toxic to fish species.	ATSDR 1999
Ingestion toxicity	A minimal risk level of 2.0 mg/kg/day was estimated for intermediate (15 to 364 days) oral exposure, based on the most sensitive toxicity endpoint (neurotoxicity) identified in studies in laboratory animals.	ATSDR 1999
Carcinogenicity	Chronic ingestion studies in mice and rats using aluminum potassium sulfate or aluminum phosphide led reviewers to conclude that aluminum has not demonstrated carcinogenicity in laboratory animals.	ATSDR 1999
Mammalian tox	Oral LD_{50}s are 162 and 164 mg/kg in rat and mouse, respectively.	ATSDR 1999
Avian tox	14-day LD_{50} >8,000 mg/kg in northern bobwhite and 4,997 in Japanese quail for monoethyl ester phosphonic acid aluminum salt (CAS # 39148-24-8), equivalent to >2,303 and 1,439 mg Al_2O_3/kg, respectively.	EPA 2002
Fish toxicity	96-hour LC_{50} in rainbow trout = 0.310 mg Al/L, equal to 1.17 mg Al_2O_3/L	EPA 2002
Aq. invert. tox	24-hour LC_{50}s in water fleas (Daphnia spp.) were 2.6 and 3.5 mg/L	EPA 2002
Aq. amph. tox	96-hour LC_{50} for aluminum in Jefferson salamander embryos is approximately 0.38 mg/L, equivalent to 1.4 mg Al_2O_3/L LC_{10} (NOEC) was 0.3 mg/L, 24-hour LC_{50} was 0.5 mg/L, LC_{100} was 0.7 mg/L for aluminum in common toad embryos 7-day LC_{50} for aluminum in eastern narrowmouth toad embryo-larvae was 0.05 mg/L	Pauli et al. 2000

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Relevant sections of ATSDR ToxFAQs document:

ToxFAQs™ for Aluminum, CAS# 7429-90-5, June 1999

HIGHLIGHTS: Everyone is exposed to low levels of aluminum from food, air, and water. Exposure to high levels of aluminum may result in respiratory problems.

Aluminum occurs naturally and makes up about 8% of the surface of the earth. It is always found combined with other elements such as oxygen, silicon, and fluorine.

What happens to aluminum when it enters the environment? It binds to particles in the air. It can dissolve in lakes, streams, and rivers depending on the quality of the water. Acid rain may dissolve aluminum from soil and rocks. It can be taken up into some plants from soil. It is not known to bioconcentrate up the food chain.

How might I be exposed to aluminum? Eating small amounts of aluminum in food. Breathing higher levels of aluminum dust in workplace air. Drinking water with high levels of aluminum near waste sites, manufacturing plants, or areas naturally high in aluminum. Eating substances containing high levels of aluminum (such as antacids) especially when eating or drinking citrus products at the same time. Very little enters your body from aluminum cooking utensils.

How can aluminum affect my health? Low-level exposure to aluminum from food, air, water, or contact with skin is not thought to harm your health. Aluminum, however, is not a necessary substance for our bodies and too much may be harmful. People who are exposed to high levels of aluminum in air may have respiratory problems including coughing and asthma from breathing dust. Some studies show that people with Alzheimer's disease have more aluminum than usual in their brains. We do not know whether aluminum causes the disease or whether the buildup of aluminum happens to people who already have the disease. Infants and adults who received large doses of aluminum as a treatment for another problem developed bone diseases, which suggests that aluminum may cause skeletal problems. Some sensitive people develop skin rashes from using aluminum chlorohydrate deodorants.

How likely is aluminum to cause cancer? The Department of Health and Human Services, the International Agency for Research on Cancer, and the EPA have not classified aluminum for carcinogenicity. Aluminum has not been shown to cause cancer in animals.

How does aluminum affect children? Children with kidney problems who were given aluminum in their medical treatments developed bone diseases. Other health effects of aluminum on children have not been studied. It is not known whether aluminum affects children differently than adults, or what the long-term effects might be in adults exposed as children. Large amounts of aluminum have been shown to be harmful to unborn and developing animals because it can cause delays in skeletal and neurological development. Aluminum has been shown to cause lower birthweights in some animals.

Calcium Sulfate, CAS #7778-18-9 ($CaSO_4$, plaster of Paris, gypsum)

Data Point	Data Summary	Reference
Water solubility	3,000 mg/L	HSDB 2002
K_{oc}	No data.	
Soil half-life	Stable. Naturally occurring compound as gypsum.	HSDB 2002
BCF	No data.	
Ingestion toxicity	Substance added directly to human food affirmed as generally recognized as safe (GRAS).	21 CFR 184.1230
Carcinogenicity	Inhalation of calcium sulfate fibers resulted in tumors in laboratory animals.	HSDB 2002
Fish toxicity	96-hour LC_{50} in bluegill sunfish >2,980 mg/L	EPA 2002
Aq. invert. tox	24-hour LC_{50} in water flea *Daphnia magna* >1,970 mg/L	EPA 2002
Aq. amph. tox	No data.	

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HSDB. See Hazardous Substances Databank.

U.S. Environmental Protection Agency. 2002. Ecotox database: Calcium sulfate. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN. http://www.epa.gov/ecotox/

Relevant sections of HSDB file:

Human Toxicity Excerpts:

GYPSUM DUST HAS AN IRRITANT ACTION ON MUCOUS MEMBRANES OF THE RESPIRATORY TRACT & EYES, & THERE HAVE BEEN REPORTS OF CONJUNCTIVITIS, CHRONIC RHINITIS, LARYNGITIS, PHARYNGITIS, IMPAIRED SENSE OF SMELL & TASTE, BLEEDING FROM THE NOSE, & REACTIONS OF TRACHEAL & BRONCHIAL MEMBRANES IN EXPOSED WORKERS. /GYPSUM/
[International Labour Office. Encyclopedia of Occupational Health and Safety. Volumes I and II. New York: McGraw-Hill Book Co., 1971. 630]**PEER REVIEWED**

Because it hardens quickly after absorbing moisture, its ingestion may result in obstruction, particularly at the pylorus. ... To delay "setting," drink glycerin or gelatin solutions, or large volumes of water. Surgical relief may be necessary. /Plaster of Paris/
[Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. II-127]**PEER REVIEWED**

Copper Oxide, CAS #1317-38-0

Data Point	Data Summary	Reference
Water solubility	Practically insoluble. In its Cu(II) state, copper forms coordination compounds or complexes with both inorganic and organic ligands. At the pH values and carbonate concentrations characteristic of natural waters, most dissolved Cu(II) exists as carbonate complexes rather than as free (hydrated) cupric ions.	HSDB 2002 ATSDR 1990
K_{oc}	No data	
Soil half-life	Copper is a stable element. Copper oxide may form complexes with soil or dissolve in water, depending on the pH and organic carbon content of the specific soil.	
BCF	The bioconcentration factor (BCF) of copper in fish obtained in field studies is 10- 100, indicating a low potential for bioconcentration.	ATSDR 1990
Ingestion toxicity	The mean daily dietary intake of copper in adults ranges between 0.9 and 2.2 mg 300 mg Cu/kg/day was the LOAEL causing death in weanling rats when administered over a period of 2 to 15 weeks. Equivalent to 376 mg CuO/kg/day.	HSDB 2002
Carcinogenicity	Inadequate data to determine carcinogenicity.	EPA 1991
Avian toxicity	500 mg Cu/kg caused adverse effects in the domestic chicken, equivalent to 626 mg CuO/kg. No LD_{50} for avian species was identified.	Eisler 1998
Fish toxicity	The 96-hour LC_{50} for rainbow trout was 25.4 mg/L.	EPA 2002
Aq. invert. tox	The 48-hour EC_{50} for intoxication for the water flea *Daphnia magna* was 0.011 to 0.039 mg/L. The 48-hour EC_{50} for mortality in *Ceriodaphnia dubia* was 0.028 mg Cu/L = 0.035 mg CuO/L.	EPA 2002
Aq. amph. tox	No data.	

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U.S. Environmental Protection Agency. 2002. Ecotox database: Cupric oxide. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN. http://www.epa.gov/ecotox/

Relevant sections of ATSDR ToxFAQs document:

HIGHLIGHTS: Copper is an element that is found naturally in the environment. Small amounts of copper are necessary for good health; however, very large amounts can cause dizziness, headaches, diarrhea, and liver and kidney damage.

What happens to copper when it enters the environment? Copper is emitted to the air through natural processes such as windblown dust and volcanic eruptions. Human activities such as copper smelting and ore processing also result in copper being released to the air. Copper may enter the air when it is applied as a fungicide to plants, wood, fabric, and leather. Copper is released to water as a result of natural weathering of soil. It may also be released to water from discharges from industries and sewage treatment plants. Copper may also be added to lakes and ponds to control algae.

How can copper affect my health? Copper is necessary for good health. However, very large doses can be harmful. Long-term exposure to copper in the air can irritate your nose, mouth, and eyes, and cause dizziness, headaches, and diarrhea. Eating or drinking very high amounts of copper can cause liver and kidney damage and effects on the blood. Drinking water with higher than normal levels of copper can cause vomiting, diarrhea, stomach cramps, and nausea. Skin contact with copper can result in an allergic reaction in some people. This reaction is usually skin irritation or a skin rash. Animal studies have shown effects on the stomach and abnormalities in development when animals were fed a diet high in copper. Copper has not been shown to cause cancer in people or animals. The International Agency for Research on Cancer (IARC) has determined that copper is not classifiable as to human carcinogenicity.

The EPA has set a treatment technique for copper in drinking water that includes an action level of 1.3 milligrams of copper per liter of water (1.3 mg/L). The EPA has also set a secondary maximum contaminant level (SMCL) of 1 mg/L of copper in drinking water. An SMCL is a nonenforceable drinking water standard based on taste, odor, or other aesthetic considerations.

Diesel Fuel, CAS #68334-30-5 (Diesel fuel no. 2)

Data Point	Data Summary	Reference
Water solubility	0.00076 mg/L.	TPHCWG 1997, 1998
K_{oc}	log K_{oc} is 6.7 (K_{oc} = 5,011,872)	TPHCWG 1997, 1998
Soil half-life	40% biodegradation in 28 days = $t_{1/2}$ of 21 days	Chevron 2001
BCF	Components of gas oil have measured or calculated log K_{ow} values in the range 3.9 to greater than 6, indicating a high potential to bioaccumulate. However there is little measured data on gas oils or their components and there are major technical difficulties in measuring bioconcentration (BCF) values with complex mixtures.	CONCAWE 1996
Ingestion toxicity	Oral LD_{50} in rats = 7,400 mg/kg Doses of 125+ mg/kg for five days increased the frequency of chromosomal aberrations in the bone marrow of Sprague-Dawley rats	API 1980a, as cited in CONCAWE 1996 WHO 1996
Carcinogenicity	Not classifiable as to carcinogenicity in humans	IARC 1989
Avian toxicity	Mallard LD_{50} = 20 mg/kg	NPS 1997
Fish toxicity	96-hour LC_{50} in rainbow trout is 21 to 210 mg/L 24-hour LC_{50}s were 1.40 to 1.97 for pink salmon, 26.7 to >55.6 for coho salmon, and >23.1 to 168.4 for rainbow trout.	Chevron 2001 WHO 1996
Aq. invert. tox	48-hour EC_{50} in *Daphnia magna* is 20 to 210 mg/L	Chevron 2001
Aq. amph. tox	96-hour LC_{50} for larvae of wood frog *Rana sylvatica* is 4.2 mg/L	Hedtke and Puglisi 1982, as cited in CONCAWE 1996

Chevron Products Co. 2001. Material safety data sheet 6894: Chevron LS diesel 2. San Ramon, CA.

CONCAWE. 1996. Gas oils (diesel fuels/heating oils). Product dossier no. 95/107. Brussels, Belgium.

IARC. See International Agency for Research on Cancer.

International Agency for Research on Cancer. 1989. Diesel fuels. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 45:219. http://193.51.164.11/htdocs/monographs/vol45/45-05.htm

NPS. See U.S. National Park Service.

Total Petroleum Hydrocarbon Criteria Working Group. 1997. Volume III: Selection of representative TPH fractions based on fate and transport considerations Amherst Scientific Publishers. Amherst, MA. http://www.aehs.com/publications/catalog/contents/Volume3.pdf

Total Petroleum Hydrocarbon Criteria Working Group. 1998. Volume I: Analysis of petroleum hydrocarbons in environmental media. Amherst Scientific Publishers. Amherst, MA. http://www.aehs.com/publications/catalog/contents/Volume1.pdf

TPHCWG. See Total Petroleum Hydrocarbon Criteria Working Group.

U.S. National Park Service. 1997. Environmental contaminants encyclopedia: Diesel oil entry. Water Resources Division, Water Operations Branch. Fort Collins, CO. http://www1.nature.nps.gov/toxic/search/

WHO. See World Health Organization.

World Health Organization. 1996. Environmental health criteria 171: Diesel fuel and exhaust emissions. Geneva. http://www.inchem.org/documents/ehc/ehc/ehc171.htm

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Relevant sections of WHO Environmental Health Criteria document:

> The evaluation of diesel fuel opens with a discussion of the complexity of these mixtures and the many variables that affect their quality and composition. An evaluation of toxicity studies in laboratory animals and in vitro test systems concludes that diesel fuel has low acute toxicity when administered via oral, dermal, and inhalation routes. Findings on embryotoxicity, teratogenicity, mutagenicity, and genotoxicity were judged to be either negative or equivocal. In view of inadequacies in the few studies of carcinogenic risks, the report concludes that the main effect of exposure on human health is dermatitis following skin contact.
>
> The second and largest part evaluates diesel exhaust emissions. A review of the abundant data demonstrating adverse effects on the environment concludes that the major components of diesel exhaust contribute to acid deposition, tropospheric ozone formation, and global warming. The most extensive sections discuss the epidemiological studies in humans and studies in experimental animals considered useful for the assessment of risks to human health. Although a number of epidemiological studies have indicated an increased risk of lung cancer in bus and railroad workers, all studies suffered from weaknesses. The report concludes that diesel exhaust is probably carcinogenic to humans, and that inhalation of diesel exhaust contributes to both neoplastic and non-neoplastic diseases, including asthma. The report further concludes that the particulate phase has the greatest effect on human health.

Gasoline, CAS # 8006-61-9

Data Point	Data Summary	Reference
Water solubility	Insoluble	ATSDR 1995
K_{oc}	Log K_{oc} = 1.81 to 4.56 (K_{oc} = 65 to 36,300)	ATSDR 1995
Soil half-life	No data.	
BCF	No data.	
Ingestion toxicity	No NOAEL identified. Lowest LOAELs for endpoints relevant to human toxicity (body weight, gastrointestinal effects) were 2,000 mg/kg in 28-day studies in rats. The oral LD_{50} in rats was 14,063 mg/kg	ATSDR 1995
Carcinogenicity	No studies were located regarding cancer in humans or animals after oral exposure to gasoline. Gasoline is possibly carcinogenic to humans (Group 2B) by inhalation exposure.	ATSDR 1995 IARC 1989
Fish toxicity	96-hour LC_{50} in rainbow trout is 2.7 mg/l (based on values for BTEX).	Chevron 2001
Aq. invert. tox	48-hour LC_{50} in Daphnia magna is 3.0 mg/L (based on values for BTEX).	Chevron 2001
Aq. amph. tox	No data.	

Agency for Toxic Substances and Disease Registry. 1995. Toxicological profile for automotive gasoline. Atlanta, GA. http://www.atsdr.cdc.gov/toxprofiles/tp72.html

ATSDR. See Agency for Toxic Substances and Disease Registry.

Chevron Products Company. 2001. MSDS 2655: Regular unleaded gasoline. San Ramon, CA.

IARC. See International Agency for Research on Cancer.

International Agency for Research on Cancer. 1989. Gasoline. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 45:159. http://193.51.164.11/htdocs/monographs/vol45/45-03.htm

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Relevant sections of ATSDR ToxFAQs document:

ToxFAQs™ for Automotive Gasoline, CAS# 8006-61-9, September 1996

"SUMMARY: Exposure to automotive gasoline most likely occurs from breathing its vapor at a service station while filling a car's fuel tank. At high levels, automotive gasoline is irritating to the lungs when breathed in and irritating to the lining of the stomach when swallowed. Exposure to high levels may also cause harmful effects to the nervous system.

Typically, gasoline contains more than 150 chemicals, including small amounts of benzene, toluene, xylene, and sometimes lead. How the gasoline is made determines which chemicals are present in the gasoline mixture and how much of each is present. The actual composition varies with the source of the crude petroleum, the manufacturer, and the time of year.

What happens to automotive gasoline when it enters the environment? Small amounts of the chemicals present in gasoline evaporate into the air when you fill the gas tank in your car or when gasoline is accidentally spilled onto surfaces and soils or into surface waters. Other chemicals in

gasoline dissolve in water after spills to surface waters or underground storage tank leaks into the groundwater. In surface releases, most chemicals in gasoline will probably evaporate; others may dissolve and be carried away by water; a few will probably stick to soil. The chemicals that evaporate are broken down by sunlight and other chemicals in the air. The chemicals that dissolve in water also break down quickly by natural processes.

Many of the harmful effects seen after exposure to gasoline are due to the individual chemicals in the gasoline mixture, such as benzene and lead. Inhaling or swallowing large amounts of gasoline can cause death. Inhaling high concentrations of gasoline is irritating to the lungs when breathed in and irritating to the lining of the stomach when swallowed. Gasoline is also a skin irritant. Breathing in high levels of gasoline for short periods or swallowing large amounts of gasoline may also cause harmful effects on the nervous system. Serious nervous system effects include coma and the inability to breathe, while less serious effects include dizziness and headaches. There is not enough information available to determine if gasoline causes birth defects or affects reproduction. The Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have not classified automotive gasoline for carcinogenicity. Automotive gasoline is currently undergoing review by the EPA for cancer classification. Some laboratory animals that breathed high concentrations of unleaded gasoline vapors continuously for 2 years developed liver and kidney tumors. However, there is no evidence that exposure to gasoline causes cancer in humans.

Iron Oxide, CAS # 1309-37-1

Data Point	Data Summary	Reference
Water solubility	Insoluble.	HSDB 2002
K_{oc}	No data.	
Soil half-life	Stable.	
BCF	No data.	
Ingestion toxicity	Iron oxide is regulated by the FDA for use as a food coloring and in food packaging; it is generally recognized as safe. Severe toxicity may result in children following ingestion of more than 0.5 g of iron. In adults, chronic excessive ingestion may lead to toxicity, manifested by hemosiderosis, disturbances in liver function, diabetes mellitus, and possible endocrine disturbances and cardiovascular effects. EPA has established a secondary drinking water regulation of 0.3 mg/L for iron, based on aesthetic endpoints. Intraperitoneal LD_{50} is 5,400 mg/kg in mice.	21 CFR 73.200, 186.1300, and 186.1374 (Amdur et al. 1991) EPA 1992 DHHS 1987
Carcinogenicity	Not classifiable as to its carcinogenicity in humans.	IARC 1987
Fish toxicity	EPA set an ambient water quality criteria level of 1 mg/L for protection of aquatic life from iron, equivalent to 2.9 mg Fe_2O_3/L.	EPA 1999
Aq. invert. tox		
Aq. amph. tox		

Amdur, M.O., J. Doull, and C.D. Klaassen (eds.). 1991 *Casarett and Doull=s Toxicology: The Basic Science of Poisons.* 4th edition. Pergamon Press, Inc. Elmsford, NY.

DHHS. See U.S. Department of Health and Human Services.

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

IARC. See International Agency for Research on Cancer.

International Agency for Research on Cancer. 1987. Haematite and ferric oxide. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Supplement 7:216. http://193.51.164.11/htdocs/monographs/suppl7/haematite.html

U.S. Department of Health and Human Services (DHHS). 1987. Registry of Toxic Effects of Chemical Substances (RTECS). DHHS NIOSH Publication No. 87-114. U.S. Government Printing Office. Washington, DC.

U.S. Environmental Protection Agency. 1992. Secondary drinking water regulations: Guidance for nuisance chemicals. EPA 810/K-92-001. Office of Water. Washington, DC.

U.S. Environmental Protection Agency. 1999. National recommended water quality criteria--Correction. EPA 822-A-99-01. Office of Water. Washington, DC.

Summary from Amdur et al. 1991:

> Acute iron toxicity is nearly always due to accidental ingestion of iron-containing medicines, and most often occurs in children. ... Severe toxicity occurs after ingestion of more than 0.5 g of iron or 2.5 g of ferrous sulfate. ... Chronic toxicity or iron overload in adults is a more common problem. ... The pathologic consequences of iron overload are similar regardless of basic cause. The body iron content is increased to between 20 and 40 g. Most of the extra iron is hemosiderin. Greatest concentrations are in parenchymal cells of liver and pancreas, as well as endocrine organs and heart. ... Further clinical effects may include disturbances in liver function, diabetes mellitus, and even endocrine disturbances and cardiovascular effects.

Lead, CAS #7439-92-1

Data Point	Data Summary	Reference
Water solubility	Insoluble.	ATSDR 1999
K_{oc}	Most lead is retained strongly in soil, and very little is transported into surface water or groundwater. Lead is strongly sorbed to organic matter in soil, and although not subject to leaching, it may enter surface waters as a result of erosion of lead-containing soil particulates.	ATSDR 1999
Soil half-life	Stable.	
BCF	Median BCF = 42 in fish.	Eisler 1988
Ingestion toxicity	EPA's reference dose workgroup concluded it was inappropriate to develop a reference dose, or an acceptable daily intake, for lead because some of lead's adverse effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. A lowest lethal dose of 1,400 mg/kg was estimated for lead oxide in dogs, equivalent to 1,307 mg/kg lead.	EPA 1993 ATSDR 1999
Carcinogenicity	Lead is a probable human carcinogen, but a quantitative estimate of risk is not appropriate given current data.	EPA 1993
Avian toxicity	5-day dietary LC_{50} in Japanese quail >5,000 ppm in food, equivalent to approximately 875 mg/kg.	HSDB 2002
Fish toxicity	96-hour LC_{50} in rainbow trout is 1.17 mg/L.	EPA 2002
Aq. invert. tox	48-hour LC_{50} in *Daphnia magna* is 4.4 mg/L.	EPA 2002
Aq. amph. tox	The 30-day LC_{50} value for *Rana pipiens* was 105 mg/L.	Eisler 1988

Agency for Toxic Substances and Disease Registry. 1999. Toxicological profile for lead Atlanta, GA. http://www.atsdr.cdc.gov/toxprofiles/tp13.html

ATSDR. See Agency for Toxic Substances and Disease Registry.

Eisler, R. 1988. Lead hazards to fish, wildlife and invertebrates: A synoptic review. Patuxent Wildlife Research Center, U.S. Fish and Wildlife Service. Laurel, MD. http://www.pwrc.usgs.gov/new/chrback.htm

EPA. See U.S. Environmental Protection Agency.

U.S. Environmental Protection Agency. 1993. Integrated risk information system. Office of Research and Development. Cincinnati, OH. http://www.epa.gov/iris/subst/0277.htm

U.S. Environmental Protection Agency. 2002. Ecotox database: Lead. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN. http://www.epa.gov/ecotox/

Relevant sections of ATSDR ToxFAQs document:

> HIGHLIGHTS: Exposure to lead can happen from breathing workplace air or dust, eating contaminated foods, or drinking contaminated water. Children can be exposed from eating lead-

based paint chips or playing in contaminated soil. Lead can damage the nervous system, kidneys, and reproductive system.

What happens to lead when it enters the environment? Lead itself does not break down, but lead compounds are changed by sunlight, air, and water. When lead is released to the air, it may travel long distances before settling to the ground. Once lead falls onto soil, it usually sticks to soil particles. Movement of lead from soil into groundwater will depend on the type of lead compound and the characteristics of the soil. Much of the lead in inner-city soils comes from old houses painted with lead-based paint.

How can lead affect my health?
Lead can affect almost every organ and system in your body. The most sensitive is the central nervous system, particularly in children. Lead also damages kidneys and the reproductive system. The effects are the same whether it is breathed or swallowed. At high levels, lead may decrease reaction time, cause weakness in fingers, wrists, or ankles, and possibly affect the memory. Lead may cause anemia, a disorder of the blood. It can also damage the male reproductive system. The connection between these effects and exposure to low levels of lead is uncertain. The Department of Health and Human Services has determined that lead acetate and lead phosphate may reasonably be anticipated to be carcinogens based on studies in animals.
There is inadequate evidence to clearly determine lead's carcinogenicity in people. Small children can be exposed by eating lead-based paint chips, chewing on objects painted with lead-based paint, or swallowing house dust or soil that contains lead. Children are more vulnerable to lead poisoning than adults. A child who swallows large amounts of lead may develop blood anemia, severe stomachache, muscle weakness, and brain damage. A large amount of lead might get into a child's body if the child ate small pieces of old paint that contained large amounts of lead. If a child swallows smaller amounts of lead, much less severe effects on blood and brain function may occur. Even at much lower levels of exposure, lead can affect a child's mental and physical growth. Exposure to lead is more dangerous for young and unborn children. Unborn children can be exposed to lead through their mothers. Harmful effects include premature births, smaller babies, decreased mental ability in the infant, learning difficulties, and reduced growth in young children. These effects are more common if the mother or baby was exposed to high levels of lead.

Manganese Dioxide, CAS #1313-13-9 (MnO_2)

Data Point	Data Summary	Reference
Water solubility	Insoluble.	ATSDR 2000
K_{oc}	Sorption of manganese is complicated by redox reactions that produce compounds of different oxidation states. Under aerobic conditions, insoluble manganese 3+ and 4+ compounds predominately form.	HSDB 2002
Soil half-life	Insoluble manganese 3+ and 4+ compounds in sediments may be reduced by manganese-reducing bacteria to soluble manganese 2+ compounds.	HSDB 2002
BCF	A BCF of 100 to 600 was estimated for fish.	ATSDR 2000
Ingestion toxicity	The mean manganese intake in the United States from foodstuffs for a 2-year-old child is estimated to be about 1.5 mg/child/day. The mean manganese intake in the United States from foodstuffs for 25- to 30-year-old man and woman are estimated to be about 2.1 and 2.7 mg/person/day, respectively. ATSDR adopted the National Research Council's upper range of the estimated safe and adequate daily dietary intake of 5 mg/day as a provisional guidance value for oral exposure to manganese; this is equivalent to 0.07 mg/kg/day. EPA has set an oral reference dose of 0.14 mg/kg/day for manganese intake. An oral LD_{50} of 11,250 mg/kg was identified for manganese in rats, equivalent to 17,803 mg MnO_2/kg.	HSDB 2002 ATSDR 2000 EPA 1996 ATSDR 2000
Carcinogenicity	Not classifiable as to carcinogenicity in humans.	EPA 1996
Fish toxicity	96-hour LC_{50} for manganese in rainbow trout was 4.83 mg/L, equivalent to 7.64 mg MnO_2/L.	Reimer 1988
Aq. invert. tox	48-hour LC_{50} for manganese in *Daphnia magna* was 4.7 to 56.1 mg/L, equivalent to 7.4 to 89 mg MnO_2/L.	Reimer 1988
Aq. amph. tox	No data.	

Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for manganese. Atlanta, GA. http://www.atsdr.cdc.gov/toxprofiles/tp151.html

ATSDR. See Agency for Toxic Substances and Disease Registry.

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

Reimer, P. 1988. Environmental effects of manganese and proposed freshwater guidelines to protect aquatic life in British Columbia. Department of Chemical and Bio-Resource Engineering. University of British Columbia.

U.S. Environmental Protection Agency. 1996. Integrated risk information system. Office of Research and Development. Cincinnati, OH. http://www.epa.gov/iris/subst/0373.htm

Relevant sections of ATSDR ToxFAQs document:

Manganese is an essential trace element and is necessary for good health. Manganese can be found in several food items, including grains and cereals, and is found in high amounts in other foods, such as tea.

What happens to manganese when it enters the environment? Manganese can enter the air from iron, steel, and power plants, coke ovens, and from dust from mining operations. It can enter the water and soil from natural deposits, disposal of wastes, or deposits from airborne sources. Manganese exists naturally in rivers, lakes, and underground water. Plants in the water can take up some of the manganese from water and concentrate it.

How can manganese affect my health? Some individuals exposed to very high levels of manganese for long periods of time in their work developed mental and emotional disturbances and slow and clumsy body movements. This combination of symptoms is a disease called "manganism." Workers usually do not develop symptoms of manganism unless they have been exposed to manganese for many months or years. Manganism occurs because too much manganese injures a part of the brain that helps control body movements. Exposure to high levels of airborne manganese, such as in a manganese foundry or battery plant, can affect motor skills such as holding one's hand steady, performing fast hand movements, and maintaining balance. Exposure to high levels of the metal may also cause respiratory problems and sexual dysfunction. There are no human cancer data available for manganese. Exposure to high levels of manganese in food resulted in a slightly increased incidence of pancreatic tumors in male rats and thyroid tumors in male and female mice. The EPA has determined that manganese is not classifiable as to human carcinogenicity.

Daily intake of small amounts of manganese is needed for growth and good health in children. Manganese is constantly present in the mother and is available to the developing fetus during pregnancy. Manganese is also transferred from a nursing mother to her infant in breast milk at levels that are appropriate for proper development. Children, as well as adults, who lose the ability to remove excess manganese from their bodies develop nervous system problems. Because at certain ages children take in more than adults, there is concern that children may be more susceptible to the toxic effects of excess manganese. Animal studies indicate that exposure to high levels of manganese can cause birth defects in the unborn. There is no information on whether mothers exposed to excess levels of manganese can transfer the excess to their developing fetus during pregnancy or to their nursing infant in breast milk.

The EPA has set a non-enforceable guideline for the level of manganese in drinking water at 0.05 milligrams per liter (0.05 mg/L). The National Research Council has recommended safe and adequate daily intake levels for manganese that range from 0.3 to 1 mg/day for children up to 1 year, 1 to 2 mg/day for children up to age 10, and 2 to 5 mg/day for children 10 and older.

MTBE, CAS #1634-04-4 (methyl *tert*-butyl ether)

Data Point	Data Summary	Reference
Water solubility	48,000 mg/L	ATSDR 1996
K_{oc}	Log K_{oc} estimated as 1.05 and calculated as 2.89 (K_{oc}s = 11.2 and 776, respectively) Log K_{oc}s reported as 1.091, 1.035, 1.049 (K_{oc}s = 12.3,10.8, and 11.2, respectively)	ATSDR 1996 Malcolm Pirnie 1999
Soil half-life	Rapid volatilization from surface soils, little degradation in subsurface.	ATSDR 1996
BCF	Insignificant (BCF = 1.5 to 3, with levels rapidly declining after exposure ends). Log BCF was 0.18 in Japanese carp (BCF = 1.5).	ATSDR 1996 EFDB 2002
Ingestion toxicity	ATSDR derived an intermediate-duration minimal risk level of 0.3 mg/kg/day. An oral rat LD_{50} of 4.0 mL/kg was identified; this is equal to 2,962 mg/kg.	ATSDR 1996 HSDB 2002
Carcinogenicity	Possible human carcinogen at high doses. Cancer slope factor = 0.004 per mg/kg/day.	EPA 1997
Fish toxicity	Rainbow trout LC_{50} is 880 to 1,240 mg/L	Johnson 1998
Aq. invert. tox	*Ceriodaphnia dubia* LC_{50} is 340 to 680 mg/L	Johnson 1998
Aq. amph. tox	100 mg/L led to increased weight, stimulated metamorphosis; <2,000 mg/L had no lethal effect on European common frog tadpoles	Pauli et al. 2000

Agency for Toxic Substances and Disease Registry. 1996. Toxicological profile for methyl *tert*-butyl ether. Atlanta, GA. http://www.atsdr.cdc.gov/toxprofiles/tp91.html

ATSDR. See Agency for Toxic Substances and Disease Registry.

EFDB. See Environmental Fate Database.

Environmental Fate Database. 2002. On-line database. Syracuse Research Corporation. http://esc.syrres.com/efdb.htm

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

Johnson, M.L. 1998. Ecological risk of MTBE in surface waters. John Muir Institute of the Environment, University of California. Davis, CA.

Malcolm Pirnie, Inc. 1999. Technical memorandum: Evaluation of fate and transport of methyl tertiary butyl ether (MTBE) in gasoline following a small spill. Prepared for Oxygenated Fuels Association, Inc. Oakland, CA.

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters, Hull, Québec, Canada. http://www.cws-scf.ec.gc.ca/nwrc/ratl/about_e.htm

U.S. Environmental Protection Agency. 1997. Drinking water advisory: Consumer acceptability advice and health effects analysis on methyl tertiary-butyl ether (MtBE). EPA-822-F-97-009. Office of Water. Washington, DC.

Relevant sections of ATSDR ToxFAQs document:

ToxFAQs™ for Methyl *tert*-Butyl Ether, CAS# 1634-04-4, September 1997

What happens to methyl tert-butyl ether (MTBE) when it enters the environment? MTBE quickly evaporates from open containers and surface water, so it is commonly found as a vapor in the air. Small amounts of MTBE may dissolve in water and get into underground water. It remains in underground water for a long time. MTBE may stick to particles in water, which will cause it to eventually settle to the bottom sediment. MTBE may be broken down quickly in the air by sunlight. MTBE does not build up significantly in plants and animals.

How can methyl tert-butyl ether (MTBE) affect my health? Breathing small amounts of MTBE for short periods may cause nose and throat irritation. Some people exposed to MTBE while pumping gasoline, driving their cars, or working in gas stations have reported having headaches, nausea, dizziness, and mental confusion. However, the actual levels of exposure in these cases are unknown. In addition, these symptoms may have been caused by exposure to other chemicals. There are no data on the effects in people of drinking MTBE. Studies with rats and mice suggest that drinking MTBE may cause gastrointestinal irritation, liver and kidney damage, and nervous system effects. There is no evidence that MTBE causes cancer in humans. One study with rats found that breathing high levels of MTBE for long periods may cause kidney cancer. Another study with mice found that breathing high levels of MTBE for long periods may cause liver cancer. The Department of Health and Human Services (DHHS), the International Agency for Research on Cancer (IARC), and the EPA have not classified MTBE as to its carcinogenicity.

Has the federal government made recommendations to protect human health? The EPA has issued guidelines recommending that, to protect children, drinking water levels of MTBE not exceed 4 milligrams per liter of water (4 mg/L) for an exposure of 1-10 days, and 3 mg/L for longer-term exposures. The American Conference of Governmental Industrial Hygienists (ACGIH) has recommended an exposure limit of 40 parts of MTBE per million parts of air (40 ppm) for an 8-hour workday, 40-hour workweek.

Polystyrene, CAS #9003-53-6

Data Point	Data Summary	Reference
Water solubility	ND	
K_{oc}	ND	
Soil half-life	ND	
BCF	ND	
Ingestion toxicity	Not absorbed when administered orally to laboratory rats.	Monte 1983
Carcinogenicity	Subcutaneous implantation of polystyrene discs, rods, spheres or powder in rats induced local sarcomas, the incidences of which varied with the size and form of the implant.	IARC 1979
Fish toxicity	ND	
Aq. invert. tox	ND	
Aq. amph. tox	ND	

International Agency for Research on Cancer. 1979. Styrene, polystyrene, and styrene-butadiene compounds. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 19:231. http://193.51.164.11/htdocs/monographs/vol19/styrene%26polymers.html

Monte, W. 1983. Lack of gut absorption of solubilized polystyrene by the rat (abstract). Journal of Agricultural and Food Chemistry 31(1):174-175.

Summary of polystyrene uses and toxicity:

Polystyrene is formed by the polymerization of styrene to form a rigid, odorless, tasteless plastic. It is widely used in consumer products, including video and audio cassettes, cosmetic containers, toys, computer housings, and packaging and insulating materials for food, including the air-blown form of polystyrene known as Styrofoam® (EPA 1995)[1].

Monte (1983) concluded that polystyrene was not absorbed when administered orally to laboratory rats. IARC (1979) reported that implantation of polystyrene materials under the skin in rats caused sarcomas. No quantitative toxicity data were available for the routes of exposure evaluated in this risk assessment; therefore, risk from polystyrene could not be quantified.

[1]U.S. Environmental Protection Agency. 1995. AP-42: Compilation of air pollutant emission factors. 5th ed., volume 1. Office of Air Quality Planning and Standards. Research Triangle Park, NC.

Potassium Chloride, CAS # 7447-40-7 (KCl), and Potassium Hydroxide, CAS # 1310-58-3 (KOH)

Data Point	Data Summary	Reference
Water solubility	281,000 mg/L (KCl) and 970,000 mg/L (KOH)	HSDB 2002
K_{oc}	No data.	
Soil half-life	No data.	
BCF	No data.	
Ingestion toxicity	Maximal nontoxic oral dose of KCl in man varies from 200 to 1,000 mg/kg/day, depending on efficiency of individual renal excretory mechanism. KOH is one of the strongest alkalies--it is extremely corrosive. Swallowing caustic alkalies causes immediate burning pain in the mouth, throat, and stomach, and the lining membranes become swollen and detached.	HSDB 2002
Carcinogenicity	No data.	
Fish toxicity	EPA has set an ambient water quality criteria level of 230 mg/L for chloride for the protection of freshwater aquatic life.	EPA 1999
Aq. invert. tox		
Aq. amph. tox		

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

U.S. Environmental Protection Agency. 1999. National recommended water quality criteria--Correction. EPA 822-A-99-01. Office of Water. Washington, DC.

Relevant sections of HSDB file:

HUMAN HEALTH EFFECTS:

HUMAN TOXICITY EXCERPTS:

LARGE DOSES BY MOUTH CAN CAUSE GI IRRITATION, PURGING, WEAKNESS AND CIRCULATORY DISTURBANCES. [The Merck Index. 9th ed. Rahway, New Jersey: Merck & Co., Inc., 1976. 990]**PEER REVIEWED**

NAUSEA, VOMITING, DIARRHEA, & ABDOMINAL DISCOMFORT COMMONLY OCCUR. OVERDOSES MAY CAUSE PARESTHESIAS, GENERALIZED WEAKNESS, FLACCID PARALYSIS, LISTLESSNESS, VERTIGO, MENTAL CONFUSION, HYPOTENSION, CARDIAC ARRHYTHMIAS, & HEART BLOCK. DEATH MAY ENSUE. [Osol, A. and J.E. Hoover, et al. (eds.). Remington's Pharmaceutical Sciences. 15th ed. Easton, Pennsylvania: Mack Publishing Co., 1975. 771]**PEER REVIEWED**

ACUTE POTASSIUM INTOXICATION BY MOUTH IS RARE BECAUSE LARGE SINGLE DOSES USUALLY INDUCE VOMITING AND BECAUSE IN THE ABSENCE OF PRE-EXISTING KIDNEY DAMAGE POTASSIUM IS RAPIDLY EXCRETED. /POTASSIUM SALTS/ [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. II-124]**PEER REVIEWED**

Potassium chloride in a commercial dietary salt substitute ... has produced a near fatal poisoning in an 8 month old infant. [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. II-124]**PEER REVIEWED**

MAXIMAL NONTOXIC ORAL DOSE OF KCL IN MAN VARIES FROM 0.2 TO 1.0 G K/KG/DAY, DEPENDING UPON EFFICIENCY OF INDIVIDUAL RENAL EXCRETORY MECHANISM; LOWER DOSES SOMETIMES CAUSE IMPAIRMENT OF RENAL FUNCTION AS SHOWN BY REDUCED INULIN & UREA CLEARANCE. ... SERUM K LEVEL OF 40 MG/100 ML IS FATAL IN MAN. [Venugopal, B. and T.D. Luckey. Metal Toxicity in Mammals, 2. New York: Plenum Press, 1978. 16]**PEER REVIEWED**

Silicon Dioxide, CAS #7631-86-9 (silica)

Data Point	Data Summary	Reference
Water solubility	Practically insoluble.	HSDB 2002
K_{oc}	Not applicable.	
Soil half-life	Stable (occurs as sand and quartz).	HSDB 2002
BCF	None.	
Ingestion toxicity	When male and female beagle dogs or CD rats were fed 800 mg silicon/kg/day as the dioxide for 1 month ... neither clinical signs of toxicity nor histologic changes were seen in these animals. It is chemically and biologically inert when ingested. It is approved for use in food products at levels up to 2%, and is Generally Recognized as Safe (GRAS).	HSDB 2002, EPA 2002
Carcinogenicity	Crystalline silica is carcinogenic.	HSDB 2002, EPA 1991
Fish toxicity	Chemically unreactive in the environment, occurs naturally in various forms and is practically non-toxic to non-target organisms.	EPA 1991
Aq. invert. tox		
Aq. amph. tox		

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

U.S. Environmental Protection Agency. 1991. Reregistration eligibility document: Silicon dioxide and silica gel. Office of Pesticide Programs. Washington, DC. http://www.epa.gov/oppsrrd1/REDs/old_reds/4081red.pdf

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Relevant sections of HSDB file:

Human Toxicity Excerpts:

The details of toxicity associated with metallurgical silicon are unknown.
[Kirk-Othmer Encyclopedia of Chemical Technology. 3rd ed., Volumes 1-26. New York, NY: John Wiley and Sons, 1978-1984.,p. V20 851 (1982)]**PEER REVIEWED**

Nuisance particulate (accumulation in lungs).
[Cralley, L.J., L.V. Cralley (eds.). Patty's Industrial Hygiene and Toxicology. Volume III: Theory and Rationale of Industrial Hygiene Practice. 2nd ed., 3A:The Work Environment. New York, NY: John Wiley Sons, 1985. 181]**PEER REVIEWED**

... Increased renal silicon (200 ppm dry weight; normal = 14-23 ppm) /was found/in an adult male bricklayer who presented with proteinuria and hypertension, but who had a normal chest roentgenogram. Moderate thickening of the glomerular basement membrane was noted on transmission electron microscopy. [American Conference of Governmental Industrial Hygienists, Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. Volumes I,II, III. Cincinnati, OH: ACGIH, 1991. 1387]**PEER REVIEWED**

Unpleasant deposits /of silicon dust/ in eyes, ears & nasal passages & injury to the skin and mucous membranes may be caused by the dust itself or by cleansing procedures used for its

removal. [Sittig, M. Handbook of Toxic and Hazardous Chemicals and Carcinogens, 1985. 2nd ed. Park Ridge, NJ: Noyes Data Corporation, 1985. 787]**PEER REVIEWED**

Silicon is not found free in nature, but occurs chiefly as the oxide, & as silicates. Sand, quartz, rock crystal, amethyst, agate, flint, jasper, & opal are some of the /oxide/ forms. Granite, hornblende, asbestos, feldspar, clay, mica ... are but a few of the numerous silicate minerals. [Lide, D.R. (ed.). CRC Handbook of Chemistry and Physics. 73rd ed. Boca Raton, FL: CRC Press Inc., 1992-1993.,p. 4-26]**PEER REVIEWED**

Strontium Oxides and Sulfate, CAS # 1314-11-0 (strontium oxide, SrO), 1314-18-7 (strontium peroxide, SrO_2), 7759-02-6 (strontium sulfate, $SrS0_4$)

Data Point	Data Summary	Reference
Water solubility	SrO forms the hydroxide with evolution of heat in presence of water. SrO_2 is almost insoluble in water, but is gradually decomposed by water with the evolution of oxygen. $SrSO_4$ is soluble in water at about 114 mg/L.	Budavari et al. 1989
K_{oc}	The distribution coefficient, K_d (amount of ion per kg of air dry soil/amount of ion per liter of soil solution), for strontium in a podsol forest soil was determined to be 140 L/kg in the top layer and 44 L/kg in the lower layer.	HSDB 2002
Soil half-life	No data.	
BCF	BCF of strontium was 576 to 1,286 in bluegill sunfish.	HSDB 2002
Ingestion toxicity	The strontium ion has a low order of toxicity. It is chemically and biologically similar to calcium. The oxides are moderately caustic materials. The human daily intake of strontium has been determined to be 2 mg. An oral reference dose of 0.6 mg/kg/day was estimated for stable strontium. An oral rat LD_{50} of 2,750 mg/kg was identified for strontium nitrate Sr(NO3)2. This is equivalent to an LD_{50} of 1,139 mg strontium/kg.	Lewis 1994 HSDB 2002 EPA 1996 Oxford 2002
Carcinogenicity	No data.	
Fish toxicity	A 96-hour LC_{10} of 0.049 mg/L was identified for Sr for newly hatched rainbow trout.	EPA 2002
Aq. invert. tox	No data.	
Aq. amph. tox	7-day LC_{50} for Sr in eastern narrowmouth toad embryo-larvae was 0.16 mg/L	Pauli et al. 2000

Budavari, S., M. O'Neil, A. Smith, and P. Heckelman, eds. 1989. *The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals.* 11th ed. Merck & Co., Inc. Rahway, NJ.

EPA. See U.S. Environmental Protection Agency.

Hazardous Substances Databank. 2002. On-line database. National Library of Medicine. Bethesda, MD. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

HSDB. See Hazardous Substances Databank.

Lewis, R. 1994. *Sax's Dangerous Properties of Industrial Materials.* 8th ed. Van Nostrand Reinhold Company. New York.

Oxford University. 2002. Safety data for strontium nitrate. The Physical and Theoretical Chemistry Laboratory. http://physchem.ox.ac.uk/MSDS/ST/strontium_nitrate.html

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters, Hull, Québec, Canada. http://www.cws-scf.ec.gc.ca/nwrc/ratl/about_e.htm

U.S. Environmental Protection Agency. 1996. Integrated risk information system. Office of Research and Development. Cincinnati, OH. http://www.epa.gov/iris/subst/0550.htm

U.S. Environmental Protection Agency. 2002. Ecotox database: Lead. Mid-Continent Ecology Division, National Health and Environmental Effects Research Laboratory, Office of Research and Development. Duluth, MN. http://www.epa.gov/ecotox/

Summary from Lewis 1994:

STRONTIUM COMPOUNDS
DPIM: SMH500 Hazard Rating: 1
SAFETY PROFILE:
The strontium ion has a low order of toxicity. It is chemically and biologically similar to calcium. Strontium salicylate is the most toxic compound. The oxides and hydroxides are moderately caustic materials. Symptoms of acute toxicity are excessive salivation, vomiting, colic, and diarrhea, and possibly respiratory failure. The gastrointestinal absorption of soluble strontium ranges from 5 to 25%. Workers in strontium salt plants have reduced activity of choline esterase and acetylcholine. Drinking water with 13 mg Sr/L caused impaired tooth development in 1-year-old children. As with other compounds, the toxicity of a given compound may be a function of the anion. Compounds are highly dangerous if they contain the radioactive isotope 90Sr.
Updated: 08/27/90

ATTACHMENT 2:
MATERIAL SAFETY DATA SHEETS

ORION SAFETY PRODUCTS
STANDARD FUSEE CORP. **NSN#: 1370-01-009-2593**
MATERIAL SAFETY DATA **EMERGENCY CONTACT:**
- RAILWAY & HIGHWAY FUSEES - CHIMFEX **CHEMTREC 1-800-424-9300**
- *ORION* MARINE SIGNAL PRODUCTS (FORMERLY OLIN SIGNAL PRODUCTS)

SECTION I - IDENTIFICATION

CHEMICAL NAMES & SYNONYMS: **FUSEE**

CHEMICAL FAMILY: PYROTECHNIC DEVICE	**FORMULA:** MIXTURE	**TRADE NAME:** *FUSEE/FLARE*
DESCRIPTION: RED CARDBOARD TUBE CONTAINING YELLOWISH-TAN SOLID MIXTURE.		**CAS NO.:** NONE ASSIGNED/MIXTURE

SECTION II - HAZARDOUS INGREDIENTS

BASIC MATERIAL	OSHA P.E.L	LD 50	LC 50	SIGNIFICANT EFFECTS
STRONTIUM NITRATE C.A.S.# 10042769	NONE EST.	2750 mg/Kg (RAT)	NO DATA	IRRITANT TO SKIN, EYES AND MUCOUS MEMBRANES.
POTASSIUM PERCHLORATE C.A.S.# 7778747	NONE EST.	NO DATA	NO DATA	IRRITANT TO SKIN, EYES AND MUCOUS MEMBRANES.
SULFUR C.A.S.# 7704349	NONE EST.	NO DATA	NO DATA	MAY CAUSE EYE AND MUCOUS MEMBRANE IRRITATION.
SAWDUST/OIL BINDER C.A.S.# MIXTURE	NONE EST.	NO DATA	NO DATA	N/A

SECTION III - NORMAL HANDLING PROCEDURES

PRECAUTIONS TO BE TAKEN IN HANDLING AND STORAGE: **KEEP ITEMS OUT OF THE REACH OF CHILDREN!!**

CONTENTS MAY BE HARMFUL IF SWALLOWED. IF TUBE IS BROKEN, DO NOT GET CONTENTS IN EYES, ON SKIN OR ON CLOTHING. UPON CONTACT WITH SKIN OR EYES, WASH WITH WATER. AVOID BREATHING DUST. STORE IN A COOL, DRY, WELL-VENTILATED PLACE AWAY FROM ALL SOURCES OF IGNITION. EXERCISE CAUTION WHEN USING THIS PRODUCT SINCE MOLTEN FLECKS MAY BE EMITTED. DO NOT USE PRODUCT NEAR ANY FLAMMABLE OR COMBUSTIBLE MATERIALS. AVOID CONTACT WITH STRONG OXIDIZERS. AVOID OPEN FLAMES AND TEMPERATURES >167°F.

PROTECTIVE EQUIPMENT	VENTILATION REQUIREMENTS
FOLLOW DIRECTIONS ON PACKAGE. ALWAYS POINT FUSEE AWAY FROM FACE AND BODY WHILE IGNITING AND AFTERWARDS.	THIS PRODUCT SHOULD ONLY BE USED OUTDOORS. IF USED INDOORS, LOCAL MECHANICAL VENTILATION IS RECOMMENDED TO MINIMIZE EXPOSURE.

SECTION IV - FIRE AND EXPLOSION DATA

FLASH POINT/METHOD: N/A CONTENTS: >167°F (OVEN)	**OSHA CLASSIFICATION:** FLAMMABLE SOLID	**FLAMMABLE EXPLOSIVE LIMITS:** UPPER: NONE ESTAB. LOWER: NONE ESTAB.

EXTINGUISHING MEDIA: USE WATER DELUGE/FLOODING METHODS; **SUFFOCATION TECHNIQUES WILL NOT BE EFFECTIVE:** NO THREAT OF MASS EXPLOSION.

SPECIAL FIRE HAZARD & FIRE FIGHTING PROCEDURES: USE **NIOSH/MSHA** APPROVED POSITIVE PRESSURE SELF-CONTAINED BREATHING APPARATUS WHEN ANY MATERIAL IS INVOLVED IN FIRE.

SECTION V - HEALTH HAZARD DATA

THRESHOLD LIMIT VALUE: NONE ESTABLISHED FOR THE MIXTURE.

SYMPTOMS OF OVER EXPOSURE: TUBE CONTENTS ARE CORROSIVE TO THE EYES AND IRRITATING TO THE RESPIRATORY TRACT AND SKIN.

SKIN: IMMEDIATELY FLUSH WITH WATER FOR 15 MINUTES, CALL A PHYSICIAN IF IRRITATION OCCURS.

EYES: IMMEDIATELY FLUSH WITH WATER FOR 15 MINUTES, CALL A PHYSICIAN.

INGESTION: IF CONTENTS ARE INGESTED: IMMEDIATELY GIVE LARGE QUANTITIES OF WATER OR EPSOM SALT SOLUTION. INDUCE VOMITING AND CALL A PHYSICIAN.

INHALATION: IMMEDIATELY REMOVE VICTIM TO FRESH AIR. CALL A PHYSICIAN.

CHEMICAL NAME: FUSEE

SECTION VI - TOXICOLOGY (PRODUCT)

ALL CHEMICAL IS CONTAINED IN CARDBOARD TUBE - NONE IS EXPOSED

ACUTE ORAL LD 50: APPROXIMATELY 3 GRAMS/kg (RAT)	CARCINOGENICITY: NOT KNOWN TO BE CARCINOGENIC
	MUTAGENICITY: NOT KNOWN TO BE MUTAGENIC
ACUTE DERMAL LD 50: APPROX. 2 GRAMS/kg (RABBIT)	EYE IRRITATION: CORROSIVE
ACUTE INHALATION LD 50: NOT ESTABLISHED	PRIMARY SKIN EFFECT: IRRITANT

PRINCIPLE ROUTES OF ABSORPTION: INHALATION; DERMAL CONTENT

EFFECTS OF ACUTE EXPOSURE: TUBE CONTENTS ARE CORROSIVE TO EYES AND IRRITATING TO RESPIRATORY TRACT AND SKIN. INHALATION OF COMBUSTION PRODUCTS WILL IRRITATE EYES, LUNGS, AND MUCOUS MEMBRANES.

EFFECTS OF CHRONIC EXPOSURE: NONE KNOWN OR REPORTED

SECTION VII - SPILL AND LEAKAGE PROCEDURES (CONTROL PROCEDURES)

ACTION FOR MATERIAL RELEASE OR SPILL: IN LARGE QUANTITY, REMOVE ALL SOURCES OF IGNITION. IF TUBE IS BROKEN, WEAR NIOSH/MSHA APPROVED DUST RESPIRATOR. FOLLOW O.S.H.A. REGULATIONS FOR RESPIRATOR USE, (SEE 29CFR 1910.134). WEAR GOGGLES, IMPERVIOUS COVERALLS, GLOVES AND BOOTS. CLEAN UP IN A MANNER TO MINIMIZE CONTAMINATION WITH ORGANIC MATERIAL. DO NOT RETURN MATERIAL TO ORIGINAL CONTAINER. PLACE IN A FRESH CONTAINER AND ISOLATE. DO NOT SEAL THE CONTAINER. IN THE EVENT OF A LARGE SPILL, CALL THE EMERGENCY TELEPHONE NUMBER.

TRANSPORTATION EMERGENCY, CONTACT CHEMTREC 800-424-9300

WASTE DISPOSAL METHOD: DISPOSE OF CONTAMINATED PRODUCT, EMPTY CONTAINER AND MATERIALS USED IN CLEANING UP SPILLS OR LEAKS IN A MANNER APPROVED FOR THIS MATERIAL. CONSULT APPROPRIATE FEDERAL, STATE AND LOCAL REGULATORY AGENCIES TO ASCERTAIN PROPER DISPOSAL PROCEDURES.
OPEN BURNING IS PREFERRED METHOD OF DISPOSAL FOR PYROTECHNICS, IN COMPLIANCE WITH STATE AND LOCAL REGULATIONS.

SECTION VIII - SHIPPING DATA

D.O.T. CLASS (DOMESTIC): FUSEE, 4.1, NA1325, PG II (GROUND ONLY) LABEL REQ: FLAMMABLE SOLID

INTERNATIONAL CLASS & DOMESTIC AIR: SIGNAL DEVICES, HAND, 1.4S, UN0373, PG II LABEL REQ: 1.4S

SECTION IX - REACTIVITY DATA

STABLE X UNSTABLE @ _____°C _____°F	HAZARDOUS POLYMERIZATION	MAY OCCUR
		WILL NOT OCCUR X

CONDITIONS TO AVOID: HIGH HEAT, IGNITION SOURCES OF ANY KIND

INCOMPATIBILITY *(MATERIAL TO AVOID)*: STRONG OXIDIZERS; STRONG ACIDS; CHLORATE SALTS.

HAZARDOUS DECOMPOSITION PRODUCTS: SULFUR OXIDES, NITROGEN OXIDES, CARBON MONOXIDE.

SECTION X - PHYSICAL DATA

MELTING POINT: NO DATA	VAPOR PRESSURE: NO DATA	VOLATILES: NO DATA
BOILING POINT: NO DATA	SOLUBILITY IN WATER: >300 g/l pwdr	EVAPORATION RATE: NO DATA
SPECIFIC GRAVITY (H_2O = 1): NO DATA	pH NO DATA	VAPOR DENSITY (AIR = 1): NO DATA

INFORMATION FURNISHED BY: JEFFREY W. JOHNSON DATE: MARCH 1998

ORION SAFETY PRODUCTS/STANDARD FUSEE CORPORATION
P.O. BOX 1047 • EASTON, MD 21601 HIGHWAY and RAILWAY PRODUCTS 1-800-637-7807
CHIMFEX and ORION MARINE PRODUCTS: 1-800-851-5260

MOBIL OIL CORP, AMERICAS MARKE -- MOBIL REGULAR UNLEADED GASOLINE

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MSDS Safety Information
==========
FSC: 9130
NIIN: 00-148-7103
MSDS Date: 05/17/2000
MSDS Num: CLDBG
Product ID: MOBIL REGULAR UNLEADED GASOLINE
MFN: 03
Responsible Party
Cage: 3U728
Name: MOBIL OIL CORP, AMERICAS MARKETING AND REFINING
Address: 3225 GALLOWS ROAD
City: FAIRFAX VA 22037
Info Phone Number: 800-662-4525/ 856-224-4644
Emergency Phone Number: 609-737-4411
Resp. Party Other MSDS No.: 33126-00
Chemtrec IND/Phone: (800)424-9300
Published: Y
==========
Contractor Summary
==========
Cage: 3U728
Name: MOBIL OIL CORP, NORTH AMERICAS MARKETING AND REFINING
Address: 3225 GALLOWS ROAD
City: FAIRFAX VA 22037
Phone: 800-662-4525/ 856-224-4644
==========
Item Description Information
==========
Item Name: GASOLINE,AUTOMOTIVE
Unit of Issue: GL
UI Container Qty: X
==========
Ingredients
==========
Cas: 8006-61-9
RTECS #: LX3300000
Name: GASOLINE (PRODUCT) (COMPONENTS FOLLOW BELOW)
Percent by Wt: 100.
OSHA PEL: 900 MG/KG;300 PPM
OSHA STEL: 1500 MG/KG;500 PPM
ACGIH TLV: 890 MG/M3;300 PPM
ACGIH STEL: 1480 MG/M3;500 PPM

Cas: 1634-04-4
RTECS #: KN5250000
Name: METHYL T- BUTYL ETHER
Percent by Wt: 15.
ACGIH TLV: 144 MG/M3;40 PPM
EPA Rpt Qty: 1 LB
DOT Rpt Qty: 1 LB

Cas: 64-17-5
RTECS #: KQ6300000
Name: ETHANOL
Percent by Wt: 11.
OSHA PEL: 1900 MG/M3;1000 PPM
ACGIH TLV: 1880 MG/M3;1000 PPM

Cas: 1330-20-7
RTECS #: ZE2100000

Name: XYLENE
Percent by Wt: 10.
OSHA PEL: 435 MG/M3; 100PPM
OSHA STEL: 655 MG/M3; 150 PPM
ACGIH TLV: 434 MG/M3;100 PPM
ACGIH STEL: 651 MG/M3;150 PPM
EPA Rpt Qty: 1000 LBS
DOT Rpt Qty: 1000 LBS

Cas: 78-78-4
RTECS #: EK4430000
Name: ISOPENTANE
Percent by Wt: 9.
ACGIH TLV: 1770 MG/M3; 600 PPM

Cas: 108-88-3
RTECS #: XS5250000
Name: TOLUENE
Percent by Wt: 5.
OSHA PEL: 375 MG/M3; 100 PPM
OSHA STEL: 560 MG/M3;150 PPM
ACGIH TLV: 188 MG/M3;50 PPM S
EPA Rpt Qty: 1000 LBS
DOT Rpt Qty: 1000 LBS

Cas: 95-63-6
RTECS #: DC3325000
Name: PSEUDOCUMENE
Percent by Wt: 5.
OSHA PEL: 125 MG/M3; 25 PPM
ACGIH TLV: 123 MG/M3; 25 PPM

Cas: 106-97-8
RTECS #: EJ4200000
Name: BUTANE
Percent by Wt: 4.
OSHA PEL: 1900 MG/M3; 800 PPM
ACGIH TLV: 1900 MG/M3;800 PPM

Cas: 107-83-5
RTECS #: SA2985000
Name: 2-METHYLPENTANE
Percent by Wt: 4.
ACGIH TLV: 1760 MG/M3;500 PPM
ACGIH STEL: 3500 MG/M3;1000 PPM

Cas: 109-66-0
RTECS #: RZ9450000
Name: PENTANE
Percent by Wt: 4.
OSHA PEL: 1800 MG/M3; 600 PPM
OSHA STEL: 2250 MG/M3; 750 PPM
ACGIH TLV: 1770 MG/M3;600 PPM

Cas: 25551-13-7
RTECS #: DC3220000
Name: TRIMETHYL BENZENE
Percent by Wt: 3.
OSHA PEL: 125 MG/M3; 25 PPM
ACGIH TLV: 123 MG/M3;25 PPM

Cas: 96-14-0
Name: 3-METHYLPENTANE

Percent by Wt: 2.
ACGIH TLV: 1760 MG/M3; 500 PPM
ACGIH STEL: 3500 MG/M3;1000 PPM

Cas: 71-43-2
RTECS #: CY1400000
Name: BENZENE
Percent by Wt: 2.
OSHA PEL: 1 PPM
OSHA STEL: 5 PPM
ACGIH TLV: 0.5 MG/M3;1.60 PPM
ACGIH STEL: 2.5 MG/M3; 8 PPM
EPA Rpt Qty: 10 LBS
DOT Rpt Qty: 10 LBS

Cas: 79-29-8
RTECS #: EJ9350000
Name: 2,3-DIMETHYLBUTANE
Percent by Wt: 2.
ACGIH TLV: 1760 MG/M3;500 PPM
ACGIH STEL: 3500 MG/M3;1000 PPM

Cas: 110-54-3
RTECS #: MN9275000
Name: N-HEXANE
Percent by Wt: 2.
OSHA PEL: 180 MG/M3; 50 PPM
ACGIH TLV: 176 MG/M3;50 PPM
EPA Rpt Qty: 1 LB
DOT Rpt Qty: 1 LB

Cas: 100-41-4
RTECS #: DA0700000
Name: ETHYL BENZENE
Percent by Wt: 2.
OSHA PEL: 435 MG/M3;100 PPM
OSHA STEL: 545 MG/M3; 125 PPM
ACGIH TLV: 434 MG/M3;100 PPM
ACGIH STEL: 543 MG/M3;125 PPM
EPA Rpt Qty: 1000 LBS
DOT Rpt Qty: 1000 LBS

Cas: 589-34-4
Name: 3-METHYLHEXANE
Percent by Wt: 2.
Other REC Limits: 1640 MG/M3; 400 PPM

Cas: 591-76-4
Name: 2-METHYLHEXANE
Percent by Wt: 1.
Other REC Limits: 1640 MG/M3; 400 PPM

Cas: 108-87-2
RTECS #: GV6125000
Name: METHYLCYCLOHEXANE
Percent by Wt: 1.
OSHA PEL: 1600 MG/M3;400 PPM
ACGIH TLV: 1610 MG/M3;400 PPM
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Health Hazards Data
==========
LD50 LC50 Mixture: ORAL, RATS, LD50:>2000 MG/KG.
Route Of Entry Inds - Inhalation: YES

Skin: YES
Effects of Exposure: EYE IRRITATION, RESPIRATORY IRRITATION, DIZZINESS, NAUSEA, LOSS OF CONSCIOUSNESS. SKIN IRRITATION. STUDIES CONDUCTED EXAMINING CAUSES OF DEATH OF DISTRBUTION WORKERS WITH LONG-TERM EXPOSURE TO GASOLIN E HAVE NOT FOUND ANY GASOLINE-RELATED HEALTH EFFECTS. REPORTS OF CHRONIC GASOLINE ABUSE (SUCH AS SNIFFING) AND CHRONIC MISUSE OF GASOLINE AS SOLVENT OR CLEANING AGENT HAVE REPORTED A RANGE OF NEUROLOG ICAL EFFECTS (NERVOUS SYSTEM EFFECTS), SUDDEN DEATHS FROM CARDIAC ARREST, HEMATOLOGIC CHANGES (BLOOD EFFECTS) AND LEUKEMIA. THESE EFFECTS ARE NOT EXPECTED TO OCCUR AT EXPOSURE LEVELS ENCOUNTERED IN DI STRIBUTION AND USE AS A MOTOR FUEL.
Explanation Of Carcinogenicity: LONG-TERM EXPOSURE TO GASOLINE VAPOR HAS CAUSED KIDNEY AND LIVER CANCER IN LABORATORY ANIMALS.
Signs And Symptions Of Overexposure: EYES: IRRITATION. SKIN: IRRITATION. INHALATION: RESPIRATORY IRRITATION, DIZZINESS, NAUSEA, LOSS OF CONSCIOUSNESS. CHRONIC GASOLINE ABUSE (EFFECTS NOT EXPECTED TO OCCUR AT EXPOSURE LEVELS ENCOUNTERED IN THE DISTRIBUTATION AND USE OF GASOLINE AS A MOTOR FUEL.) : NERVOUS SYSTEM EFFECTS; SUDDEN DEATH FROM CARDIAC ARREST; HEMATOLOGIC CHANGES (BLOOD EFFECTS); LUKEMIA.
First Aid: EYE CONTACT: FLUSH THOROUGHLY WITH WATER. IF IRRITATION OCCURS, CALL A PHYSICIAN. SKIN CONTACT: WASH CONTACT AREAS WITH SOAP AND WATER. REMOVE CONTAIMINTED CLOTHING. LAUNDER CONTAMINATED CLOTHING BEFO RE REUSE. INHALATION: MOVE TO FRESH AIR. IF RESPIRATORY IRRITATION, DIZZINESS, NAUSEA, OR UNCONSCIOUSNESS OCCCURS, SEEK MEDICAL ASSISTANCE. IF BREATHING STOPPED, ASSIST VENTILATION WITH BAG-VALVE-MASK DEVISE OR USE MOUTH-TO-MOUTH RESUSCITATION. INGESTION: SEEK IMMEDIATE MEDICAL ATTENTION. DO NOT INDUCE VOMITING.

Handling and Disposal

Spill Release Procedures: ELIMINATE IGNITION SOURCES. RUNOFF MAY CREATE FIRE OR EXPLOSION HAZARD IN SEWER SYSTEM. ABSORB ON FIRE RETARDANT TREATED SAWDUST, DIATOMACEOUS EARTH, ETC. SHOVEL UP AND DISPOSE. PREVENT SPILLS FROM EN TERING STORM SEWERS, DRAINS, SOIL. REPORT SPILLS AS REQUIRED TO AUTHORITIES. U.S. COAST GUARD REQUIRES IMMEDIATE REPORTING OF SPILLS THAT COULD REACH ANY WATERWAY INCLUDING INTERMITTENT (SEE BELOW).
Neutralizing Agent: (FROM ABOVE) DRY CREEKS. REPORT SPILL TO COAST GUARD TOLL FREE (800-424-8802); ROAD SPILLS NOTIFY CHEMTREC.
Waste Disposal Methods: PRODUCT IS SUITABLE FOR BURNING FOR FUEL VALUE IN COMPLIANCE WITH APPLICABLE LAWS AND REGULATIONS.DISPOSAL OF UNUSED PRODUCT MAY BE SUBJECT TO RCRA REGULATIONS (40 CFR 261) DUE TO BENZENE (2.32%, TCLP , FLASH<-40F). DISPOSAL OF USED PRODUCT MAY ALSO BE REGULATED DUE TO IGNITABILITY, CORROSIVITY, REAC TIVITY, OR TOXICITY AS DETERMINED BY TCLP.
Handling And Storage Precautions: NEVER SIPHON GASOLINE BY MOUTH AND DO NOT USE AS A SOLVENT OR CLEANING AGENT. USE NON-SPARKING TOOLS AND EXPLOSION-PROOF EQUIPMENT. USE IN WELL VENTILATED AREAS AWAY FROM IGNITION SOURCES. PORTABLE CO NTAINERS MUST BE PLACED ON GROUND AND NOZZLE KEPT IN CONTACT WHEN FILLING TO PREVENT STATIC SPARKS.
Other Precautions: DRUMS MUST BE GROUNDED AND BONDED AND EQUIPPED WITH SELF-CLOSING VALVES, PRESSURE VACUUM BUNGS, AND FLAME ARRESTERS. STORE AWAY FROM IGNITION SOURCES IN A COOL AREA EQUIPPED WITH AUTOMATIC SPRINKLING SYSTEM. OUTSIDE OR DETACHED STORAGE PREFERRED. STORAGE CONTAINERS SHOULD BE GROUNDED AND BONDED.

Fire and Explosion Hazard Information

Flash Point Method: TCC
Flash Point: <-40.C, -40.F
Lower Limits: 1.4
Upper Limits: 7.6
Extinguishing Media: CARBON DIOXIDE, FOAM, DRY CHEMICAL, WATER FOG. NFPA HAZARD ID: HEALTH: 1; FLAMMABILITY: 3; REACTIVITY: 0.
Fire Fighting Procedures: FOR FIRES IN ENCLOSED AREAS, FIRE FIGHTERS MUST USE SELF-CONTAINED BREATHING APPARATUS. FOR LARGE SPILLS, FOAM IS THE PREFERRED

AGENT; BLANKETING THE GASOLINE SURFACE. WATER SPRAY MAY BE USED TO FLUSH SPILL AWAY FROM EXPOSURE; PREVENT SPREADING GASOLINE INTO SEWERS, STREAMS, DRINKING WATER SUPPLIES.
Unusual Fire/Explosion Hazard: EXTREMELY FLAMMABLE. VAPOR ACCUMULATION COULD FLASH AND/OR EXPLODE IF IN CONTACT WITH OPEN FLAME. IF SPILL HAS NOT IGNITED, ADD FOAM BLANKET TO SUPPRESS RELEASE OF VAPORS. IF FOAM NOT AVAILABLE, A WAT ER SPRAY CURTAIN CAN BE USED TO DISPERSE VAPORS AND PROTECT PERSONNEL ATTEMPTING TO STOP THE LEAK.

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Control Measures

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Respiratory Protection: APPROVED RESPIRATORY EQUIPMENT MUST BE USED WHEN AIRBORNE CONCENTRATIONS ARE UNKNOWN OR EXCEED THE TLV.
Ventilation: USE IN WELL VENTILATED AREA WITH LOCAL EXHAUST VENTILATION. VENTILATION REQUIRED AND EQUIPMENT MUST BE EXPLOSION PROOF. USE AWAY FROM ALL IGNITION SOURCES.
Protective Gloves: IMPERVIOUS GLOVES SHOULD BE WORN.
Eye Protection: SAFETY GLASSES WITH SIDE SHIELDS OR CHEMICAL GOGGLES IF SPLASH IS POSSIBLE.
Work Hygienic Practices: GOOD PERSONAL HYGIENE PRACTICES SHOULD ALWAYS BE FOLLOWED.

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Physical/Chemical Properties

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HCC: F2
Boiling Point: >35.C, 95.F
Vapor Pres: >400.0 MMHG @20C
Vapor Density: 3.0
Spec Gravity: 0.79
Viscosity: <1.0 CST@ 40 C
Evaporation Rate & Reference: NOT ESTABLISHED
Solubility in Water: NEGLIGIBLE
Appearance and Odor: CLEAR LIQUID (MAY BE DYED), GASOLINE ODOR.

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Reactivity Data

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Stability Indicator: YES
Stability Condition To Avoid: HEAT, SPARKS, FLAME AND BUILD UP OF STATIC ELECTRICITY.
Materials To Avoid: HALOGENS, STRONG ACIDS, ALKALIES, AND OXIDIZERS.
Hazardous Decomposition Products: CARBON MONOXIDE.
Hazardous Polymerization Indicator: NO
Conditions To Avoid Polymerization: WILL NOT OCCUR.

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Toxicological Information

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Toxicological Information: ORAL, RATS, LD50: >2000 MG/KG; PRACTICALLY NON-TOXIC. DERMAL, RABBITS, LD50: > 2000 MG/KG, PRACTICALLY NON-TOXIC. INHALATION, RATS, LC50: > 5 MG/L, PRACTICALLY NON-TOXIC. EYE IRRITATION, RABBITS, DRAI ZE SCORE: >6 BUT 15 OR LESS, PRACTICALLY NON-IRRITATING. SKIN IRRITATION, RABBITS, PRIMARY IRRITATION INDEX: 3 OR > BUT < 5. IRRITANT. OTHER ACUTE TOXICITY DATA: INHALATION OF VAPORS/MISTS MAY CAUSE R ESPIRATORY SYSTEM IRRITATION. EXPOSURE TO HIGH CONCENTRATIONS OF CARBON MONOXIDE CAN CAUSE LOSS OF CONSCIOUSNESS, HEART DAMAGE, BRAIN DAMAGE, DEATH. EXPOSURE TO HIGH CONCENTRATIONS OF CARBON DIOXIDE C AN CAUSE (CONTD. SEE "ECOLOGICAL")

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Ecological Information

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Ecological: ENVIRONMENTAL FATE AND EFFECTS: NOT ESTABLISHED. NOTE: MOBIL PRODUCTS ARE NOT FORMULATED TO CONTAIN PCBS. USE: UNLEADED MOTOR FUEL. (CONTD. FROM "TOXICOLOGICAL'') ASPHYXIATION. NEUROTOXICOLOGY: NO SI GNIFICIANT ADVERSE EFFECTS IN STUDY WITH RATS. REPRODUCTIVE TOXICOLOGY: ONE

GENERATION REPRODUCTION STUDIES, SHOWED NO ADVERSE EFFECTS IN RATS. A TWO GENERATION STUDY SHOWED NO REPRODUCTIVE OR DEVELOP MENTAL EFFECTS IN RATS. A TERATOLOGY INHALATION STUDY IN RABBITS SHOWED NO DEVELOPMENTAL EFFECTS. CHRONIC TOXICOLOGY: AN INCREASE INCIDENCE OF KIDNEY AND LIVER TUMORS (CONTD. SEE "FEDERAL")

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MSDS Transport Information

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Transport Information: USA DOT: PSN: GASOLINE; CLASS: 3; UN 1203; ERG NUMBER: 128; PG I I; LABEL: FLAMMABLE LIQUID; PLACARD: FLAMMABLE; RQ: N/A. RID/ ADR: CLASS: 3;SUB-CLASS: 3(B); LABEL: 3; DANGER NUMBER: 33; UN 1203; SHIP PING NAME: HYDROCARBONS, LIQUID HAVING A FLASH POINT BELOW 21C. IMO: CLASS: 3.1; UN 1203; PG I I; SHIPPING NAME: GASOLINE; LABEL: FLAMMABLE LIQUID. ICAO/ IATA: CLASS:3; UN 1203; PG I I; SHIPPING NAME : GASOLINE; LABEL: FLAMMABLE LIQUID.

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Regulatory Information

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Sara Title III Information: THIS PRODUCT CONTAINS N0 "EXTREMELY HAZARDOUS SUBSTANCES". SARA (311 / 312) REPORTABLE HAZARD CATEGORIES: FIRE, CHRONIC, ACUTE. THIS PRODUCT CONTAINS SARA (313) TOXIC RELEASE CHEMICALS: SEE MANUFACTUR ER'S MSDS FOR TOXIC RELEASE CHEMICALS AND THE LIST CITATIONS UNDER WHICH THE LISTED INGREDIENTS ARE CITED. PRECAUTIONARY LABEL TEXT: CONTAINS GASOLINE. DANGER! SEE MANUFACTURER'S MSDS FOR PRECAUTIONAR Y LABEL TEXT.

Federal Regulatory Information: ALL COMPONENTS COMPLY WITH TSCA, AND EINECS/ ELINCS. (CONTD. FROM "ECOLOGICAL") WAS OBSERVED IN LABORATORY ANIMALS. THESE EFFECTS ARE NOT CONSIDERED SIGNIFICANT TO HUMANS. SKIN SENSITIZATION: NEGATIVE GUINEA PIG TEST. GASOLINE AND REFINERY STREAMS: STUDIES CONDUCTED BY THE AMERICAN PETROLEUM INSTITUTE EXAMINED A REFERENCE UNLEADED GASOLINE FOR MUTAGENIC, TERATOGENIC, AND SENSITIZATION POTENTIAL; N O EVIDENCE OF THESE HAZARDS WAS FOUND. AS FAR AS SCIENTISTS KNOW, LOW LEVEL OR INFREQUENT EXPOSURES TO GASOLINE VAPORS ARE UNLIKELY TO BE ASSOCIATED WITH CANCER OR OTHER SERIOUS DISEASES IN HUMANS.

State Regulatory Information: THIS WARNING IS GIVEN TO COMPLY WITH CALIFORNIA HEALTH AND SAFETY CODE 25249.6 AND DOES NOT CONSTITUTE AN ADMISSION OR A WAIVER OF RIGHTS. THIS PRODUCT CONTAINS A CHEMICAL KNOWN TO STATE OF CALIFORNIA TO CAUSE CANCER, BIRTH DEFECTS, OR OTHER REPRODUCTIVE HARM. REFER TO PRODUCT MATERIAL SAFETY DATA BULLETIN FOR FURTHER SAFETY AND HEALTH INFORMATION.

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Other Information

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Other Information: EU LABELING: SYMBOL: F+ T EXTREMELY FLAMMABLE, TOXIC; RISK PHRASE: R 12-45-38-22; EXTREMELY FLAMMABLE. MAY CAUSE CANCER. IRRITATING TO SKIN. HARMFUL IF SWALLOWED. SAFETY PHASES: S53-45-2-23-24-29-43-6 2. AVOID EXPOSURE - OBTAIN INSTRUCTIONS BEFORE USE. IN CASE OF ACCIDENT OR IF YOU FEEL UNWELL, SEEK MEDICAL ADVICE (SHOW LABEL WHERE POSSIBLE). KEEP OUT OF REACH OF CHILDREN. DO NOT BREATHE VAPOR. AV OID CONTACT WITH SKIN. DO NOT EMPTY INTO DRAINS. IN CASE OF FIRE USE CARBON DIOXIDE, FOAM, DRY CHEMICAL, WATER FOG. IF SWALLOWED, DO NOT INDUCE VOMITING: SEEK MEDICAL ADVICE AND SHOW LABEL. CONTAINS : LOW BOILING POINT NAPHTHA.

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Transportation Information

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Responsible Party Cage: 3U728
Trans ID NO: 157074
Product ID: MOBIL REGULAR UNLEADED GASOLINE
MSDS Prepared Date: 05/17/2000
Review Date: 05/23/2001
MFN: 3
Net Unit Weight: BULK
Multiple KIT Number: 0
Unit Of Issue: GL

Container QTY: X
Additional Data: TRANSPORTATION DATA PER MANUFACTURER'S MSDS.
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Detail DOT Information
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DOT PSN Code: GTN
DOT Proper Shipping Name: GASOLINE
Hazard Class: 3
UN ID Num: UN1203
DOT Packaging Group: II
Label: FLAMMABLE LIQUID
Special Provision: B33,B101,T8
Non Bulk Pack: 202
Bulk Pack: 242
Max Qty Pass: 5 L
Max Qty Cargo: 60 L
Vessel Stow Req: E
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Detail IMO Information
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IMO PSN Code: HRV
IMO Proper Shipping Name: GASOLINE
IMDG Page Number: 3141
UN Number: 1203
UN Hazard Class: 3.1
IMO Packaging Group: II
Subsidiary Risk Label: -
EMS Number: 3-07
MED First Aid Guide NUM: 311
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Detail IATA Information
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IATA PSN UC
IATA UN ID Num: 1203
IATA Proper Shipping Name: GASOLINE
IATA UN Class: 3
IATA Label: FLAMMABLE LIQUID
UN Packing Group: II
Packing Note Passenger: 305
Max Quant Pass: 5L
Max Quant Cargo: 60L
Packaging Note Cargo: 307
Exceptions: A100
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Detail AFI Information
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AFI PSN UC
AFI Proper Shipping Name: GASOLINE
AFI Hazard Class: 3
AFI UN ID NUM: UN1203
AFI Packing Group: II
Special Provisions: P5
Back Pack Reference: A7.3
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HAZCOM Label
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Product ID: MOBIL REGULAR UNLEADED GASOLINE
Cage: 3U728
Company Name: MOBIL OIL CORP, NORTH AMERICAS MARKETING AND REFINING
Street: 3225 GALLOWS ROAD
City: FAIRFAX VA
Zipcode: 22037
Health Emergency Phone: 609-737-4411

Label Required IND: Y
Date Of Label Review: 05/23/2001
Status Code: A
Label Date: 05/23/2001
Origination Code: F
Eye Protection IND: YES
Skin Protection IND: YES
Signal Word: DANGER
Respiratory Protection IND: YES
Health Hazard: Moderate
Contact Hazard: Moderate
Fire Hazard: Severe
Reactivity Hazard: None
Hazard And Precautions: DANGER ! CONTAINS GASOLINE. EXTREMELY FLAMMABLE LIQUID AND VAPOR. VAPOR MAY CAUSE FLASH FIRE. MAY CAUSE SKIN, NOSE, THROAT, LUNG IRRITATION, DIZZINESS, NAUSEA, AND LOSS OF CONSCIOUSNESS. IF SWALLOWED, MAY BE ASPIRATED AND CAN CAUSE SERIOUS LUNG DAMAGE.

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CONOCO INC -- DIESEL FUEL NO. 2, LOW/HIGH SULFUR; 3502;3504

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MSDS Safety Information

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FSC: 9140
NIIN: 00-000-0184
MSDS Date: 01/10/1994
MSDS Num: BRTDK
Product ID: DIESEL FUEL NO. 2, LOW/HIGH SULFUR; 3502;3504;3510;4152.
MFN: 01
Responsible Party
Cage: 5R396
Name: CONOCO INC
Box: 2197
City: HOUSTON TX 77252
Info Phone Number: 713-293-5550
Emergency Phone Number: 800-441-3637/800-424-9300(CHEMTREC)
Preparer's Name: MSDS ADMINISTRATOR
Review Ind: Y
Published: Y

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Preparer Co. when other than Responsible Party Co.

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Cage: 5R396
Name: CONOCO INC
Address: 5 GREENWAY PLAZA E
Box: 2197
City: HOUSTON TX 77252

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Contractor Summary

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Cage: 5R396
Name: CONOCO INC
Address: 5 GREENWAY PLAZA E
Box: 2197
City: HOUSTON TX 77252
Phone: 713-293-5550PRODUCT/ 800-4413637MED

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Item Description Information

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Item Name: USED TO BE 26648
Specification Number: VV-F-800
Type/Grade/Class: DF2,LOW SULFUR
Unit of Issue: GL
UI Container Qty: X
Type of Container: UNKNOWN

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Ingredients

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Cas: 68476-34-6
Name: DIESEL FUEL, NO. 2 (PETROLEUM MID-DISTILLATE).
% Wt: 100
Other REC Limits: NONE RECOMMENDED
OSHA PEL: NOT ESTABLISHED
ACGIH TLV: NOT ESTABLISHED

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Health Hazards Data

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LD50 LC50 Mixture: LD50 ORAL RAT = 9ML/KG
Route Of Entry Inds - Inhalation: YES
Skin: YES
Ingestion: NO
Carcinogenicity Inds - NTP: NO

IARC: NO
OSHA: NO
Effects of Exposure: MAY CAUSE IRRIT TO EYES/LUNGS/SKIN AFT PROLONG/REPEAT EXPOSURE. ASPIRATION INTO LUNGS MAY CAUSE LUNG DAMAGE & DEATH.
Explanation Of Carcinogenicity: THERE ARE NO INGREDIENTS ABOVE 0.1% WHICH ARE IDENTIFIED AS CARCINOGENS BY NTP,IARC OR OSHA.
Signs And Symptions Of Overexposure: OVEREXPOSURE MAY CAUSE WEAKNESS,HEADACHE, NAUSEA,CONFUSION,BLURRED VISION,DROWSINESS,UNSPECIFIED CNS EFFECTS.LARGE EXPOSURE MAY CAUSE DIZZINESS,SLURRED SPEECH,FLUSHED FACE,UNCONSCIOUSNESS, CONVULSIONS .STUDIES IN MICE/RATS W/CHRONIC EXPOUSRE HAVE SHOWN DIESEL EXHAUST MAY PRODUCE LUNG TUMORS AND LYMPHOMAS.
Medical Cond Aggravated By Exposure: NONE SPECIFIED BY MFG.
First Aid: INHAL:REMOVE TO FRESH AIR.GIVE OXYGEN IF BREATH DIFFI OR ARTI RESP IF NOT BRETH.CALL PHYSICIAN.SKIN:WASH W/SOAP & WATER.IF IRRIT DEVELOP/PERSIST,CALL PHYSICIAN.EYES:IMMED FLUSH W/PLENTY OF WATER FOR @ 15MINS.CALL PHYSICIAN.INGEST:DO NOT INDUCE VOMITING.IF CONSCIOUS GIVE 2 GLASSES OF WATER.PHYSICIAN:GIVE 5ML/KG (OR 350ML) OF CHARCOAL SOLUTION (50GMS CHARCOAL IN 400ML WATER).

Handling and Disposal

Spill Release Procedures: REMOVE IGNITION SOURCES.USE EXPLOSION-PROOF EQPMT & APPROPRIATE PPE.DIKE & PREVENT FROM ENTERING SEWERS/WATERWAYS.SOAK UP W/ABSORBENT MATL.(SAWDUST,SAND,OIL DRY).IF SPILLED INTO NAVIGABLE WATERS REPOR T TO NAT RESP CNTR 800-424-8802.READ MSDS.
Neutralizing Agent: NONE
Waste Disposal Methods: TREATMENT/STORAGE/TRANSP/DISPOSAL MUST BE IAW APPLICABLE FED/STATE/PROVINCIAL/LOC REGS.DO NOT FLUSH TO SURFACE WATER/SANITARY SEWER SYS.BY ITSELF LIQ IS EXPECTED TO BE RCRA IGNITABLE HAZ WASTE.CONTAIN S PETRO HYDROCARBONS-RQ(FILM/SHEEN/DISCOLO WATER.
Handling And Storage Precautions: STORE IN WELL-VENTILATED AREA.KEEP CONTAINER TIGHTLY CLOSED.STORE IAW NAT FIRE PROTECTION ASSOC RECOMMENDATIONS.STORE AWAY FROM HEAT/SPARKS/FLAME/OXID
Other Precautions: DO NOT BREATH VAPORS/MISTS.GROUND CONTAINERS WHEN TRANSFERRING LIQUID (FLOWING FUEL GENERATES STATIC ELECTRICITY).

Fire and Explosion Hazard Information

Flash Point Method: TCC
Flash Point Text: 130F,54C
Lower Limits: 0.4
Upper Limits: 6
Extinguishing Media: WATER SPRAY, FOAM, DRY CHEMICAL, CARBON DIOXIDE. NFPA CLASSIFICATION:CLASS II COMBUSTIBLE LIQUID.
Fire Fighting Procedures: DON'T ENTER ENCLOSE/CONFINE SPACE W/O PROPER PROT EQPMT INCLUDING RESP PROT.W/WATER SPRAY COOL FIRE EXPOSED CNTNRS & DISPERSE/FLUSH VAP/PROTECT FROM SPILL/LEAK.
Unusual Fire/Explosion Hazard: PRODUCTS OF COMBUSTION MAY CONTAIN CARBON MONOXIDE, CARBON DIOXIDE & OTHER TOXIC MATERIALS.

Control Measures

Respiratory Protection: SELECT APPROPRIATE NIOSH-APPROVED RESP PROTECTION WHEN NEEDED TO AVOID INHAL OF MIST/VAPORS AND TO MAINTAIN EXPOSURES BELOW ACCEPTABLE LIMITS.
Ventilation: USE ONLY WITH ADEQUATE VENTILATION. MECHANICAL (GENERAL) VENTILATION TO MAINTAIN TLV/PEL.
Protective Gloves: NEOPRENE, NBR GLOVES.
Eye Protection: SAFETY GLASSES W/SIDE SHIELDS, CHEM GOGG
Other Protective Equipment: COVERALLS IF SPLASHING IS PROBABLE.
Work Hygienic Practices: WASH HANDS AFTER HANDLING. LAUNDER CONTAMIN CLOTHES PRIOR TO REUSE.
Supplemental Safety and Health: NOT SUBJECT TO CA PROP 65. CONTAINS DIESEL FUEL

OIL SUBJECT TO PEN WORKER/COMM RIGHT TO KNOW.

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Physical/Chemical Properties

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HCC: F4
B.P. Text: 350F,177C
Vapor Pres: 1MM@68F
Vapor Density: >1
Spec Gravity: 0.84-0.88 @60C
Viscosity: 1.9CAT@40C
Solubility in Water: INSOLUBLE
Appearance and Odor: AROMATIC ODOR;LIQUID;HIGH SULFUR-GREEN;LOW SULFUR-RED OR UNDYED(CLEAR OR STRAW)
Percent Volatiles by Volume: NIL

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Reactivity Data

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Stability Indicator: YES
Stability Condition To Avoid: AVOID HEAT, SPARKS, FLAME.
Materials To Avoid: INCMPATIBLE OR CAN REAC WTIH STRONG OXIDIZERS.
Hazardous Decomposition Products: CARBON MONOXIDE, CARBON DIOXIDE, AND OTHER TOXIC MATERIALS.
Hazardous Polymerization Indicator: NO
Conditions To Avoid Polymerization: NOT RELEVANT.

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Toxicological Information

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Ecological Information

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MSDS Transport Information

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Regulatory Information

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Other Information

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Transportation Information

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Responsible Party Cage: 5R396
Trans ID NO: 43111
Product ID: DIESEL FUEL NO. 2, LOW/HIGH SULFUR; 3502;3504;3510;4152.
MSDS Prepared Date: 01/10/1994
Review Date: 09/02/1994
MFN: 1
Net Unit Weight: UNKNOWN
AF MMAC Code: NR
Multiple KIT Number: 0
Review IND: Y
Unit Of Issue: GL
Container QTY: X
Type Of Container: UNKNOWN
Additional Data: PER MSDS DOMESTIC PROPER SHIPPING NAME DIESEL FUEL, UN 1993, HAZ CLASS COMBUST LIQ. IF SHIPPED BY VE SSEL/AIR USE INTERNATIONAL DESCRIPTION WHICH IS GAS OIL, UN 1202, PACK GR III, FLAMM LIQ.

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Detail DOT Information

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DOT PSN Code: EXF
Symbols: D

DOT Proper Shipping Name: DIESEL FUEL
Hazard Class: 3
UN ID Num: NA1993
DOT Packaging Group: III
Label: NONE
Special Provision: B1
Non Bulk Pack: 203
Bulk Pack: 242
Max Qty Pass: 60 L
Max Qty Cargo: 220 L
Vessel Stow Req: A

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Detail IMO Information

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IMO PSN Code: HRR
IMO Proper Shipping Name: GAS OIL
IMDG Page Number: 3375
UN Number: 1202
UN Hazard Class: 3.3
IMO Packaging Group: III
Subsidiary Risk Label: -
EMS Number: 3-07
MED First Aid Guide NUM: 311

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Detail IATA Information

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IATA PSN TX
IATA UN ID Num: 1202
IATA Proper Shipping Name: GAS OIL
IATA UN Class: 3
IATA Label: FLAMMABLE LIQUID
UN Packing Group: III
Packing Note Passenger: 309
Max Quant Pass: 60L
Max Quant Cargo: 220L
Packaging Note Cargo: 310
Exceptions: A3

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Detail AFI Information

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AFI PSN Code: JEV
AFI Proper Shipping Name: DIESEL FUEL
AFI PSN Modifier: ,ALSO SEE GAS OIL
AFI Hazard Class: 3
AFI UN ID NUM: UN1202
AFI Packing Group: III
Special Provisions: P5
Back Pack Reference: A7.3

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HAZCOM Label

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Product ID: DIESEL FUEL NO. 2, LOW/HIGH SULFUR; 3502;3504;3510;4152.
Cage: 5R396
Company Name: CONOCO INC
Street: 5 GREENWAY PLAZA E
PO Box: 2197
City: HOUSTON TX
Zipcode: 77252
Health Emergency Phone: 800-441-3637/800-424-9300(CHEMTREC)
Label Required IND: Y
Date Of Label Review: 09/02/1994
Status Code: C
MFG Label NO: UNKNOWN

Label Date: 09/02/1994
Origination Code: F
Eye Protection IND: YES
Skin Protection IND: YES
Signal Word: WARNING
Respiratory Protection IND: YES
Health Hazard: Moderate
Contact Hazard: Moderate
Fire Hazard: Moderate
Reactivity Hazard: None
Hazard And Precautions: MAY CAUSE IRRIT TO EYES/LUNGS/SKIN AFT PROLONG/REPEAT EXPOSURE. ASPIRATION INTO LUNGS MAY CAUSE LUNG DAMAGE & DEATH. TARGET ORGANS:EYE/LUNGS/SKIN. FIRST AID: INHAL:REMOVE TO FRESH AIR.GIVE OXYGEN IF B REATH DIFFI OR ARTI RESP IF NOT BRETH.CALL PHYSICIAN.SKIN:WASH W/SOAP & WATER.IF IRRIT DEVELOP/PERSIST,CALL PHYSICIAN.EYES:IMMED FLUSH W/PLENTY OF WATER FOR @ 15MINS.CALL PHYSICIAN.INGEST:DO NOT INDUC E VOMITING.IF CONSCIOUS GIVE 2 GLASSES OF WATER.PHYSICIAN:GIVE 5ML/KG (OR 350ML) OF CHARCOAL SOLUTION (50GMS CHARCOAL IN 400ML WATER).

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FIRE-TROL HOLDINGS, L.L.C.
MATERIAL SAFETY DATA SHEET
FIRE-TROL® FIREGEL™

Fire-Trol Holdings, L.L.C.
2620 N. 37th Dr.
Phoenix, AZ 85009
(602) 262-5401
(530) 865-4932 (24 hr. number)

CAUTION

Avoid eye contact; may be irritating. Avoid unprotected exposure of the skin. Work in a ventilated area to avoid possible irritation of respiratory tract.

CLASSIFICATION: NON-HAZARDOUS

A. Product Identification. FIRE-TROL FIREGEL is an aluminum soap for use in thickening gasoline, kerosene or mineral spirits.

B. Occupational Control Procedures

1. Avoid eye contact. Wear goggles when handling.
2. Avoid skin contact. Use rubber or plastic gloves to avoid prolonged skin contact.
3. Avoid excessive inhalation of powder by wearing an OSHA approved dust mask.
4. Handle product in a well ventilated area. Permissible concentration in air of 10 mg/m3. (Nuisance dust.)
5. Avoid ingestion. (Estimated LD_{50}>50 mg/kg; oral, rat.)

C. Fire Protection Information

1. Extinguishing media for concentrate: Carbon dioxide, dry chemical, foam, or water spray. Class A, BC, or ABC fire extinguishers, sand/earth.
2. Special fire fighting procedures in enclosed areas: Fire fighters must be equipped to prevent breathing of vapors or products of combustion. Wear an approved self-contained breathing apparatus and protective clothing.
3. Unusual fire or explosion hazards: Hazardous only when present as a dust. Dust explosions can occur under conditions of high dust concentration in the presence of a spark or open flame.

D. Physical Data

1.	Color:	Greenish powder
2.	Odor:	Mild fatty
3.	Specific gravity:	1.01 to 1.03 (approximately), whereas water is 1.0
4.	Melting point:	Over 390°F
5.	pH:	5 to 6 in a 5% dispersion
6.	Percent volatile by weight:	1.5% (moisture)

E. Reactivity Data

1. Stability: Product has excellent long-term stability for an indefinite period.
2. Hazardous decomposition products: Carbon monoxide, carbon dioxide - these gases can be harmful in enclosed areas so fire fighters must wear an approved self-contained breathing apparatus and protective clothing.
3. Hazardous polymerization will not occur.
4. Incompatibility (keep away from): Flames and sparks under dusty conditions. Avoid strong acids and oxidizers.

F. Spill, Leak and Disposal Information. Sweep up and discard in closed containers. Dispose of in accordance with all applicable federal, state and local regulations.

G. Transportation Data

1. DOT: Not regulated
2. Reportable Quantity: Not applicable
3. Freight Classification: Metallic soaps of fatty acids
4. Non-hazardous, non-flammable, non-corrosive

H. Emergency & First Aid Procedures (for Concentrate)

1. Eye Contact. Flush eyes immediately with plenty of water for at least fifteen minutes and call a physician.
2. Skin Contact. Wash off with detergent and water.
3. Inhalation. Remove person to fresh air and provide oxygen if breathing is difficult. Get medical attention.
4. If swallowed, call a physician immediately.

Effective Date: January 4, 2000
Supersedes all previous dates for FIRE-TROL® FIREGEL™

MATERIAL SAFETY DATA SHEET

***** SECTION I *****

PRODUCT NAME:	SUREFIRE	SIZE:
CHEMICAL NAME:		
FORMULA:	ALUMINUM SOAPS AND OTHER COMPOUNDS	
DISTRIBUTOR:	SIMPLEX MANUFACTURING COMPANY	
ADDRESS:	13340 NE WHITAKER WAY, PORTLAND, OR 97230 USA	
FOR INFORMATION ON HEALTH HAZARDS CALL:	(503) 257-3511	
FOR OTHER INFORMATION CALL:	AS ABOVE	INFORMATION EFFECTIVE AS OF:

SECTION II
***** HAZARDOUS INGREDIENTS OF MIXTURES *****

PRINCIPAL HAZARDOUS COMPONENTS (S)	%	TLV (Units)
NONE		

SECTION III
***** PHYSICAL DATA *****

BOILING POINT (°F.)	N.A.	SPECIFIC GRAVITY (H_2O=1)	1.01-1.03
VAPOR PRESSURE (mm Hg.)	N.A.	PERCENT VOLATILE BY VOLUME (%)	5
VAPOR DENSITY (AIR=1)	N.A.	EVAPORATION RATE (=1)	N.A.
SOLUBILITY IN WATER	INSOLUBLE		
APPEARANCE AND ODOR	TAN POWDER WITH CHARACTERISTIC ODOR		

SECTION IV
***** FIRE AND EXPLOSION HAZARD DATA *****

FLASH POINT (Method used)	FLAMMABLE LIMIT	Lel	Uel

EXTINGUISHING MEDIA: CO_2, H_2O

SPECIAL FIRE FIGHTING PROCEDURES
NONE

UNUSUAL FIRE & EXPLOSION HAZARDS
AVOID HIGH DUST CONCENTRATIONS

SECTION V
***** HEALTH HAZARD DATA *****

THRESHOLD LIMIT VALUE

NOT AVAILABLE

EFFECTS OF OVEREXPOSURE

NOT AVAILABLE: BUT TOXICITY WOULD PROBABLY BE VERY LOW

EMERGENCY AND FIRST AID PROCEDURES

EYE: AS FOR NUISANCE DUST.

SKIN CONTACT: WASH WITH SOAP AND WATER; (VERY LOW PROBABILITY OF DERMATITIS)

SECTION VI
***** REACTIVITY DATA *****

STABILITY	INSTABLE		CONDITIONS TO AVOID
	STABLE	X	
INCOMPATIBILITY (Materials to avoid)	STRONG ACIDS, OXIDIZERS		

HAZARDOUS DECOMPOSITION PRODUCTS

CO_x

HAZARDOUS POLYMERIZATION		CONDITIONS TO AVOID
May Occur	Will Not Occur	
	X	

SECTION VII
***** SPILL OR LEAK PROCEDURES *****

STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED

SWEEP UP

WASTE DISPOSAL METHOD

LAND FILL (AS PER LOCAL REGULATIONS)

SECTION VIII
***** SPECIAL PROTECTION INFORMATION *****

RESPIRATORY PROTECTION (Specify type)	DUST MASK		
VENTILATION	LOCAL EXHAUST		SPECIAL NONE
	MECHANICAL (general) ACCEPTABLE		OTHER NONE
PROTECTIVE GLOVES	GLOVES	EYE PROTECTION	GOGGLES
OTHER PROTECTIVE EQUIPMENT	NONE		

SECTION IX
***** SPECIAL PRECAUTIONS *****

PRECAUTIONS TO BE TAKEN IN HANDLING AND STORING

NO SPECIAL PRECAUTIONS

OTHER PRECAUTIONS

TAKE REASONABLE CAUTION, AND PRACTICE PERSONAL CLEANLINESS

MATERIAL SAFETY DATA SHEET

SECTION I

HMIS
HEALTH: 2
FLAMMABILITY: 1
REACTIVITY: 1
PERSONAL PROT: 2

SIMPLEX

SIMPLEX MANUFACTURING CO.
13340 NE Whitaker Way
Portland, Oregon
97230

Emergency Telephone Number (503) 257 -3511

Chemical Name and Synonyms

Trade Name and Synonyms SURE-FIRE II

Chemical Family ALUMINUM CARBOXYLATES Formula

SECTION II — INGREDIENTS

MATERIAL	CAS NO.	%	TLV (UNITS)	MATERIAL	CAS NO.	%	TLV (UNITS)
Aluminum Soaps	NA	95	NA				
Others	NA	5	NA				

SECTION III — PHYSICAL DATA

Boiling Point (°F)	NA	Specific Gravity (H_2O=1)	>1.0
Vapor Pressure (mm Hg.)	NA	Percent Volatile by Volume (%)	1.0
Vapor Density (AIR=1)	NA	Evaporation Rate (Butyl Acetate=1)	<<<1.0
Solubility in Water	Negligible		

Appearance and Color
Off white powder

SECTION IV — FIRE AND EXPLOSION HAZARD DATA

Flash Point (Method Used)	Flammable Limits	Lel	Uel
NA	NA		

Extinguishing Media
WATER, CO_2, FOG, DRY CHEMICAL

Special Fire Fighting Procedures
NA

Unusual Fire and Explosion Hazards
AS A FINE DUST IN AIR, IT MAY BE A HAZARD WITH AN IGNITION SOURCE.

SECTION V — HEALTH HAZARD DATA

PRIMARY ROUTE OF ENTRY: INHALATION

eshold Limit Value:
NOT LISTED BY ACGIH

ects of Overexposure:
MAY CAUSE EYE AND LUNG IRRITATION

ergency First Aid Procedures:
WASH WITH SOAP AND WATER. FLUSH EYES WITH WATER.

SECTION VI — REACTIVITY DATA

bility	Unstable	Conditions to Avoid:
	Stable XX	AVOID HIGH DUST CONCENTRATIONS

ompatability (Materials to Avoid):
STRONG OXIDIZERS, STRONG ACIDS

:ardous Decomposition Products:
CO_2, NORMAL COMBUSTION PRODUCTS OF ORGANIC MATERIAL

ardous Polymerization	May Occur	Conditions to Avoid:
	Will Not Occur XX	NONE

SECTION VII — SPILL OR LEAK PROCEDURE

ps to be Taken in Case Material is Released or Spilled:
REMOVE IGNITION SOURCES. SHOVEL INTO DRUMS.

ste Disposal Method:
DISPOSE OF CONSISTENT WITH LOCAL, STATE AND FEDERAL REGULATIONS.

SECTION VIII — SPECIAL PROTECTION INFORMATION

iratory Protection (Specify Type):
NIOSH APPROVED DUST MASK.

ilation	Local Exhaust	Special
	SUFFICIENT TO CONTROL DUST.	
	Mechanical (General)	Other

ective Gloves:
RUBBER

Eye Protection:
CHEMICAL GOGGLES

er Protective Equipment:
IMPERVIOUS OUTERWEAR

SECTION IX — SPECIAL PRECAUTIONS

autions to be taken in Handling and Storing:
STORE IN COOL, DRY PLACE

er Precautions: DUST EXPLOSIONS CAN OCCUR UNDER CONDITIONS OF HIGH DUST CONCENTRATION IN PRESENCE OF AN IGNITION SOURCE.

OCTOBER 19, 199C

This information is furnished without warranty of any kind. Employers should use this information only as a supplement to other information gathered, and must make independent determinations of suitability and completeness from all sources to assure use of these materials and the safety and health of employees.

Potassium Permanganate ($KMnO_4$)

Material Safety Data Sheet

U.S. DEPARTMENT OF LABOR
Occupational Safety and Health Administration
Required under USDL Safety and Health Regulations for Ship Repairing, Shipbuilding, and Shipbreaking (29 CFR 1915, 1916, 1917)

Form Approved
OMB No. 44-R1387

Section I

MANUFACTURER'S NAME: CARUS CHEMICAL COMPANY		EMERGENCY TELEPHONE NO.: 815/223-1500	
ADDRESS: 1500 Eighth Street – LaSalle, IL 61301			
CHEMICAL NAME AND SYNONYMS: Potassium Permanganate		CAS Registry No. 7722-64-7	
CHEMICAL FAMILY: Oxidizer	TRADE NAME AND SYNONYMS: CAIROX®	FORMULA: $KMnO_4$	

Section II Hazardous Ingredients

	%	TLV (Units)
Section II is not applicable since product is a single compound – Potassium Permanganate.	98	by wt

Section III Physical Data

BOILING POINT (°F.): 302°F	Decomposes with evolution of O_2	SPECIFIC GRAVITY (H_2O=1): 2.7	
SOLUBILITY IN WATER: 6.5 g/100mL water at 20°C (68°F)			

Section IV Fire and Explosion Hazard Data

FLASH POINT (Method used): Not Flammable	EXTINGUISHING MEDIA: Water

SPECIAL FIRE FIGHTING PROCEDURES
Use plenty of water. Watch for rapid burning and be prepared to retreat to a safe distance. If yellow-brown fumes are present, wear a positive pressure, self-contained breathing apparatus.

UNUSUAL FIRE AND EXPLOSION HAZARDS:
May decompose spontaneously if exposed to intense heat, concentrated acids, hydrogen peroxide, reducing agents, or organic substances generally. In confined areas, this decomposition may become explosive.

Section V Health Hazard Data

THRESHOLD LIMIT VALUE
Manganese and its inorganic compounds as Mn C 5 mg/m3 (C=ceiling value).

EFFECTS OF OVEREXPOSURE
Prolonged inhalation of manganese in the form of its inorganic compounds may cause manganism.

EMERGENCY AND FIRST AID PROCEDURES

BODY: Wash contaminated areas with copious amounts of water.

EYES: Flood with water for 15 minutes, holding the eyelids open. Consult physician immediately. Do NOT attempt to neutralize chemically.

INTERNAL: Give lemon or orange juice to drink; in addition, a milk or sugar solution may be given. If none of these are available, give large quantities of water to drink. Consult physician.

(Continued on reverse side)

Section VI Reactivity Data

STABILITY	CONDITIONS TO AVOID
Stable	Exposure to incompatible materials or heat.

INCOMPATIBILITY (Materials to avoid)
Including but not limited to alcohols, arsenites, iodides, acids, charcoal, organic substances generally, ferrous or mercurous salts, hypophosphites, hyposulfites, sulfites, peroxides, oxalates.

HAZARDOUS DECOMPOSITION PRODUCTS	HAZARDOUS POLYMERIZATION
With hydrochloric acid, chlorine is liberated.	Will not occur

Section VII Spill or Leak Procedures

STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED

Sweep up and remove as much as $KMnO_4$ as possible; do not return contaminated material to original drum; transfer to clean metal drum and dispose in hazardous landfill; flush floor with abundant quantities of water into sewer, if permitted by Federal, State and Local regulations; if not, treat chemically (see below).

WASTE DISPOSAL METHOD

Reduce $KMnO_4$ solution with sodium thiosulfate solution, mix the sludge with soda ash (Na_2CO_3) and deposit in an approved landfill. Where permitted, the sludge can be drained into a sewer with large quantities of water.

Section VIII Special Protection Information

RESPIRATORY PROTECTION (Specify type)	VENTILATION
Mechanical (General)	For dust, use a NIOSH approved dust mask.
PROTECTIVE GLOVES Rubber or Plastic Gloves	**EYE PROTECTION** Goggles/Face Shield

Section IX Special Precautions

PRECAUTIONS TO BE TAKEN IN HANDLING AND STORING

Store in a cool, dry area in closed containers separate from organics, concentrated acids, peroxides, ammonium compounds, metallic powders, elemental sulfur, phosphorous, carbon, metal hydrides, hydrazine, hydroxylamines.

OTHER PRECAUTIONS

DOT class; oxidizer; reportable quantity – 100 lb;

RCRA; Oxidizers such as potassium permanganate meet the criteria of ignitable waste.

carus CHEMICAL COMPANY

Division of Carus Corporation
1500 Eighth Street • LaSalle, Illinois 61301
Telephone: (815) 223-1500 Cable: Carchemco
Telex: 404452

MSDS Number: **E5125** * * * * * *Effective Date:* ***11/02/01*** * * * * * *Supercedes:* ***02/25/99***

ETHYLENE GLYCOL

1. Product Identification

Synonyms: 1,2-Ethanediol; glycol; 1,2-Dihydroxyethane; Ethylene Alcohol; Ethulene Dihydrate
CAS No.: 107-21-1
Molecular Weight: 62.07
Chemical Formula: CH2OHCH2OH
Product Codes:
J.T. Baker: 5387, 5845, 9140, 9298, 9300, 9346, 9349, 9356, L715
Mallinckrodt: 5001, 5037

2. Composition/Information on Ingredients

Ingredient	CAS No	Percent	Hazardous
Ethylene Glycol	107-21-1	99 - 100%	Yes

3. Hazards Identification

Emergency Overview

WARNING! HARMFUL OR FATAL IF SWALLOWED. HARMFUL IF INHALED OR ABSORBED THROUGH SKIN. MAY CAUSE ALLERGIC SKIN REACTION. MAY CAUSE IRRITATION TO SKIN, EYES, AND RESPIRATORY TRACT. AFFECTS CENTRAL NERVOUS SYSTEM.

J.T. Baker SAF-T-DATA(tm) Ratings (Provided here for your convenience)

Health Rating: 2 - Moderate

Flammability Rating: 1 - Slight
Reactivity Rating: 1 - Slight
Contact Rating: 2 - Moderate
Lab Protective Equip: GOGGLES; LAB COAT; VENT HOOD; PROPER GLOVES
Storage Color Code: Orange (General Storage)

Potential Health Effects

Inhalation:
Vapor inhalation is generally not a problem unless heated or misted. Exposure to vapors over an extended time period has caused throat irritation and headache. May cause nausea, vomiting, dizziness and drowsiness. Pulmonary edema and central nervous system depression may also develop. When heated or misted, has produced rapid, involuntary eye movement and coma.
Ingestion:
Initial symptoms in massive dosage parallel alcohol intoxication, progressing to CNS depression, vomiting, headache, rapid respiratory and heart rate, lowered blood pressure, stupor, collapse, and unconsciousness with convulsions. Death from respiratory arrest or cardiovascular collapse may follow. Lethal dose in humans: 100 ml (3-4 ounces).
Skin Contact:
Minor skin irritation and penetration may occur.
Eye Contact:
Splashes may cause irritation, pain, eye damage.
Chronic Exposure:
Repeated small exposures by any route can cause severe kidney problems. Brain damage may also occur. Skin allergy can develop. May damage the developing fetus.
Aggravation of Pre-existing Conditions:
Persons with pre-existing skin disorders, eye problems, or impaired liver, kidney, or respiratory function may be more susceptible to the effects of this substance.

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4. First Aid Measures

Inhalation:
Remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Call a physician.
Ingestion:
Induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. Get medical attention.
Skin Contact:
Remove any contaminated clothing. Wash skin with soap and water for at least 15

minutes. Get medical attention if irritation develops or persists.
Eye Contact:
Immediately flush eyes with plenty of water for at least 15 minutes, lifting lower and upper eyelids occasionally. Get medical attention immediately.

Note to Physician:
Give sodium bicarbonate intravenously to treat acidosis. Urinalysis may show low specific gravity, proteinuria, pyuria, cylindruria, hematuria, calcium oxide, and hippuric acid crystals. Ethanol can be used in antidotal treatment but monitor blood glucose when administering ethanol because it can cause hypoglycemia. Consider infusion of a diuretic such as mannitol to help prevent or control brain edema and hemodialysis to remove ethylene glycol from circulation.

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5. Fire Fighting Measures

Fire:
Flash point: 111C (232F) CC
Autoignition temperature: 398C (748F)
Flammable limits in air % by volume:
lel: 3.2; uel: 15.3
Slight to moderate fire hazard when exposed to heat or flame.
Explosion:
Above flash point, vapor-air mixtures are explosive within flammable limits noted above. Containers may explode when involved in a fire.
Fire Extinguishing Media:
Dry chemical, foam or carbon dioxide. Water or foam may cause frothing. Water spray may be used to extinguish surrounding fire and cool exposed containers. Water spray will also reduce fume and irritant gases.
Special Information:
In the event of a fire, wear full protective clothing and NIOSH-approved self-contained breathing apparatus with full facepiece operated in the pressure demand or other positive pressure mode. Toxic gases and vapors may be released if involved in a fire.

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6. Accidental Release Measures

Ventilate area of leak or spill. Remove all sources of ignition. Wear appropriate personal protective equipment as specified in Section 8. Isolate hazard area. Keep unnecessary and unprotected personnel from entering. Contain and recover liquid when possible. Use non-sparking tools and equipment. Collect liquid in an appropriate container or absorb with an inert material (e. g., vermiculite, dry sand, earth), and place in a chemical waste container. Do not use combustible materials, such as saw dust. Do not flush to sewer! US Regulations (CERCLA) require reporting spills and releases to soil, water and air in excess of reportable quantities. The toll free number for the US Coast Guard National Response

Center is (800) 424-8802.

7. Handling and Storage

Keep in a tightly closed container, stored in a cool, dry, ventilated area. Protect against physical damage. Separate from acids and oxidizing materials. Containers of this material may be hazardous when empty since they retain product residues (vapors, liquid); observe all warnings and precautions listed for the product.

8. Exposure Controls/Personal Protection

Airborne Exposure Limits:
-OSHA Permissible Exposure Limit (PEL):
50 ppm Ceiling

-ACGIH Threshold Limit Value (TLV):
50 ppm Ceiling (vapor)
Ventilation System:
A system of local and/or general exhaust is recommended to keep employee exposures below the Airborne Exposure Limits. Local exhaust ventilation is generally preferred because it can control the emissions of the contaminant at its source, preventing dispersion of it into the general work area. Please refer to the ACGIH document, *Industrial Ventilation, A Manual of Recommended Practices*, most recent edition, for details.
Personal Respirators (NIOSH Approved):
If the exposure limit is exceeded, a half-face respirator with an organic vapor cartridge and particulate filter (NIOSH type P95 or R95 filter) may be worn for up to ten times the exposure limit or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest. A full-face piece respirator with an organic vapor cartridge and particulate filter (NIOSH P100 or R100 filter) may be worn up to 50 times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest. Please note that N series filters are not recommended for this material. For emergencies or instances where the exposure levels are not known, use a full-face piece positive-pressure, air-supplied respirator. WARNING: Air-purifying respirators do not protect workers in oxygen-deficient atmospheres.
Skin Protection:
Wear protective gloves and clean body-covering clothing.
Eye Protection:
Use chemical safety goggles. Maintain eye wash fountain and quick-drench facilities in work area.

9. Physical and Chemical Properties

Appearance:
Clear oily liquid.
Odor:
Odorless.
Solubility:
Miscible in water.
Specific Gravity:
1.1 @20C/4C
pH:
No information found.
% Volatiles by volume @ 21C (70F):
100
Boiling Point:
197.6C (388F)
Melting Point:
-13C (9F)
Vapor Density (Air=1):
2.14
Vapor Pressure (mm Hg):
0.06 @ 20C (68F)
Evaporation Rate (BuAc=1):
No information found.

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10. Stability and Reactivity

Stability:
Stable under ordinary conditions of use and storage.
Hazardous Decomposition Products:
Carbon dioxide and carbon monoxide may form when heated to decomposition. May produce acrid smoke and irritating fumes when heated to decomposition.
Hazardous Polymerization:
Will not occur.
Incompatibilities:
Strong oxidizing agents. Reacts violently with chlorosulfonic acid, oleum, sulfuric acid, perchloric acid. Causes ignition at room temperature with chromium trioxide, potassium permanganate and sodium peroxide; causes ignition at 212F(100C) with ammonium dichromate, silver chlorate, sodium chloride and uranyl nitrate.
Conditions to Avoid:
Heat, flames, ignition sources, water (absorbs readily) and incompatibles.

---

11. Toxicological Information

Toxicological Data:
Oral rat LD50: 4700 mg/kg; skin rabbit LD50: 9530 mg/kg.
Irritation - skin rabbit: 555mg(open), mild; eye rabbit: 500mg/24H, mild.
Investigated as a tumorigen, mutagen, reproductive effector.
Reproductive Toxicity:
Has shown teratogenic effects in laboratory animals.

```
  --------\Cancer Lists\-----------------------------------------------------
-
                                          ---NTP Carcinogen---
  Ingredient                              Known    Anticipated    IARC
Category
  ----------------------------------   -----    -----------    ------------
-
  Ethylene Glycol (107-21-1)             No          No            None
```

12. Ecological Information

Environmental Fate:
When released into the soil, this material is expected to readily biodegrade. When released into the soil, this material is expected to leach into groundwater. When released into the soil, this material is not expected to evaporate significantly. When released into water, this material is expected to readily biodegrade. When released into the water, this material is expected to have a half-life between 1 and 10 days. This material is not expected to significantly bioaccumulate. This material has a log octanol-water partition coefficient of less than 3.0. When released into water, this material is not expected to evaporate significantly. When released into the air, this material is expected to be readily degraded by reaction with photochemically produced hydroxyl radicals. When released into the air, this material is expected to have a half-life between 1 and 10 days.
Environmental Toxicity:
The LC50/96-hour values for fish are over 100 mg/l.

13. Disposal Considerations

Whatever cannot be saved for recovery or recycling should be managed in an appropriate and approved waste disposal facility. Processing, use or contamination of this product may change the waste management options. State and local disposal regulations may differ from federal disposal regulations. Dispose of container and unused contents in accordance with federal, state and local requirements.

14. Transport Information

Not regulated.

15. Regulatory Information

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  --------\Chemical Inventory Status - Part 1\-------------------------------
--
  Ingredient                                      TSCA  EC   Japan
Australia
  ----------------------------------------------  ----  ---  -----  --------
-
  Ethylene Glycol (107-21-1)                       Yes  Yes   Yes      Yes

  --------\Chemical Inventory Status - Part 2\-------------------------------
--
                                                         --Canada--
  Ingredient                                      Korea  DSL   NDSL  Phil.
  ----------------------------------------------  -----  ---   ----  -----
  Ethylene Glycol (107-21-1)                       Yes   Yes   No     Yes

  --------\Federal, State & International Regulations - Part 1\--------------
--
                                          -SARA 302-    ------SARA 313----
--
  Ingredient                              RQ    TPQ     List  Chemical
Catg.
  ----------------------------------------  ---   -----   ----  ------------
--
  Ethylene Glycol (107-21-1)              No    No      Yes        No

  --------\Federal, State & International Regulations - Part 2\--------------
--
                                                    -RCRA-    -TSCA-
  Ingredient                              CERCLA    261.33     8(d)
  ----------------------------------------  ------    ------    ------
  Ethylene Glycol (107-21-1)              5000      No         No


Chemical Weapons Convention:  No     TSCA 12(b):  No     CDTA:  No
SARA 311/312:  Acute: Yes      Chronic: Yes  Fire: No  Pressure: No
Reactivity: No          (Pure / Liquid)
```

Australian Hazchem Code: None allocated.
Poison Schedule: None allocated.
WHMIS:
This MSDS has been prepared according to the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all of the information required by the CPR.

16. Other Information

NFPA Ratings: Health: **1** Flammability: **1** Reactivity: **0**

Label Hazard Warning:

WARNING! HARMFUL OR FATAL IF SWALLOWED. HARMFUL IF INHALED OR ABSORBED THROUGH SKIN. MAY CAUSE ALLERGIC SKIN REACTION. MAY CAUSE IRRITATION TO SKIN, EYES, AND RESPIRATORY TRACT. AFFECTS CENTRAL NERVOUS SYSTEM.

Label Precautions:

Do not breathe vapor or mist.
Use only with adequate ventilation.
Keep container closed.
Avoid contact with eyes, skin and clothing.
Wash thoroughly after handling.

Label First Aid:

If inhaled, remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. In case of contact, immediately flush skin or eyes with plenty of water for at least 15 minutes. Call a physician if irritation develops or persists. If swallowed, give water or milk to drink and induce vomiting. Never give anything by mouth to an unconscious person. In all cases call a physician.

Product Use:

Laboratory Reagent.

Revision Information:

MSDS Section(s) changed since last revision of document include: 8.

Disclaimer:

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Prepared by: Environmental Health & Safety
Phone Number: (314) 654-1600 (U.S.A.)

MATERIAL SAFETY DATA SHEET (MSDS)	HEALTH = 1 SLIGHT FLAMMABILITY = 3 SEVERE REACTIVITY = 0 NONE

SECTION I	CRITICAL NUMBERS
TRADE NAME **Fire Quick Flares Hot Shot", Sure Shot", Stubby , 2 ½-Inch"**	**EMERGENCY TELEPHONE** **(800) 535-5053**
Proper Shipping Name Flammable Solid, Inorganic, n.o.s (Aluminum Powder)	**DOT Classification** UN 3178, 4.1, Packaging Group III
Manufacture Name **Quoin International, Inc.**	**MANUFACTURER PHONE** (760) 446-4052
Address 1331 N. Inyo, Ridgecrest, CA 93555	

SECTION II HAZARDOUS INGREDIENTS		WEIGHT (grams per flare)
Aluminum	(CAS # 7429-90-5)	20 - 60
Calcium Sulfate (di-hydride)		10 - 70
Iron Oxide	(CAS # 1309-37-1)	4 - 10
Copper Oxide	(CAS # 1317-39-1)	1
Silicon		.1
Potassium Perchlorate	(CAS#07778-74-7)	.1
Lead Oxide		.1

SECTION III PHYSICAL DATA FOR MATERIAL			
Boiling Point	N/A	Specific Gravity	1.43
Vapor Pressure	N/A	Physical State	Solid
Solubility in Water	Insoluble	pH	Neutral
Color	Gray	Odor Threshold	N/A

SECTION IV FIRE AND EXPLOSION HAZARD DATA	
Flash Point Self Ignites at 800 ^{0}F	**UEL:** N/A **LEL:** N/A
MEANS OF EXTINCTION: FLOODING WITH WATER, DRY SAND, CARBON DIOXIDE. FLARES CAN IGNITE IF PACKAGING IS BURNING. LARGE AMOUNTS OF WATER PREVENT FLARE IGNITION IF PACKAGING IS BURNING.	
SPECIAL PROCEDURES: FIRE QUICK FLARES ARE PACKAGED IN BOXES OF 10 TO 50 EACH. FLARES ARE REACTIVE PRODUCING MOLTEN MATERIALS IN EXCESS OF 4000 ^{0}F AND LOCALIZED SMOKE. HEAT/FLAME IS THE ONLY KNOWN IGNITION SOURCE. FLOOD WITH WATER TO EXTINGUISH, DON'T INHALE FUMES	
EXPLOSIVE HAZARD: FIARES WILL NOT EXPLODE. DELAY FUSE IS PROTECTED BY SEAL, WHICH IS BROKEN BY THE OPERATOR BEFORE USE. FUSE LIGHTS FLARE RESULTING IN AGRESSIVE BURNING, MAY CAUSE FLARE MOTION.	**HAZARDOUS COMBUSTION PRODUCTS**: REACTION PRODUCES SUPERHEATED CALCIUM SULFIDE, WHICH REACTS WITH WATER TO PRODUCE HYDROGEN SULFIDE GAS. HUMAN EXPOSURE TO H_2S @ 800-1000 PPM FOR 30 MIN. MAY BE FATAL.

SECTION V REACTIVITY DATA
STABILITY: STABLE UNDER NORMAL CONDITIONS OF USE.
INCOMPATIBLE CONDITIONS: AVOID TEMPERATURE ABOVE 800 ^{0}F. STRONG ACIDS.
HAZARDOUS DECOMPOSITION PRODUCTS: CALCIUM SULFIDE

SECTION VI HEALTH DATA	
ROUTE OF ENTRY:	INHALATION: YES, OF COMBUSTION PRODUCTS INGESTION: N/A EYE CONTACT: YES, OF COMBUSTION PRODUCTS SKIN ABSORBTION: NO
SYMPTOM/EFFECT OF OVEREXPOSURE: THE FLARE CAN BE HANDLED IN ITS PACKAGED STATE WITH NO RISK OF EXPOSURE. PRIMARY HAZARD IS EXPOSURE TO HOT COMBUSTION PRODUCTS CAUSING BURNS TO SKIN AND EYES. INHALATION OF COMBUSTION PRODUCTS IS PRIMARY ROUTE OF ENTRY TO HUMANS. THESE PRODUCTS CONTAIN CALCIUM OR HYDROGEN SULFIDE WHICH IS IRRATANT TO EYES AND MUCUS MEMBRANES. **CARCINOGENICITY:** NONE KNOWN	
MEDICAL CONDITIONS GENERALLY AGRAVATED BY EXPOSURE: **FLARE**: NONE KNOWN **COMBUSTION REACTION:** PROTECT EYES FROM "FLASH" DURING REACTION. SKIN BURNS CAN RESULT IF EXPOSED TO HOT COMBUSTION PRODUCTS. **INHALATION:** PRE-EXISTING UPPER RESPRITORY AND LUNG DISEASES SUCH AS, BUT NOT LIMITED TO, BRONCHITIS, PHYSERA, AND ASTHMA.	

SECTION VII PREVENTIVE MEASURES
GLOVES: SUITABLE FOR PROTECTION FROM POTENTIAL HIGH TEMPERATURE PRODUCTS
EYEWARE: SAFETY GLASSES AND/OR GOGGLES SHOULD BE WORN TO PROTECT FROM COMBUSTION MATERIALS.
VENTILATION: FLARE IS TO BE USED IN OUTDOOR ENVIRONMENT ONLY. FLARE IGNITION NO CLOSER THAN 20 FEET TO HUMAN IS REQUIRED.
SPILLAGE: IF FLARES ARE CRUSHED DURING HANDLING, CAREFULLY PICK UP DAMAGED FIARE AND DISCARD IN APPROVED LANDFILL OR INCINERATE.

SECTION VIII SPECIAL PRECAUTIONS
STORE MATERIAL IN A COOL DRY AREA AND RESTRICT SMOKING AND OPEN FLAMES. DO NOT BREATHE SMOKE GENERATED DURING REACTION.

APPROVAL:  CEO DATE: 8/12/1998

The information contained herein has been developed based on current available data. New information may be developed from time to time which may render the conclusions of this report obsolete. Therefore, no warranty of any kind is made with respect hereto. Since the Company shall have no control of the use of the product described herein, the Company assumes no liability for loss or damage incurred from the proper or improper use of the product.

MATERIAL SAFETY DATA SHEET
EQUILON MSDS: 00436ET 01/04/99

TEXACO PROPANE
TELEPHONE NUMBER:
24 HOUR EMERGENCY ASSISTANCE — GENERAL MSDS ASSISTANCE
EQUIVA SERVICES: 877-276-7283 — 877-276-7285
CHEMTREC: 800-424-9300
NAME AND ADDRESS:
EQUILON ENTERPRISES LLC
PRODUCT STEWARDSHIP
P.O. BOX 674414
HOUSTON, TX 77267-4414

LEGEND:
N.D. - NOT DETERMINED N.A. - NOT APPLICABLE N.T- NOT TESTED
< - LESS THAN > - GREATER THAN

1.NAME

MATERIAL IDENTITY
Product Code and Name:
00436 TEXACO PROPANE
Chemical Name and/or Family or Description:
Aliphatic Hydrocarbon

2. COMPOSITION/INFORMATION ON INGREDIENTS

THE CRITERIA FOR LISTING COMPONENTS IN THE COMPOSITION SECTION IS AS FOLLOWS: CARCINOGENS ARE LISTED WHEN PRESENT AT 0.1 % OR GREATER; COMPONENTS WHICH ARE OTHERWISE HAZARDOUS ACCORDING TO OSHA ARE LISTED WHEN PRESENT AT 1.0 % OR GREATER; NON-HAZARDOUS COMPONENTS ARE LISTED AT 3.0 % OR GREATER. THIS IS NOT INTENDED TO BE A COMPLETE COMPOSITIONAL DISCLOSURE. REFER TO SECTION 14 FOR APPLICABLE STATES' RIGHT TO KNOW AND OTHER REGULATORY INFORMATION.

Product and/or Component(s) Carcinogenic According to:

OSHA	IARC	NTP	OTHER	NONE
				X

Composition: (Sequence Number and Chemical Name)

Seq.	Chemical Name	CAS Number	Range in %
	This product may be odorized. The odorant content may vary from 0-50 ppm; common odorants include mercaptans and thiopane.		
01 *	Propane	74-98-6	100.00

PRODUCT IS HAZARDOUS ACCORDING TO OSHA (1910.1200).
* COMPONENT IS HAZARDOUS ACCORDING TO OSHA.

Exposure Limits referenced by Sequence Number in the Composition Section

Seq.	Limit	
01	1000	ppm TWA-OSHA
01	-	- ASPHYXIANT (ACGIH)

3. HAZARD IDENTIFICATION

EMERGENCY OVERVIEW
Appearance:
Gas
Odor:
If odorized will have rotten egg odor - otherwise, odorless

WARNING STATEMENT

DANGER ! FLAMMABLE GAS - MAY CAUSE FLASH FIRE
DELAYED EVAPORATION FROM CONTAMINATED CLOTHING MAY BE A FIRE HAZARD

LIQUID MAY CAUSE FROSTBITE
MAY CAUSE DIZZINESS AND DROWSINESS
GAS REDUCES OXYGEN AVAILABLE FOR BREATHING
GAS MAY ACCUMULATE IN CONFINED SPACES AND CAUSE SUFFOCATION

HMIS			NFPA		
Health:	1	Reactivity: 0	Health:	1	Reactivity: 0
Flammability:	4	Special : -	Flammability:	4	Special : -

POTENTIAL HEALTH EFFECTS

	EYE	SKIN	INHALATION	INGESTION
Primary Route of Exposure:	X	X	X	

EFFECTS OF OVEREXPOSURE

Acute:

Eyes:

Eye contact with liquid product or gas under pressure can cause frostbite (cold burns).

Skin:

Brief contact is not irritating.

Product is a gas - not expected to be absorbed through the skin.

Skin contact with liquid product can cause frostbite (cold burns).

Inhalation:

Gas may be irritating and cause discomfort in nose and throat, nasal discharge, and coughing. Prolonged overexposure may cause difficulty breathing.

Inhalation may cause dizziness, drowsiness, euphoria, loss of coordination, disorientation, headache, nausea, and vomiting. In poorly ventilated areas or confined spaces, unconsciousness and asphyxiation may result.

Ingestion:

Product is a gas - not expected to cause toxic effects due to ingestion.

This material is a gas. Gas or liquid under pressure may cause frostbite (cold burns).

Sensitization Properties:

Unknown.

Chronic:

No adverse effects have been documented in humans as a result of chronic exposure. Section 11 may contain applicable animal data.

Medical Conditions Aggravated by Exposure:

There is no evidence that this product aggravates an existing medical condition.

Other Remarks:

If purchased for consumer use, contains or may release alkyl mercaptans (e.g., methyl mercaptan, ethyl mercaptan). Mercaptan concentrations above permissible concentrations can cause headache, dizziness, nausea, vomiting, and diarrhea. At concentrations above 400 ppm, respiratory paralysis, causing unconsciousness and death can occur.

4. FIRST AID MEASURES

Eyes:

Flush eyes with plenty of water for several minutes. Get medical attention if eye irritation persists.

Skin:

Wash skin with plenty of soap and water for several minutes. Get medical attention if skin irritation develops or persists.

In case of cold burn, immediately place affected area in warm water (105 F) and keep at this temperature until circulation returns. Get medical attention.

If clothing becomes wetted, drench individual with water and remove contaminated clothing if possible. Slowly warm affected area of skin.

Ingestion:

No emergency care anticipated. This material is a gas at standard temperature and pressure.

Inhalation:

If inhaled, remove to fresh air. If not breathing, clear person's airway and give artificial respiration. If breathing is difficult, qualified medical personnel may administer oxygen. Get medical attention immediately.

Other Instructions:

Overexposure to this material may sensitize the heart to catecholamine-induced arrhythmias. Do not administer catecholamines to overexposed individuals. Contact a Poison Control Center for further treatment information.

This material is an asphyxiant which may have anesthetic properties at high concentrations. If present in sufficient concentrations to reduce the oxygen level below 18% in inhaled air, rapid respiration, mental dullness, incoordination, poor judgement, nausea, and unconsciousness may result. Oxygen deficiency may occur without warning in areas where this gas may displace air.

NOTE TO EMERGENCY RESPONDERS: The odor of mercaptans such as methyl mercaptan or ethyl mercaptan is offensive and similar to rotten eggs. The presence of odors is not a reliable warning signal. DO NOT use odor to estimate the amount of mercaptan vapors present.

5. FIRE-FIGHTING MEASURES

Ignition Temperature - AIT (degrees F):

Not determined.

Flash Point (degrees F):

-156

Flammable Limits (%):

Lower: 2.3

Upper: 9.5

Recommended Fire Extinguishing Agents And Special Procedures:

Fight fire from protected location or maximum possible distance. Stop flow of gas before attempting to extinguish flames. Use water spray to cool fire-exposed containers and to protect persons attempting to stop the flow of gas. Use flooding quantities of water as fog or spary. Use dry chemical or carbon dioxide to extinguish flames.

Unusual or Explosive Hazards:

Explosive air-vapor mixtures may form.

Danger! Readily forms explosive air-vapor mixtures; may release explosive vapors that travel, be ignited at remote locations, and flash back. Containers may explode in fire. Do not expose to heat, sparks, flame, static, or other sources of ignition. When handling, use non-sparking tool, ground and bond all containers.

Extinguishing Media Which Must Not Be Used:

Not determined.

Special Protective Equipment for Firefighters:

Wear full protective clothing and positive pressure breathing apparatus.

6. ACCIDENTAL RELEASE MEASURES (Transportation Spills: CHEMTREC (800)424-9300)

Procedures in Case of Accidental Release, Breakage or Leakage:

Eliminate all ignition sources including internal combustion engines and power tools. Ventilate area. Keep people away. Stay upwind and warn of possible downwind explosion hazard. Avoid breathing vapor. Avoid contact with eyes, skin, or clothing. Pressure demand air supplied respirators should always be worn when the airborne concentration of the contaminant or oxygen is unknown. Otherwise, wear respiratory protection and other personal protective equipment as appropriate for the potential exposure hazard.

If more than 2,000,000 pounds of product is spilled, then report spill according to SARA 304 and/or CERCLA 102(a) requirements, unless product qualifies for the petroleum exemption (CERCLA Section 101(14)).

7. HANDLING AND STORAGE

Precautions to be Taken in
Handling:
Use spark-proof tools. Material may be at elevated temperatures and/or pressures. Exercise care when opening bleeders and sampling ports.
Storage:
Ground and bond shipping container, transfer line, and receiving container. Keep away from heat, sparks, flame, and other sources of ignition.

8. EXPOSURE CONTROLS/PERSONAL PROTECTION

Protective Equipment (Type)
Eye/Face Protection:
Safety glasses, chemical type goggles, or face shield recommended to prevent eye contact.
Skin Protection:
Protective clothing such as coveralls or lab coats should be worn. Launder or dry-clean when soiled. Gloves and boots resistant to chemicals and petroleum distillates required. Insulated gloves also required if contact with liquid-cooled product or equipment is expected.
Respiratory Protection:
Airborne concentrations should be kept to lowest levels possible. If vapor, mist or dust is generated and the occupational exposure limit of the product, or any component of the product, is exceeded, use appropriate NIOSH or MSHA approved air purifying or air supplied respirator after determining the airborne concentration of the contaminant. Air supplied respirators should always be worn when airborne concentration of the contaminant or oxygen content is unknown.
Ventilation:
Use explosion-proof equipment to maintain adequate ventilation to meet occupational exposure limits, if applicable (see below), prevent accumulation of explosive air-gas mixtures, and avoid significant oxygen displacement. Oxygen levels should be at least 19.5% in confined spaces or other work areas (OSHA value).
Exposure Limit for Total Product:
Propane: OSHA PEL-TWA 1000 ppm.

9. PHYSICAL AND CHEMICAL PROPERTIES

Appearance:
Gas
Odor:
If odorized will have rotten egg odor - otherwise, odorless
Boiling Point (degrees F):
-40
Melting/Freezing point (degrees F):
Not applicable.
Specific Gravity (water=1):
.5077
pH of undiluted product:
Not applicable.
Vapor Pressure:
10 mmHg at 80.6
Viscosity:
Not applicable.
VOC Content:
Not determined.
Vapor Density (air=1):
1.5
Solubility in Water (%):
> 10
Other: None

10. STABILITY AND REACTIVITY

This Material Reacts Violently With:
(If Others is checked below, see comments for details)

Air	Water	Heat	Strong Oxidizers	Others	None of These
		X	X		
_	_	_	_	_	_

Comments:
None
Products Evolved When Subjected to Heat or Combustion:
Toxic levels of carbon monoxide, carbon dioxide, irritating aldehydes and ketones.
Hazardous Polymerizations: DO NOT OCCUR

11. TOXICOLOGICAL INFORMATION

TOXICOLOGICAL INFORMATION(ANIMAL TOXICITY DATA)
Median Lethal Dose
Oral:
Not applicable; material is a gas.
Inhalation:
Not determined.
Dermal:
Not applicable; material is a gas.
Irritation Index, Estimation of Irritation (Species)
Skin:
(Draize) Believed to be < .50 /8.0 (rabbit) no appreciable effect
Eyes:
(Draize) Believed to be < 15.00 /110 (rabbit) no appreciable effect
Sensitization:
Not determined.
Other:
None

12. DISPOSAL CONSIDERATIONS

Waste Disposal Methods
This product (as presently constituted) has the RCRA characteristics of ignitability, and, if discarded in its present form, would have the hazardous waste number of D001. Under RCRA, it is the responsibility of the user of the product to determine, at the time of disposal, whether the product meets RCRA criteria for hazardous waste. This is because product uses, transformations, mixtures, processes, etc. may change the classification to non-hazardous, or hazardous for reasons other than, or in addition to ignitability.
Remarks
This product is potentially biodegradable.

13. TRANSPORT INFORMATION

Transportation
DOT:
Proper Shipping Name:
Liquified Petroleum Gas
Hazard Class:
2.1
Identification Number: UN 1075
Packing Group:
Label Required:
Flammable gas
IMDG:
Proper Shipping Name:
Not evaluated
ICAO:

Proper Shipping Name:
Not evaluated
TDG:
Proper Shipping Name:
Not evaluated

14. REGULATORY INFORMATION

Federal Regulations:
SARA Title III:
Section 302/304 Extremely Hazardous Substances

Seq.	Chemical Name	CAS Number	Range in %
01	Methyl mercaptan (if odorized - 50 ppm max)	74-93-1	0.005

Section 302/304 Extremely Hazardous Substances (CONT)

Seq.	TPQ	RQ
01	500	100

Section 311 Hazardous Categorization:

Acute	Chronic	Fire	Pressure	Reactive	N/A
X		X			

Section 313 Toxic Chemical

Chemical Name	CAS Number	Concentration

None
CERCLA 102(a)/DOT Hazardous Substances: (+ indicates DOT Hazardous Substance)

Seq.	Chemical Name	CAS Number	Range in %
01+	Methyl mercaptan (if odorized - 50 ppm max)	74-93-1	0.005

CERCLA/DOT Hazardous Substances (Sequence Numbers and RQ's):

Seq.	RQ
01+	100

TSCA Inventory Status:
This product, or its components, are listed on or are exempt from the Toxic Substance Control Act (TSCA) Chemical Substance Inventory.
Other:
None.
State Regulations:
California Proposition 65:
The following detectable components of this product are substances, or belong to classes of substances, known to the State of California to cause cancer and/or reproductive toxicity.

Chemical Name	CAS Number

None
International Regulations:
Export Notification (TSCA-12b):
This product may be subject to export notification under TSCA section 12(b); contains:
Methyl mercaptan (if odorized - 50 ppm max)
WHMIS Classification:
Not determined
Canada Inventory Status:
This product, or its components, are listed on or are exempt from the Canadian Domestic Substance List (DSL).
EINECS Inventory Status:
This product, or its components, are listed on or are exempt from the European Inventory of Existing Chemical Substances (EINECS) or the European List of Notified Chemical Substances (ELINCS).
Australia Inventory Status:
Not determined.

Japan Inventory Status:
Not determined.

15. ENVIRONMENTAL INFORMATION

Aquatic Toxicity:
Not determined.
Mobility:
Not determined.
Persistence and Biodegradability:
Not determined.
Potential to Bioaccumulate:
Not determined.
Remarks:
None

16. OTHER INFORMATION

This product is currently on the FDA's GRAS (generally regarded as safe) list.
NFPA NO. 58 REQUIRES ODORIZATION OF PROPANE SOLD FOR GENERAL CONSUMER USE. ODORIZATION PROVIDES A METHOD OF DETECTION IN THE EVENT OF A LEAK. COMMON ODORANTS INCLUDE ETHYL MERCAPTAN AND THIOPANE.
A BRIEF SUMMARY OF THE SAFETY INFORMATION REGARDING THE ODORANT IS PROVIDED HERE. FOR MORE DETAILED INFORMATION, PLEASE REFER TO THE REFERENCE SECTION. DO NOT RELY ON ODOR TO WARN OF PRESENCE OF GAS. IT IS IMPORTANT TO NOTE THAT NO ODORANT IS EFFECTIVE 100% OF THE TIME UNDER ALL CONDITIONS. THE EFFECTIVENESS OF THE ODORANT CAN BE REDUCED BY EXPOSURE TO SMALL AMOUNTS OF OXYGEN, MOISTURE, RUST OR SCALE. IN ADDITION, THE ODORANT MAY BE ABSORBED BY SOIL, NEW TANK SURFACES, NEW PIPING, OR CERTAIN BUILDING MATERIALS SUCH AS MASONRY. WHENEVER AN EMPTY TANK IS FILLED, IT MUST BE COMPLETELY PURGED IN ACCORDANCE WITH NPGA BULLETIN 133-89 TO REMOVE AIR AND WATER. THE INTEGRITY OF UNDERGROUND PIPES SHOULD BE CHECKED PERIODICALLY. IF PROPANE LEAKS FROM AN UNDERGROUND PIPE, THE SOIL MAY ABSORB THE ODORANT AS THE GAS MIGRATES TO THE SURFACE, WHICH COULD LEAVE THE GAS UNDETECTED BY SMELL. IF A PROPANE SYSTEM HAS NOT BEEN USED FOR AN EXTENDED PERIOD, IT SHOULD BE THOROUGHLY CHECKED BEFORE CONTINUING USE.
CERTAIN PHYSICAL CIRCUMSTANCES SUCH AS COLDS, ALLERGIES, SMOKING, ALCOHOL, AGE OR STRONG COMPETING ODORS MAY AFFECT A PERSON'S ABILITY TO SMELL ANY ODOR. IN ADDITION, AS WITH ANY ODOR, CONTINUED EXPOSURE TO PROPANE ODORANT CAN REDUCE A PERSON'S ABILITY TO DETECT THE ODORANT.
REFERENCES
NPGA BULLETIN NO. 133-80 "PURGING NEW CONTAINERS"
NFPA BULLETIN NO. 58, "STORAGE AND HANDLING OF LIQUIFIED PETROLEUM GAS"
Dispose of as a vapor, venting at a safe location, keeping gas below explosive limit (LEL).
The information below is given to call attention to the issue of "naturally occurring radioactive materials". Although radon-222 levels in this product do not present any direct radon exposure, customers should be aware of the potential of radon daughter product buildup within their processing streams whatever the source of their product streams. Radon-222 is a naturally occurring radioactive gas which can be a contaminant in natural gas. During subsequent processing, radon tends to be concentrated in the liquified petroleum gas stream and in product streams having a similar boiling point range. Industry experience has shown that this product may contain small amounts of radon-222 and its radioactive decay products, called radon "daughters". The actual concentration of Radon-222 and radioactive daughters in the process equipment (IE lines, filters, pumps and reactor units) may accumulate significant levels of radioactive daughters and show a gamma radiation reading during operation. A potential external radiation hazard exists at or near any pipe, valve or vessel containing a radon-enriched stream or containing internal deposits of radioactive material, due to the transmission of gamma radiation through

its wall.
Field studies in the literature and conducted by company personnel at selected sites, have not shown any conditions that subject workers to cumulative exposures in excess of general population limits. Equipment emitting gamma radiation should be presumed to be internally contaminated with alpha-emitting decay products which may be a hazard if inhaled or ingested. During maintenance operations that require the opening of contaminated process equipment, the flow of gas should be stopped and a four hour delay enforced to allow the gamma radiation to drop to back-ground levels. Protective equipment E.G. coveralls, gloves and respirator (NIOSH/MSHA approved for high efficiency particulates and radionuclides, or supplied air) should be worn by personnel entering a vessel or working on contaminated process equipment to prevent skin contamination, ingestion or inhalation of any residue containing alpha radiation. Air-borne contamination may be minimized by handling scale and/or contaminated materials in a wet state.

THE INFORMATION CONTAINED IN THIS DATA SHEET IS BASED ON THE DATA AVAILABLE TO US AT THIS TIME, AND IS BELIEVED TO BE ACCURATE BASED UPON THAT DATA. IT IS PROVIDED INDEPENDENTLY OF ANY SALE OF THE PRODUCT, FOR PURPOSE OF HAZARD COMMUNICATION. IT IS NOT INTENDED TO CONSTITUTE PRODUCT PERFORMANCE INFORMATION, AND NO EXPRESS OR IMPLIED WARRANTY OF ANY KIND IS MADE WITH RESPECT TO THE PRODUCT, UNDERLYING DATA OR THE INFORMATION CONTAINED HEREIN. YOU ARE URGED TO OBTAIN DATA SHEETS FOR ALL PRODUCTS YOU BUY, PROCESS USE OR DISTRIBUTE, AND ARE ENCOURAGED TO ADVISE THOSE WHO MAY COME IN CONTACT WITH SUCH PRODUCTS OF THE INFORMATION CONTAINED HEREIN.

TO DETERMINE THE APPLICABILITY OR EFFECT OF ANY LAW OR REGULATION WITH RESPECT TO THE PRODUCT, YOU SHOULD CONSULT WITH YOUR LEGAL ADVISOR OR THE APPROPRIATE GOVERNMENT AGENCY. WE WILL NOT PROVIDE ADVICE ON SUCH MATTERS, OR BE RESPONSIBLE FOR ANY INJURY FROM THE USE OF THE PRODUCT DESCRIBED HEREIN. THE UNDERLYING DATA, AND THE INFORMATION PROVIDED HEREIN AS A RESULT OF THAT DATA, IS THE PROPERTY OF EQUIVA SERVICES, LLC AND IS NOT TO BE THE SUBJECT OF SALE OR EXCHANGE WITHOUT THE EXPRESS WRITTEN CONSENT OF EQUIVA SERVICES, LLC.

Date: 1999-01-04 New X Revised, Supersedes: 1997-03-24

Inquiries regarding MSDS should be directed to:

Equiva Services LLC
Manager Product Stewardship
P.O. Box 674414
Houston, TX 77267-4414

17. PRODUCT LABEL

Label Date: 1999-01-04

READ AND UNDERSTAND MATERIAL SAFETY DATA SHEET BEFORE HANDLING OR DISPOSING OF PRODUCT. THIS LABEL COMPLIES WITH THE REQUIREMENTS OF THE OSHA HAZARD COMMUNICATION STANDARD (29 CFR 1910.1200) FOR USE IN THE WORKPLACE. THIS LABEL IS NOT INTENDED TO BE USED WITH PACKAGING INTENDED FOR SALE TO CONSUMERS AND MAY NOT CONFORM WITH THE REQUIREMENTS OF THE CONSUMER PRODUCT SAFETY ACT OR OTHER RELATED REGULATORY REQUIREMENTS.

00436 TEXACO PROPANE

WARNING STATEMENT

DANGER ! FLAMMABLE GAS - MAY CAUSE FLASH FIRE
DELAYED EVAPORATION FROM CONTAMINATED CLOTHING MAY BE A FIRE HAZARD
LIQUID MAY CAUSE FROSTBITE
MAY CAUSE DIZZINESS AND DROWSINESS
GAS REDUCES OXYGEN AVAILABLE FOR BREATHING
GAS MAY ACCUMULATE IN CONFINED SPACES AND CAUSE SUFFOCATION

PRECAUTIONARY MEASURES

-Keep away from heat, sparks or flame.
-Use only with adequate ventilation.

- This gas deadens sense of smell. Do not depend on odor to detect presence of gas.
- Do not enter storage areas or confined spaces unless adequately ventilated.
- Use supplied air respiratory protection for cleaning large spills or upon entry into tanks, vessels, or other confined spaces.
- Avoid breathing vapor, mist, or gas.
- Rescue procedures should be attempted ONLY after notifying others of emergency and ONLY if appropriate personal equipment is available.
- Wear insulated gloves if contact with liquid cooled equipment is expected.
- Keep container closed.
- Workers should wash exposed skin several times daily with soap and water.

FIRST AID

Eye Contact:
Flush eyes with plenty of water for several minutes. Get medical attention if eye irritation persists.

Skin Contact:
Wash skin with plenty of soap and water for several minutes. Get medical attention if skin irritation develops or persists.
In case of cold burn, immediately place affected area in warm water (105 F) and keep at this temperature until circulation returns. Get medical attention.
If clothing becomes wetted, drench individual with water and remove contaminated clothing if possible. Slowly warm affected area of skin.

Ingestion:
No emergency care anticipated. This material is a gas at standard temperature and pressure.

Inhalation:
If inhaled, remove to fresh air. If not breathing, clear person's airway and give artificial respiration. If breathing is difficult, qualified medical personnel may administer oxygen. Get medical attention immediately.

Note to Physician:
Overexposure to this material may sensitize the heart to catecholamine-induced arrhythmias. Do not administer catecholamines to overexposed individuals. Contact a Poison Control Center for further treatment information.
This material is an asphyxiant which may have anesthetic properties at high concentrations. If present in sufficient concentrations to reduce the oxygen level below 18% in inhaled air, rapid respiration, mental dullness, incoordination, poor judgement, nausea, and unconsciousness may result. Oxygen deficiency may occur without warning in areas where this gas may displace air.

FIRE

In case of fire, use dry chemical or carbon dioxide to extinguish flames.
Use water spray to keep containers cool and protect personnel attempting to stop the flow of gas.
If more than 2,000,000 pounds of product is spilled, then report spill according to SARA 304 and/or CERCLA 102(a) requirements, unless product qualifies for the petroleum exemption (CERCLA Section 101(14)).

Chemical Name	CAS Number	Range in %
This product may be odorized. The odorant content may vary from 0-50 ppm; common odorants include mercaptans and thiopane.		
* Propane	74-98-6	100.00

PRODUCT IS HAZARDOUS ACCORDING TO OSHA (1910.1200).
* COMPONENT IS HAZARDOUS ACCORDING TO OSHA.

Pennsylvania Special Hazardous Substance(s)	CAS Number	Range in %

None

HMIS			NFPA		
Health:	1	Reactivity: 0	Health:	1	Reactivity: 0
Flammability:	4	Special : -	Flammability:	4	Special : -

Transportation

DOT:

Proper Shipping Name:
Liquified Petroleum Gas

Hazard Class:
2.1

Identification Number: UN 1075

Packing Group:

Label Required:
Flammable gas

CAUTION: Misuse of empty containers can be hazardous. Empty containers can be hazardous if used to store toxic, flammable, or reactive materials. Cutting or welding of empty containers might cause fire, explosion or toxic fumes from residues. Do not pressurize or expose to open flame or heat. Keep container closed and drum

MSDS Number: **A2844** * * * * * *Effective Date:* ***11/02/01*** * * * * * *Supercedes:* ***11/15/99***

ALUMINUM OXIDE

1. Product Identification

Synonyms: AluminAR® CC-10; Aluminum oxide; Alumina; activated Alumina; alpha-Alumina
CAS No.: 1344-28-1
Molecular Weight: 101.96
Chemical Formula: Al2O3 (contains about 11-12% bound water)
Product Codes:
J.T. Baker: 0536, 0537, 0538, 0539, 0540
Mallinckrodt: 0065

2. Composition/Information on Ingredients

Ingredient	CAS No	Percent	Hazardous
Aluminum Oxide	1344-28-1	90 - 100%	Yes

3. Hazards Identification

Emergency Overview

CAUTION! MAY CAUSE IRRITATION TO SKIN, EYES, AND RESPIRATORY TRACT.

J.T. Baker SAF-T-DATA(tm) Ratings (Provided here for your convenience)

Health Rating: 1 - Slight
Flammability Rating: 0 - None
Reactivity Rating: 0 - None
Contact Rating: 1 - Slight
Lab Protective Equip: GOGGLES; LAB COAT
Storage Color Code: Orange (General Storage)

Potential Health Effects

Inhalation:
Hazard is principally that of a nuisance dust. Coughing or shortness of breath may occur in cases of excessive inhalation.
Ingestion:

No adverse effects expected.
Skin Contact:
May cause irritation with redness and pain.
Eye Contact:
No adverse effects expected but dust may cause mechanical irritation.
Chronic Exposure:
No adverse effects expected.
Aggravation of Pre-existing Conditions:
Not expected to be a health hazard.

---

4. First Aid Measures

Inhalation:
Remove to fresh air. Get medical attention for any breathing difficulty.
Ingestion:
Give several glasses of water to drink to dilute. If large amounts were swallowed, get medical advice.
Skin Contact:
Immediately flush skin with plenty of water for at least 15 minutes. Remove contaminated clothing and shoes. Wash clothing before reuse. Thoroughly clean shoes before reuse. Get medical attention if irritation develops.
Eye Contact:
Immediately flush eyes with plenty of water for at least 15 minutes, lifting upper and lower eyelids occasionally. Get medical attention if irritation persists.

---

5. Fire Fighting Measures

Fire:
Not considered to be a fire hazard.
Explosion:
Not considered to be an explosion hazard.
Fire Extinguishing Media:
Use any means suitable for extinguishing surrounding fire.
Special Information:
Use protective clothing and breathing equipment appropriate for the surrounding fire and to protect against the aluminum oxide dust that may be dispersed in the air.

---

6. Accidental Release Measures

Ventilate area of leak or spill. Wear appropriate personal protective equipment as specified in Section 8. Spills: Sweep up and containerize for reclamation or disposal. Vacuuming or wet sweeping may be used to avoid dust dispersal.

---

7. Handling and Storage

Keep in a tightly closed container, stored in a cool, dry, ventilated area. Protect against physical damage. Containers of this material may be hazardous when empty since they retain product residues (dust, solids); observe all warnings and precautions listed for the product.

---

8. Exposure Controls/Personal Protection

Airborne Exposure Limits:
Alumina (Aluminum Oxide):
-OSHA Permissible Exposure Limit (PEL):
alpha alumina, 15 mg/m3 total dust, 5 mg/m3 respirable fraction
-ACGIH Threshold Limit Value (TLV):
aluminum oxide, 10 mg/m3 (TWA) inhalable (total) particulate matter containing no asbestos and < 1% crystalline silica, A4

Ventilation System:
A system of local and/or general exhaust is recommended to keep employee exposures below the Airborne Exposure Limits. Local exhaust ventilation is generally preferred because it can control the emissions of the contaminant at its source, preventing dispersion of it into the general work area. Please refer to the ACGIH document, *Industrial Ventilation, A Manual of Recommended Practices*, most recent edition, for details.

Personal Respirators (NIOSH Approved):
If the exposure limit is exceeded and engineering controls are not feasible, a half facepiece particulate respirator (NIOSH type N95 or better filters) may be worn for up to ten times the exposure limit or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest.. A full-face piece particulate respirator (NIOSH type N100 filters) may be worn up to 50 times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency, or respirator supplier, whichever is lowest. If oil particles (e.g. lubricants, cutting fluids, glycerine, etc.) are present, use a NIOSH type R or P filter. For emergencies or instances where the exposure levels are not known, use a full-facepiece positive-pressure, air-supplied respirator. WARNING: Air-purifying respirators do not protect workers in oxygen-deficient atmospheres.

Skin Protection:
Wear protective gloves and clean body-covering clothing.

Eye Protection:
Use chemical safety goggles. Maintain eye wash fountain and quick-drench facilities in work area.

---

9. Physical and Chemical Properties

Appearance:
White powder.

Odor:
Odorless.

Solubility:
Insoluble in water.

Density:
4.0 @ 20C/4C

pH:
No information found.

% Volatiles by volume @ 21C (70F):
0

Boiling Point:
2980C (5396F)

Melting Point:
ca. 2000C (ca. 3632F)

Vapor Density (Air=1):
No information found.

Vapor Pressure (mm Hg):
No information found.

Evaporation Rate (BuAc=1):
No information found.

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10. Stability and Reactivity

Stability:
Stable under ordinary conditions of use and storage.
Hazardous Decomposition Products:
No information found.
Hazardous Polymerization:
Will not occur.
Incompatibilities:
Chlorine trifluoride, Ethylene oxide.
Conditions to Avoid:
Incompatibles.

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11. Toxicological Information

No LD50/LC50 information found relating to normal routes of occupational exposure.
Investigated as a tumorigen.

--------\Cancer Lists\----------

Ingredient	---NTP Carcinogen--- Known	Anticipated	IARC Category
Aluminum Oxide (1344-28-1)	No	No	None

---

12. Ecological Information

Environmental Fate:
No information found.
Environmental Toxicity:
No information found.

---

13. Disposal Considerations

Whatever cannot be saved for recovery or recycling should be managed in an appropriate and approved waste disposal facility. Processing, use or contamination of this product may change the waste management options. State and local disposal regulations may differ from federal disposal regulations. Dispose of container and unused contents in accordance with federal, state and local requirements.

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14. Transport Information

Not regulated.

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15. Regulatory Information

--------\Chemical Inventory Status - Part 1\----------

Ingredient	TSCA	EC	Japan	Australia
Aluminum Oxide (1344-28-1)	Yes	Yes	Yes	Yes

\-------\Chemical Inventory Status - Part 2\----------

Ingredient	Korea	--Canada-- DSL	NDSL	Phil.
Aluminum Oxide (1344-28-1)	Yes	Yes	No	Yes

\-------\Federal, State & International Regulations - Part 1\----------

Ingredient	-SARA 302- RQ	TPQ	------SARA 313---- List	Chemical Catg.
Aluminum Oxide (1344-28-1)	No	No	Yes	No

\-------\Federal, State & International Regulations - Part 2\----------

Ingredient	CERCLA	-RCRA- 261.33	-TSCA- 8(d)
Aluminum Oxide (1344-28-1)	No	No	No

Chemical Weapons Convention: No TSCA 12(b): No CDTA: No
SARA 311/312: Acute: Yes Chronic: No Fire: No Pressure: No
Reactivity: No (Pure / Solid)

Australian Hazchem Code: None allocated.
Poison Schedule: None allocated.
WHMIS:
This MSDS has been prepared according to the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all of the information required by the CPR.

---

16. Other Information

NFPA Ratings: Health: **1** Flammability: **0** Reactivity: **0**
Label Hazard Warning:
CAUTION! MAY CAUSE IRRITATION TO SKIN, EYES, AND RESPIRATORY TRACT.
Label Precautions:
Use with adequate ventilation.
Keep container closed.
Avoid breathing dust.
Wash thoroughly after handling.
Avoid contact with eyes, skin and clothing.
Label First Aid:
If inhaled, remove to fresh air. Get medical attention for any breathing difficulty. In case of

contact, immediately flush eyes or skin with plenty of water for at least 15 minutes. Get medical attention if irritation develops or persists.

Product Use:

Laboratory Reagent.

Revision Information:

MSDS Section(s) changed since last revision of document include: 8.

Disclaimer:

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Mallinckrodt Baker, Inc. provides the information contained herein in good faith but makes no representation as to its comprehensiveness or accuracy. This document is intended only as a guide to the appropriate precautionary handling of the material by a properly trained person using this product. Individuals receiving the information must exercise their independent judgment in determining its appropriateness for a particular purpose. MALLINCKRODT BAKER, INC. MAKES NO REPRESENTATIONS OR WARRANTIES, EITHER EXPRESS OR IMPLIED, INCLUDING WITHOUT LIMITATION ANY WARRANTIES OF MERCHANTABILITY, FITNESS FOR A PARTICULAR PURPOSE WITH RESPECT TO THE INFORMATION SET FORTH HEREIN OR THE PRODUCT TO WHICH THE INFORMATION REFERS. ACCORDINGLY, MALLINCKRODT BAKER, INC. WILL NOT BE RESPONSIBLE FOR DAMAGES RESULTING FROM USE OF OR RELIANCE UPON THIS INFORMATION.

****

Prepared by: Environmental Health & Safety
Phone Number: (314) 654-1600 (U.S.A.)

MSDS Number: **C0497** * * * * * *Effective Date:* ***11/02/01*** * * * * * *Supercedes:* ***11/17/99***

CALCIUM SULFATE, ANHYDROUS, POWDER

1. Product Identification

Synonyms: Anhydrous Gypsum; Anhydrite; Anhydrous Calcium Sulfate
CAS No.: 7778-18-9
Molecular Weight: 136.14
Chemical Formula: CaSO4
Product Codes: 1458

2. Composition/Information on Ingredients

Ingredient	CAS No	Percent	Hazardous
Calcium Sulfate (anhydrous)	7778-18-9	98 - 100%	Yes

3. Hazards Identification

Emergency Overview

WARNING! CAUSES IRRITATION TO SKIN, EYES AND RESPIRATORY TRACT. MAY BE HARMFUL IF SWALLOWED.

J.T. Baker SAF-T-DATA(tm) Ratings (Provided here for your convenience)

Health Rating: 1 - Slight
Flammability Rating: 0 - None
Reactivity Rating: 0 - None
Contact Rating: 1 - Slight
Lab Protective Equip: GOGGLES; LAB COAT
Storage Color Code: Orange (General Storage)

Potential Health Effects

Inhalation:
Causes irritation to the respiratory tract. Symptoms may include coughing, shortness of breath.
Ingestion:
May cause obstruction in stomach, as it hardens with moisture. Symptoms include stomach pain, distress.

Skin Contact:
Causes irritation, redness, pain.
Eye Contact:
Causes irritation, redness, and pain.
Chronic Exposure:
No information found.
Aggravation of Pre-existing Conditions:
No information found.

---

4. First Aid Measures

Inhalation:
Remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Get medical attention.
Ingestion:
Induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. Get medical attention.
Skin Contact:
Immediately flush skin with plenty of water for at least 15 minutes. Remove contaminated clothing and shoes. Get medical attention. Wash clothing before reuse. Thoroughly clean shoes before reuse.
Eye Contact:
Immediately flush eyes with plenty of water for at least 15 minutes, lifting lower and upper eyelids occasionally. Get medical attention immediately.

Note to Physician:
Drinking glycerin, gelatin solutions, or large volumes of water may delay the hardening of calcium sulfate in the stomach. Surgical relief of obstruction, particularly at the pylorus, may be necessary.

---

5. Fire Fighting Measures

Fire:
Not considered to be a fire hazard.
Explosion:
Not considered to be an explosion hazard.
Fire Extinguishing Media:
Use any means suitable for extinguishing surrounding fire.
Special Information:
In the event of a fire, wear full protective clothing and NIOSH-approved self-contained breathing apparatus with full facepiece operated in the pressure demand or other positive pressure mode.

---

6. Accidental Release Measures

Ventilate area of leak or spill. Wear appropriate personal protective equipment as specified in Section 8. Spills: Sweep up and containerize for reclamation or disposal. Vacuuming or wet sweeping may be used to avoid dust dispersal.

---

7. Handling and Storage

Keep in a tightly closed container, stored in a cool, dry, ventilated area. Protect against physical damage. Containers of this material may be hazardous when empty since they retain product residues (dust, solids); observe all warnings and precautions listed for the product.

---

8. Exposure Controls/Personal Protection

Airborne Exposure Limits:
-OSHA Permissible Exposure Limit (PEL):
15 mg/m3 total dust, 5 mg/m3 respirable fraction
-ACGIH Threshold Limit Value (TLV):
10 mg/m3 total dust containing no asbestos and < 1% crystalline silica
Ventilation System:
A system of local and/or general exhaust is recommended to keep employee exposures below the Airborne Exposure Limits. Local exhaust ventilation is generally preferred because it can control the emissions of the contaminant at its source, preventing dispersion of it into the general work area. Please refer to the ACGIH document, *Industrial Ventilation, A Manual of Recommended Practices*, most recent edition, for details.
Personal Respirators (NIOSH Approved):
If the exposure limit is exceeded and engineering controls are not feasible, a half facepiece particulate respirator (NIOSH type N95 or better filters) may be worn for up to ten times the exposure limit or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest.. A full-face piece particulate respirator (NIOSH type N100 filters) may be worn up to 50 times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency, or respirator supplier, whichever is lowest. If oil particles (e.g. lubricants, cutting fluids, glycerine, etc.) are present, use a NIOSH type R or P filter. For emergencies or instances where the exposure levels are not known, use a full-facepiece positive-pressure, air-supplied respirator. WARNING: Air-purifying respirators do not protect workers in oxygen-deficient atmospheres.
Skin Protection:
Wear protective gloves and clean body-covering clothing.
Eye Protection:
Use chemical safety goggles. Maintain eye wash fountain and quick-drench facilities in work area.

---

9. Physical and Chemical Properties

Appearance:
White granules or powder.
Odor:
Odorless.
Solubility:
Slight, 0.24g in 100g of water.
Specific Gravity:
2.96
pH:
No information found.
% Volatiles by volume @ 21C (70F):
0
Boiling Point:
1193C (2179F)
Melting Point:
1450C (2642F) Monoclinic
Vapor Density (Air=1):
No information found.
Vapor Pressure (mm Hg):

No information found.
Evaporation Rate (BuAc=1):
No information found.

---

10. Stability and Reactivity

Stability:
Very hygroscopic. Stable under ordinary conditions of use and storage.
Hazardous Decomposition Products:
Burning may produce sulfur oxides.
Hazardous Polymerization:
Will not occur.
Incompatibilities:
Diazomethane, aluminum, phosphorous.
Conditions to Avoid:
Air, moisture, and incompatibles.

---

11. Toxicological Information

No LD50/LC50 information found relating to normal routes of occupational exposure.

--------\Cancer Lists\----------

Ingredient	---NTP Carcinogen--- Known	Anticipated	IARC Category
Calcium Sulfate (anhydrous) (7778-18-9)	No	No	None

---

12. Ecological Information

Environmental Fate:
No information found.
Environmental Toxicity:
No information found.

---

13. Disposal Considerations

Whatever cannot be saved for recovery or recycling should be managed in an appropriate and approved waste disposal facility. Processing, use or contamination of this product may change the waste management options. State and local disposal regulations may differ from federal disposal regulations. Dispose of container and unused contents in accordance with federal, state and local requirements.

---

14. Transport Information

Not regulated.

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15. Regulatory Information

--------\Chemical Inventory Status - Part 1\--------------------------------

Ingredient	TSCA	EC	Japan	Australia
Calcium Sulfate (anhydrous) (7778-18-9)	Yes	Yes	Yes	Yes

--------\Chemical Inventory Status - Part 2\--------------------------------

Ingredient	Korea	--Canada-- DSL	NDSL	Phil.
Calcium Sulfate (anhydrous) (7778-18-9)	Yes	Yes	No	Yes

--------\Federal, State & International Regulations - Part 1\--------------

Ingredient	-SARA 302- RQ	TPQ	------SARA 313---- List	Chemical Catg.
Calcium Sulfate (anhydrous) (7778-18-9)	No	No	No	No

--------\Federal, State & International Regulations - Part 2\--------------

Ingredient	CERCLA	-RCRA- 261.33	-TSCA- 8(d)
Calcium Sulfate (anhydrous) (7778-18-9)	No	No	No

Chemical Weapons Convention: No TSCA 12(b): No CDTA: No
SARA 311/312: Acute: Yes Chronic: No Fire: No Pressure: No
Reactivity: No (Pure / Solid)

Australian Hazchem Code: None allocated.
Poison Schedule: None allocated.
WHMIS:
This MSDS has been prepared according to the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all of the information required by the CPR.

---

16. Other Information

NFPA Ratings: Health: **1** Flammability: **0** Reactivity: **0**
Label Hazard Warning:
WARNING! CAUSES IRRITATION TO SKIN, EYES AND RESPIRATORY TRACT. MAY BE HARMFUL IF SWALLOWED.
Label Precautions:
Avoid breathing dust.
Keep container closed.

Use only with adequate ventilation.
Wash thoroughly after handling.
Avoid contact with eyes, skin and clothing.

Label First Aid:

If swallowed, induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. If inhaled, remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. In case of contact, immediately flush eyes or skin with plenty of water for at least 15 minutes. Remove contaminated clothing and shoes. Wash clothing before reuse. In all cases, get medical attention.

Product Use:

Laboratory Reagent.

Revision Information:

MSDS Section(s) changed since last revision of document include: 8.

Disclaimer:

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Mallinckrodt Baker, Inc. provides the information contained herein in good faith but makes no representation as to its comprehensiveness or accuracy. This document is intended only as a guide to the appropriate precautionary handling of the material by a properly trained person using this product. Individuals receiving the information must exercise their independent judgment in determining its appropriateness for a particular purpose. MALLINCKRODT BAKER, INC. MAKES NO REPRESENTATIONS OR WARRANTIES, EITHER EXPRESS OR IMPLIED, INCLUDING WITHOUT LIMITATION ANY WARRANTIES OF MERCHANTABILITY, FITNESS FOR A PARTICULAR PURPOSE WITH RESPECT TO THE INFORMATION SET FORTH HEREIN OR THE PRODUCT TO WHICH THE INFORMATION REFERS. ACCORDINGLY, MALLINCKRODT BAKER, INC. WILL NOT BE RESPONSIBLE FOR DAMAGES RESULTING FROM USE OF OR RELIANCE UPON THIS INFORMATION.

****

Prepared by: Environmental Health & Safety
Phone Number: (314) 654-1600 (U.S.A.)

MSDS Number: **C5885** * * * * * *Effective Date:* ***01/31/00*** * * * * * *Supercedes:* ***12/08/96***

CUPRIC OXIDE

1. Product Identification

Synonyms: Black copper oxide; copper (II) oxide
CAS No.: 1317-38-0
Molecular Weight: 79.55
Chemical Formula: CuO
Product Codes:
J.T. Baker: 1814, 1820, 5255, 5256
Mallinckrodt: 3887, 4832

2. Composition/Information on Ingredients

Ingredient	CAS No	Percent	Hazardous
Cupric Oxide	1317-38-0	100%	Yes

3. Hazards Identification

Emergency Overview

WARNING! HARMFUL IF SWALLOWED. AFFECTS THE LIVER AND KIDNEYS. CAUSES IRRITATION TO SKIN, EYES AND RESPIRATORY TRACT.

J.T. Baker SAF-T-DATA(tm) Ratings (Provided here for your convenience)

Health Rating: 2 - Moderate
Flammability Rating: 1 - Slight
Reactivity Rating: 0 - None
Contact Rating: 1 - Slight
Lab Protective Equip: GOGGLES; LAB COAT
Storage Color Code: Orange (General Storage)

Potential Health Effects

Inhalation:
Causes irritation to respiratory tract, symptoms may include coughing, sore throat, and shortness of breath. May result in ulceration and perforation of respiratory tract. When heated, this

compound may give off copper fume, which can cause symptoms similar to the common cold, including chills and stuffiness of the head.

Ingestion:

Systemic copper poisoning may result from ingestion of this compound. Symptoms may include capillary damage, headache, cold sweat, weak pulse, kidney and liver damage, central nervous excitation followed by depression, jaundice, convulsions, blood effects, paralysis and coma. Death may occur from shock or renal failure.

Skin Contact:

Causes irritation, redness, pain.

Eye Contact:

Causes irritation with redness, pain. May cause eye damage.

Chronic Exposure:

Prolonged or repeated skin exposure may cause dermatitis. Prolonged or repeated exposure to dusts of copper salts may cause discoloration of the skin or hair, blood and liver damage, ulceration and perforation of the nasal septum, runny nose, metallic taste, and atrophic changes and irritation of the mucous membranes.

Aggravation of Pre-existing Conditions:

Persons with pre-existing skin disorders, impaired liver, kidney, or pulmonary function, glucose 6-phosphate-dehydrogenase deficiency, or pre-existing Wilson's disease may be more susceptible to the effects of this material.

---

4. First Aid Measures

Inhalation:

Remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Call a physician.

Ingestion:

Induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. Call a physician immediately.

Skin Contact:

In case of contact, wipe off excess material from skin then immediately flush skin with plenty of water for at least 15 minutes. Remove contaminated clothing and shoes. Wash clothing before reuse. Call a physician.

Eye Contact:

Immediately flush eyes with plenty of water for at least 15 minutes, lifting lower and upper eyelids occasionally. Get medical attention immediately.

---

5. Fire Fighting Measures

Fire:

Not considered to be a fire hazard. Large masses exposed to moist air at over 100C can result in spontaneous combustion.

Explosion:

Not considered to be an explosion hazard. Reactions with incompatibles may pose an explosion hazard.

Fire Extinguishing Media:

Use any means suitable for extinguishing surrounding fire.

Special Information:

In the event of a fire, wear full protective clothing and NIOSH-approved self-contained breathing apparatus with full facepiece operated in the pressure demand or other positive pressure mode.

---

6. Accidental Release Measures

Ventilate area of leak or spill. Keep unnecessary and unprotected people away from area of spill. Wear appropriate personal protective equipment as specified in Section 8. Spills: Pick up and place in a suitable container for reclamation or disposal, using a method that does not generate dust. US Regulations (CERCLA) require reporting spills and releases to soil, water and air in excess of reportable quantities. The toll free number for the US Coast Guard National Response Center is (800) 424-8802.

---

7. Handling and Storage

Keep in a tightly closed container, stored in a cool, dry, ventilated area. Protect against physical damage. Isolate from incompatible substances. Containers of this material may be hazardous when empty since they retain product residues (dust, solids); observe all warnings and precautions listed for the product.

---

8. Exposure Controls/Personal Protection

Airborne Exposure Limits:
-OSHA Permissible Exposure Limit (PEL):
1 mg/m3 (TWA) for copper dusts & mists as Cu
-ACGIH Threshold Limit Value (TLV):
1 mg/m3 (TWA) for copper dusts & mists as Cu
Ventilation System:
A system of local and/or general exhaust is recommended to keep employee exposures below the Airborne Exposure Limits. Local exhaust ventilation is generally preferred because it can control the emissions of the contaminant at its source, preventing dispersion of it into the general work area. Please refer to the ACGIH document, *Industrial Ventilation, A Manual of Recommended Practices*, most recent edition, for details.
Personal Respirators (NIOSH Approved):
If the exposure limit is exceeded, a half-face dust/mist respirator may be worn for up to ten times the exposure limit or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest. A full-face piece dust/mist respirator may be worn up to 50 times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency, or respirator supplier, whichever is lowest. For emergencies or instances where the exposure levels are not known, use a full-facepiece positive-pressure, air-supplied respirator. WARNING: Air-purifying respirators do not protect workers in oxygen-deficient atmospheres.
Skin Protection:
Wear protective gloves and clean body-covering clothing.
Eye Protection:
Use chemical safety goggles and/or full face shield where dusting or splashing of solutions is possible. Maintain eye wash fountain and quick-drench facilities in work area.

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9. Physical and Chemical Properties

Appearance:
Black to brownish-black powder, granules, or wire.
Odor:
Odorless.
Solubility:
Insoluble in water.
Density:
6.32 @ 14C/4C

pH:
No information found.
% Volatiles by volume @ 21C (70F):
0
Boiling Point:
Not applicable.
Melting Point:
1026C (1879F)
Vapor Density (Air=1):
No information found.
Vapor Pressure (mm Hg):
No information found.
Evaporation Rate (BuAc=1):
No information found.

---

10. Stability and Reactivity

Stability:
Stable under ordinary conditions of use and storage.
Hazardous Decomposition Products:
Toxic metal fumes may form when heated to decomposition.
Hazardous Polymerization:
Will not occur.
Incompatibilities:
Aluminum, boron, cesium acetylene carbide, dirubidium acetylide, hydrazine, hydrogen, hydrogen sulfide, lead oxide, magnesium, metals, phospham, potassium, phthalic anhydride, rubidium acetylene carbide, sodium, titanium, and zirconium. Forms acetylides with acetylene, sodium hypobromite and nitromethane.
Conditions to Avoid:
Incompatibles.

---

11. Toxicological Information

No LD50/LC50 information found relating to normal routes of occupational exposure.

--------\Cancer Lists\----------

Ingredient	---NTP Carcinogen--- Known	Anticipated	IARC Category
Cupric Oxide (1317-38-0)	No	No	None

---

12. Ecological Information

Environmental Fate:
When released into the soil, this material is not expected to biodegrade. When released into water, this material is not expected to biodegrade. When released into water, this material is not expected to evaporate significantly.
Environmental Toxicity:
No information found.

13. Disposal Considerations

Whatever cannot be saved for recovery or recycling should be managed in an appropriate and approved waste disposal facility. Processing, use or contamination of this product may change the waste management options. State and local disposal regulations may differ from federal disposal regulations. Dispose of container and unused contents in accordance with federal, state and local requirements.

14. Transport Information

Not regulated.

15. Regulatory Information

--------\Chemical Inventory Status - Part 1\--------

Ingredient	TSCA	EC	Japan	Australia
Cupric Oxide (1317-38-0)	Yes	Yes	Yes	Yes

--------\Chemical Inventory Status - Part 2\--------

Ingredient	Korea	--Canada-- DSL	NDSL	Phil.
Cupric Oxide (1317-38-0)	Yes	Yes	No	Yes

--------\Federal, State & International Regulations - Part 1\--------

Ingredient	-SARA 302- RQ	TPQ	------SARA 313---- List	Chemical Catg.
Cupric Oxide (1317-38-0)	No	No	No	Copper compo

--------\Federal, State & International Regulations - Part 2\--------

Ingredient	CERCLA	-RCRA- 261.33	-TSCA- 8(d)
Cupric Oxide (1317-38-0)	No	No	No

Chemical Weapons Convention: No TSCA 12(b): No CDTA: No
SARA 311/312: Acute: Yes Chronic: Yes Fire: No Pressure: No
Reactivity: No (Pure / Solid)

Australian Hazchem Code: None allocated.

Poison Schedule: None allocated.
WHMIS:
This MSDS has been prepared according to the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all of the information required by the CPR.

---

16. Other Information

NFPA Ratings: Health: **2** Flammability: **1** Reactivity: **0**
Label Hazard Warning:
WARNING! HARMFUL IF SWALLOWED. AFFECTS THE LIVER AND KIDNEYS. CAUSES IRRITATION TO SKIN, EYES AND RESPIRATORY TRACT.
Label Precautions:
Avoid breathing dust.
Keep container closed.
Use only with adequate ventilation.
Wash thoroughly after handling.
Avoid contact with eyes, skin and clothing.
Label First Aid:
If swallowed, induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. If inhaled, remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. In case of contact, immediately flush eyes or skin with plenty of water for at least 15 minutes. Remove contaminated clothing and shoes. Wash clothing before reuse. In all cases, get medical attention.
Product Use:
Laboratory Reagent.
Revision Information:
MSDS Section(s) changed since last revision of document include: 3, 9, 16.
Disclaimer:
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Mallinckrodt Baker, Inc. provides the information contained herein in good faith but makes no representation as to its comprehensiveness or accuracy. This document is intended only as a guide to the appropriate precautionary handling of the material by a properly trained person using this product. Individuals receiving the information must exercise their independent judgment in determining its appropriateness for a particular purpose. MALLINCKRODT BAKER, INC. MAKES NO REPRESENTATIONS OR WARRANTIES, EITHER EXPRESS OR IMPLIED, INCLUDING WITHOUT LIMITATION ANY WARRANTIES OF MERCHANTABILITY, FITNESS FOR A PARTICULAR PURPOSE WITH RESPECT TO THE INFORMATION SET FORTH HEREIN OR THE PRODUCT TO WHICH THE INFORMATION REFERS. ACCORDINGLY, MALLINCKRODT BAKER, INC. WILL NOT BE RESPONSIBLE FOR DAMAGES RESULTING FROM USE OF OR RELIANCE UPON THIS INFORMATION.
****
Prepared by: Environmental Health & Safety
Phone Number: (314) 654-1600 (U.S.A.)

MSDS Number: **F1306** * * * * * Effective Date: **05/17/01** * * * * * Supercedes: **06/30/98**

Ferric Oxide

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1. Product Identification

Synonyms: Iron (III) Oxide; Red Iron Oxide; C.I. 77491; Iron Sesquioxide
CAS No.: 1309-37-1
Molecular Weight: 159.69
Chemical Formula: Fe2O3
Product Codes: 2024

---

2. Composition/Information on Ingredients

Ingredient	CAS No	Percent	Hazardous
Iron Oxide, Fe2O3	1309-37-1	98 - 100%	Yes

---

3. Hazards Identification

Emergency Overview

WARNING! HARMFUL IF INHALED. AFFECTS RESPIRATORY SYSTEM. MAY CAUSE IRRITATION TO EYES AND RESPIRATORY TRACT.

J.T. Baker SAF-T-DATA(tm) Ratings (Provided here for your convenience)

Health Rating: 1 - Slight
Flammability Rating: 0 - None
Reactivity Rating: 1 - Slight
Contact Rating: 1 - Slight
Lab Protective Equip: GOGGLES; LAB COAT
Storage Color Code: Orange (General Storage)

Potential Health Effects

Inhalation:
May cause irritation to the respiratory tract. Symptoms may include coughing and shortness of breath.
Ingestion:

Extremely large oral dosages may produce gastrointestinal disturbances.
Skin Contact:
No adverse effects expected.
Eye Contact:
May cause mechanical irritation.
Chronic Exposure:
Long-term inhalation exposure to iron has resulted in mottling of the lungs, a condition referred to as siderosis. This is considered a benign pneumoconiosis and does not ordinarily cause significant physiological impairment. Long term eye exposures may stain the eyes and leave a "rust ring".
Aggravation of Pre-existing Conditions:
Persons with impaired respiratory function may be more susceptible to the effects of the substance.

---

4. First Aid Measures

Inhalation:
Remove to fresh air. Get medical attention for any breathing difficulty.
Ingestion:
If large amounts were swallowed, give water to drink and get medical advice.
Skin Contact:
Wash exposed area with soap and water. Get medical advice if irritation develops.
Eye Contact:
Immediately flush eyes with plenty of water for at least 15 minutes, lifting upper and lower eyelids occasionally. Get medical attention if irritation persists.

---

5. Fire Fighting Measures

Fire:
Not expected to be a fire hazard.
Explosion:
No information found.
Fire Extinguishing Media:
Use any means suitable for extinguishing surrounding fire.
Special Information:
In the event of a fire, wear full protective clothing and NIOSH-approved self-contained breathing apparatus with full facepiece operated in the pressure demand or other positive pressure mode.

---

6. Accidental Release Measures

Ventilate area of leak or spill. Wear appropriate personal protective equipment as specified in Section 8. Spills: Pick up and place in a suitable container for reclamation or disposal, using a method that does not generate dust.

---

7. Handling and Storage

Keep in a tightly closed container, stored in a cool, dry, ventilated area. Protect against physical damage. Containers of this material may be hazardous when empty since they retain product residues (dust, solids); observe all warnings and precautions listed for the product.

---

8. Exposure Controls/Personal Protection

Airborne Exposure Limits:
- OSHA Permissible Exposure Limit (PEL) -
Iron oxide fume: 10 mg/m3
- ACGIH Threshold Limit Value (TLV) -
Iron oxide dust and fume (Fe2O3) as Fe: 5 mg/m3 (TWA), inhalable particulate;
for particulate matter containing no asbestos and < 1% crystalline silica.
A4 - Not classifiable as a human carcinogen.

Ventilation System:
A system of local and/or general exhaust is recommended to keep employee exposures below the Airborne Exposure Limits. Local exhaust ventilation is generally preferred because it can control the emissions of the contaminant at its source, preventing dispersion of it into the general work area. Please refer to the ACGIH document, *Industrial Ventilation, A Manual of Recommended Practices*, most recent edition, for details.

Personal Respirators (NIOSH Approved):
If the exposure limit is exceeded, a half-face dust/mist respirator may be worn for up to ten times the exposure limit or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest. A full-face piece dust/mist respirator may be worn up to 50 times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency, or respirator supplier, whichever is lowest. For emergencies or instances where the exposure levels are not known, use a full-facepiece positive-pressure, air-supplied respirator. WARNING: Air-purifying respirators do not protect workers in oxygen-deficient atmospheres.

Skin Protection:
Wear protective gloves and clean body-covering clothing.

Eye Protection:
Use chemical safety goggles. Maintain eye wash fountain and quick-drench facilities in work area.

---

9. Physical and Chemical Properties

Appearance:
Reddish-brown powder.

Odor:
No information found.

Solubility:
Negligible (< 0.1%)

Specific Gravity:
5.24

pH:
No information found.

% Volatiles by volume @ 21C (70F):
0

Boiling Point:
No information found.

Melting Point:
1565C (2849F)

Vapor Density (Air=1):
Not applicable.

Vapor Pressure (mm Hg):
Not applicable.

Evaporation Rate (BuAc=1):
No information found.

---

10. Stability and Reactivity

Stability:
Stable under ordinary conditions of use and storage.
Hazardous Decomposition Products:
No information found.
Hazardous Polymerization:
Will not occur.
Incompatibilities:
Carbon monoxide, hydrazine, calcium hypochloride, performic acid, bromine pentafluoride.
Conditions to Avoid:
Incompatibles.

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11. Toxicological Information

No LD50/LC50 information found relating to normal routes of occupational exposure.
Investigated as a tumorigen.

```
  --------\Cancer Lists\-----------------------------------------------------
-
                                        ---NTP Carcinogen---
  Ingredient                           Known    Anticipated    IARC
Category
  ----------------------------------   -----    -----------    ------------
-
  Iron Oxide, Fe2O3 (1309-37-1)         No          No              3
```

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12. Ecological Information

Environmental Fate:
No information found.
Environmental Toxicity:
No information found.

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13. Disposal Considerations

Whatever cannot be saved for recovery or recycling should be managed in an appropriate and approved waste disposal facility. Processing, use or contamination of this product may change the waste management options. State and local disposal regulations may differ from federal disposal regulations. Dispose of container and unused contents in accordance with federal, state and local requirements.

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14. Transport Information

Not regulated.

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15. Regulatory Information

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  --------\Chemical Inventory Status - Part 1\--------------------------------
--
  Ingredient                                    TSCA  EC   Japan
Australia
```

Ingredient	EINECS	TSCA	Canada DSL	Canada NDSL	Canada Phil.
Iron Oxide, Fe2O3 (1309-37-1)	Yes	Yes	Yes	Yes	Yes

Ingredient	Korea	Canada DSL	Canada NDSL	Canada Phil.
Iron Oxide, Fe2O3 (1309-37-1)	Yes	Yes	No	Yes

Ingredient	SARA 302 RQ	SARA 302 TPQ	SARA 313 List	SARA 313 Chemical Catg.
Iron Oxide, Fe2O3 (1309-37-1)	No	No	No	No

Ingredient	CERCLA	RCRA 261.33	TSCA 8(d)
Iron Oxide, Fe2O3 (1309-37-1)	No	No	No

Chemical Weapons Convention: No TSCA 12(b): No CDTA: No
SARA 311/312: Acute: Yes Chronic: Yes Fire: No Pressure: No
Reactivity: No (Pure / Solid)

Australian Hazchem Code: None allocated.
Poison Schedule: None allocated.
WHMIS:
This MSDS has been prepared according to the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all of the information required by the CPR.

---

16. Other Information

NFPA Ratings: Health: **1** Flammability: **0** Reactivity: **0**
Label Hazard Warning:
WARNING! HARMFUL IF INHALED. AFFECTS RESPIRATORY SYSTEM. MAY CAUSE IRRITATION TO EYES AND RESPIRATORY TRACT.
Label Precautions:
Avoid contact with eyes, skin and clothing.
Wash thoroughly after handling.
Avoid breathing dust.
Keep container closed.
Use only with adequate ventilation.
Label First Aid:
If inhaled, remove to fresh air. Get medical attention for any breathing difficulty. In case of eye contact, immediately flush eyes with plenty of water for at least 15 minutes. Get medical attention

if irritation develops or persists.

Product Use:

Laboratory Reagent.

Revision Information:

No changes.

Disclaimer:

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Prepared by: Environmental Health & Safety
Phone Number: (314) 654-1600 (U.S.A.)

MSDS Number: **L2347** * * * * * *Effective Date:* ***11/02/01*** * * * * * *Supercedes:* ***11/17/99***

LEAD METAL

1. Product Identification

Synonyms: Granular lead, pigment metal; C.I. 77575
CAS No.: 7439-92-1
Molecular Weight: 207.19
Chemical Formula: Pb
Product Codes:
J.T. Baker: 2256, 2266
Mallinckrodt: 5668

2. Composition/Information on Ingredients

Ingredient	CAS No	Percent	Hazardous
Lead	7439-92-1	95 - 100%	Yes

3. Hazards Identification

Emergency Overview

POISON! DANGER! MAY BE FATAL IF SWALLOWED OR INHALED. CAUSES IRRITATION TO SKIN, EYES AND RESPIRATORY TRACT. NEUROTOXIN. AFFECTS THE GUM TISSUE, CENTRAL NERVOUS SYSTEM, KIDNEYS, BLOOD AND REPRODUCTIVE SYSTEM. POSSIBLE CANCER HAZARD. MAY CAUSE CANCER BASED ON ANIMAL DATA. Risk of cancer depends on duration and level of exposure.

J.T. Baker SAF-T-DATA(tm) Ratings (Provided here for your convenience)

Health Rating: 3 - Severe (Life)
Flammability Rating: 0 - None
Reactivity Rating: 0 - None
Contact Rating: 1 - Slight
Lab Protective Equip: GOGGLES; LAB COAT; VENT HOOD; PROPER GLOVES
Storage Color Code: Blue (Health)

Potential Health Effects

Inhalation:

Lead can be absorbed through the respiratory system. Local irritation of bronchia and lungs can occur and, in cases of acute exposure, symptoms such as metallic taste, chest and abdominal pain, and increased lead blood levels may follow. See also Ingestion.

Ingestion:

POISON! The symptoms of lead poisoning include abdominal pain and spasms, nausea, vomiting, headache. Acute poisoning can lead to muscle weakness, "lead line" on the gums, metallic taste, definite loss of appetite, insomnia, dizziness, high lead levels in blood and urine with shock, coma and death in extreme cases.

Skin Contact:

Lead and lead compounds may be absorbed through the skin on prolonged exposure; the symptoms of lead poisoning described for ingestion exposure may occur. Contact over short periods may cause local irritation, redness and pain.

Eye Contact:

Absorption can occur through eye tissues but the more common hazards are local irritation or abrasion.

Chronic Exposure:

Lead is a cumulative poison and exposure even to small amounts can raise the body's content to toxic levels. The symptoms of chronic exposure are like those of ingestion poisoning; restlessness, irritability, visual disturbances, hypertension and gray facial color may also be noted.

Aggravation of Pre-existing Conditions:

Persons with pre-existing kidney, nerve or circulatory disorders or with skin or eye problems may be more susceptible to the effects of this substance.

---

4. First Aid Measures

Inhalation:

Remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Get medical attention.

Ingestion:

Induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. Get medical attention.

Skin Contact:

Immediately flush skin with plenty of soap and water for at least 15 minutes. Remove contaminated clothing and shoes. Get medical attention. Wash clothing before reuse. Thoroughly clean shoes before reuse.

Eye Contact:

Immediately flush eyes with plenty of water for at least 15 minutes, lifting lower and upper eyelids occasionally. Get medical attention immediately.

---

5. Fire Fighting Measures

Fire:

Not considered to be a fire hazard. Powder/dust is flammable when heated or exposed to flame.

Explosion:

Not considered to be an explosion hazard.

Fire Extinguishing Media:

Use any means suitable for extinguishing surrounding fire. Do not allow water runoff to enter sewers or waterways.

Special Information:

In the event of a fire, wear full protective clothing and NIOSH-approved self-contained breathing

apparatus with full facepiece operated in the pressure demand or other positive pressure mode. Can produce toxic lead fumes at elevated temperatures and also react with oxidizing materials.

---

6. Accidental Release Measures

Ventilate area of leak or spill. Wear appropriate personal protective equipment as specified in Section 8. Spills: Sweep up and containerize for reclamation or disposal. Vacuuming or wet sweeping may be used to avoid dust dispersal. US Regulations (CERCLA) require reporting spills and releases to soil, water and air in excess of reportable quantities. The toll free number for the US Coast Guard National Response Center is (800) 424-8802.

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7. Handling and Storage

Keep in a tightly closed container, stored in a cool, dry, ventilated area. Protect against physical damage. Isolate from incompatible substances. Areas in which exposure to lead metal or lead compounds may occur should be identified by signs or appropriate means, and access to the area should be limited to authorized persons. Containers of this material may be hazardous when empty since they retain product residues (dust, solids); observe all warnings and precautions listed for the product.

---

8. Exposure Controls/Personal Protection

Airborne Exposure Limits:
For lead, metal and inorganic dusts and fumes, as Pb:
-OSHA Permissible Exposure Limit (PEL): 0.05 mg/m3 (TWA)
For lead, elemental and inorganic compounds, as Pb:
-ACGIH Threshold Limit Value (TLV): 0.05 mg/m3 (TWA), A3 animal carcinogen
ACGIH Biological Exposure Indices (BEI): 30 ug/100ml, notation B (see actual Indices for more information).
For lead, inorganic:
-NIOSH Recommended Exposure Limit (REL): 0.1 mg/m3 (TWA)
Ventilation System:
A system of local and/or general exhaust is recommended to keep employee exposures below the Airborne Exposure Limits. Local exhaust ventilation is generally preferred because it can control the emissions of the contaminant at its source, preventing dispersion of it into the general work area. Please refer to the ACGIH document, *Industrial Ventilation, A Manual of Recommended Practices*, most recent edition, for details.
Personal Respirators (NIOSH Approved):
If the exposure limit is exceeded and engineering controls are not feasible, a half-face high efficiency particulate respirator (NIOSH type N100 filter) may be worn for up to ten times the exposure limit or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest. A full-face piece high efficiency particulate respirator (NIOSH type N100 filter) may be worn up to 50 times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest. If oil particles (e.g. lubricants, cutting fluids, glycerine, etc.) are present, use a NIOSH type R or P filter. For emergencies or instances where the exposure levels are not known, use a full-facepiece positive-pressure, air-supplied respirator. WARNING: Air-purifying respirators do not protect workers in oxygen-deficient atmospheres.
Skin Protection:
Wear impervious protective clothing, including boots, gloves, lab coat, apron or coveralls, as appropriate, to prevent skin contact.
Eye Protection:

Use chemical safety goggles and/or full face shield where dusting or splashing of solutions is possible. Maintain eye wash fountain and quick-drench facilities in work area.
Other Control Measures:
Eating, drinking, and smoking should not be permitted in areas where solids or liquids containing lead compounds are handled, processed, or stored. See OSHA substance-specific standard for more information on personal protective equipment, engineering and work practice controls, medical surveillance, record keeping, and reporting requirements. (29 CFR 1910.1025).

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9. Physical and Chemical Properties

Appearance:
Small, white to blue-gray metallic shot or granules.
Odor:
Odorless.
Solubility:
Insoluble in water.
Density:
11.34
pH:
No information found.
% Volatiles by volume @ 21C (70F):
0
Boiling Point:
1740C (3164F)
Melting Point:
327.5C (622F)
Vapor Density (Air=1):
No information found.
Vapor Pressure (mm Hg):
1.77 @ 1000C (1832F)
Evaporation Rate (BuAc=1):
No information found.

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10. Stability and Reactivity

Stability:
Stable under ordinary conditions of use and storage.
Hazardous Decomposition Products:
Does not decompose but toxic lead or lead oxide fumes may form at elevated temperatures.
Hazardous Polymerization:
Will not occur.
Incompatibilities:
Ammonium nitrate, chlorine trifluoride, hydrogen peroxide, sodium azide, zirconium, disodium acetylide, sodium acetylide and oxidants.
Conditions to Avoid:
Heat, flames, ignition sources and incompatibles.

---

11. Toxicological Information

Toxicological Data:
Investigated as a tumorigen, mutagen, reproductive effector.
Reproductive Toxicity:
Lead and other smelter emissions are human reproductive hazards. (Chemical Council on Environmental Quality; Chemical Hazards to Human Reproduction, 1981).

Carcinogenicity:
EPA / IRIS classification: Group B2 - Probable human carcinogen, sufficient animal evidence.

--------\Cancer Lists\----------

Ingredient	NTP Carcinogen - Known	NTP Carcinogen - Anticipated	IARC Category
Lead (7439-92-1)	No	No	2B

12. Ecological Information

Environmental Fate:
When released into the soil, this material is not expected to leach into groundwater. This material may bioaccumulate to some extent.
Environmental Toxicity:
No information found.

13. Disposal Considerations

Whatever cannot be saved for recovery or recycling should be managed in an appropriate and approved waste facility. Although not a listed RCRA hazardous waste, this material may exhibit one or more characteristics of a hazardous waste and require appropriate analysis to determine specific disposal requirements. Processing, use or contamination of this product may change the waste management options. State and local disposal regulations may differ from federal disposal regulations. Dispose of container and unused contents in accordance with federal, state and local requirements.

14. Transport Information

Not regulated.

15. Regulatory Information

--------\Chemical Inventory Status - Part 1\----------

Ingredient	TSCA	EC	Japan	Australia
Lead (7439-92-1)	Yes	Yes	Yes	Yes

--------\Chemical Inventory Status - Part 2\----------

Ingredient	Korea	Canada - DSL	Canada - NDSL	Phil.
Lead (7439-92-1)	Yes	Yes	No	Yes

--------\Federal, State & International Regulations - Part 1\----------

Ingredient	-SARA 302- RQ	TPQ	------SARA 313---- List	Chemical Catg.
Lead (7439-92-1)	No	No	Yes	No

--------\Federal, State & International Regulations - Part 2\----------

Ingredient	CERCLA	-RCRA- 261.33	-TSCA- 8(d)
Lead (7439-92-1)	10	No	No

Chemical Weapons Convention: No TSCA 12(b): No CDTA: No
SARA 311/312: Acute: Yes Chronic: Yes Fire: No Pressure: No
Reactivity: No (Pure / Solid)

WARNING:
THIS PRODUCT CONTAINS CHEMICALS KNOWN TO THE STATE OF CALIFORNIA TO CAUSE CANCER AND BIRTH DEFECTS OR OTHER REPRODUCTIVE HARM.

Australian Hazchem Code: None allocated.
Poison Schedule: S6
WHMIS:
This MSDS has been prepared according to the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all of the information required by the CPR.

---

16. Other Information

NFPA Ratings: Health: **3** Flammability: **1** Reactivity: **0**
Label Hazard Warning:
POISON! DANGER! MAY BE FATAL IF SWALLOWED OR INHALED. CAUSES IRRITATION TO SKIN, EYES AND RESPIRATORY TRACT. NEUROTOXIN. AFFECTS THE GUM TISSUE, CENTRAL NERVOUS SYSTEM, KIDNEYS, BLOOD AND REPRODUCTIVE SYSTEM. POSSIBLE CANCER HAZARD. MAY CAUSE CANCER BASED ON ANIMAL DATA. Risk of cancer depends on duration and level of exposure.
Label Precautions:
Do not get in eyes, on skin, or on clothing.
Do not breathe dust.
Keep container closed.
Use only with adequate ventilation.
Wash thoroughly after handling.
Label First Aid:
If swallowed, induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. If inhaled, remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. In case of contact, immediately flush eyes or skin with plenty of water for at least 15 minutes. Remove contaminated clothing and shoes. Wash clothing before reuse. In all cases, get medical attention.
Product Use:
Laboratory Reagent.
Revision Information:
MSDS Section(s) changed since last revision of document include: 8.
Disclaimer:

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Prepared by: Environmental Health & Safety
Phone Number: (314) 654-1600 (U.S.A.)

MSDS Number: **M0716** * * * * * *Effective Date:* ***08/08/02*** * * * * * *Supercedes:* ***09/14/00***

MANGANESE DIOXIDE (TECHNICAL GRADE)

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1. Product Identification

Synonyms: Pyrolusite; Manganese black; Manganese peroxide; Manganese (IV) oxide
CAS No.: 1313-13-9
Molecular Weight: 86.94
Chemical Formula: MnO2
Product Codes: 2526

---

2. Composition/Information on Ingredients

Ingredient	CAS No	Percent	Hazardous
Manganese Dioxide	1313-13-9	72 - 80%	Yes
Quartz	14808-60-7	1 - 3%	Yes
Barium	7440-39-3	1 - 2%	Yes
Nonhazardous Ingredients (Nuisance Particulates)	N/A	15 - 25%	No
Lead	7439-92-1	0 - 0.2%	Yes

---

3. Hazards Identification

Emergency Overview

DANGER! OXIDIZER. CONTACT WITH OTHER MATERIAL MAY CAUSE FIRE. HARMFUL IF SWALLOWED OR INHALED. AFFECTS LUNGS, CENTRAL NERVOUS SYSTEM, BLOOD AND KIDNEYS. MAY CAUSE IRRITATION TO EYES AND RESPIRATORY TRACT. MAY AFFECT THE GUM TISSUE AND REPRODUCTIVE SYSTEM. INHALATION CANCER HAZARD. CONTAINS QUARTZ WHICH CAN CAUSE CANCER. Risk of cancer depends upon duration and level of exposure.

SAF-T-DATA(tm) Ratings (Provided here for your convenience)

Health Rating: 3 - Severe (Cancer Causing)
Flammability Rating: 0 - None
Reactivity Rating: 3 - Severe (Oxidizer)

Contact Rating: 2 - Moderate
Lab Protective Equip: GOGGLES & SHIELD; LAB COAT & APRON; VENT HOOD; PROPER GLOVES
Storage Color Code: Yellow (Reactive)

Potential Health Effects

Inhalation:
Inhalation can cause a flu-like illness (metal fume fever). This 24- to 48-hour illness is characterized by chills, fever, aching muscles, dryness in the mouth and throat and headache. May irritate the respiratory tract. May increase the incidence of upper respiratory infections (pneumonia). Absorption of inorganic manganese salts through the lungs is poor but may occur in chronic poisoning. Lead can be absorbed through the respiratory system.
Ingestion:
May cause abdominal pain and nausea. Although they are poorly absorbed through the intestines, inorganic manganese salts may produce hypoglycemia and decreased calcium blood levels should absorption occur. The symptoms of lead poisoning include abdominal pain and spasms, nausea, vomiting, headache. Acute poisoning can lead to muscle weakness, "lead line" on the gums, metallic taste, definite loss of appetite, insomnia, dizziness, high lead levels in blood and urine with shock, coma and death in extreme cases.
Skin Contact:
No adverse effects expected.
Eye Contact:
May cause irritation, redness and pain.
Chronic Exposure:
Chronic manganese poisoning can result from excessive inhalation and ingestion exposure and involves impairment of the central nervous system. Early symptoms include sluggishness, sleepiness, and weakness in the legs. Advanced cases have shown fixed facial expression, emotional disturbances, spastic gait, and falling. Illness closely resembles Parkinson's Disease. Kidney effects, blood changes and manganese psychosis also may occur as a result of chronic exposure. Chronic inhalation exposure can cause lung damage.
Inhalation of quartz is classified as a human carcinogen. Chronic exposure can cause silicosis, a form of lung scarring that can cause shortness of breath, reduced lung function, and in severe cases, death.
Lead is a cumulative poison and exposure even to small amounts can raise the body's content to toxic levels. The symptoms of chronic exposure are like those of ingestion poisoning; restlessness, irritability, visual disturbances, hypertension and gray facial color may also be noted.
Aggravation of Pre-existing Conditions:
Persons with impaired respiratory function, psychiatric or neurological disturbances, and nutritional deficiencies may be more susceptible to the effect of this substance. Inhalation of quartz may increase the progression of tuberculosis; susceptibility is apparently not increased.

---

4. First Aid Measures

Inhalation:
Remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Get medical attention.
Ingestion:
Induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. Get medical attention.
Skin Contact:
Not expected to require first aid measures. Wash exposed area with soap and water. Get medical advice if irritation develops.

Eye Contact:
Immediately flush eyes with plenty of water for at least 15 minutes, lifting upper and lower eyelids occasionally. Get medical attention if irritation persists.

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5. Fire Fighting Measures

Fire:
Not combustible, but substance is a strong oxidizer and its heat of reaction with reducing agents or combustibles may cause ignition. Increases the flammability of any combustible material.
Explosion:
Contact with oxidizable substances may cause extremely violent combustion.
Fire Extinguishing Media:
Dry chemical, foam or carbon dioxide.
Special Information:
In the event of a fire, wear full protective clothing and NIOSH-approved self-contained breathing apparatus with full facepiece operated in the pressure demand or other positive pressure mode.

---

6. Accidental Release Measures

Ventilate area of leak or spill. Keep unnecessary and unprotected people away from area of spill. Wear appropriate personal protective equipment as specified in Section 8. Spills: Pick up and place in a suitable container for reclamation or disposal, using a method that does not generate dust. Keep combustibles (wood, paper, oil, etc.) away from spilled material. US Regulations (CERCLA) require reporting spills and releases to soil, water and air in excess of reportable quantities. The toll free number for the US Coast Guard National Response Center is (800) 424-8802.

---

7. Handling and Storage

Keep in a tightly closed container, stored in a cool, dry, ventilated area. Protect against physical damage and moisture. Isolate from any source of heat or ignition. Avoid storage on wood floors. Separate from incompatibles, combustibles, organic or other readily oxidizable materials. Wear special protective equipment (Sec. 8) for maintenance break-in or where exposures may exceed established exposure levels. Wash hands, face, forearms and neck when exiting restricted areas. Shower, dispose of outer clothing, change to clean garments at the end of the day. Avoid cross-contamination of street clothes. Wash hands before eating and do not eat, drink, or smoke in workplace. Containers of this material may be hazardous when empty since they retain product residues (dust, solids); observe all warnings and precautions listed for the product.

---

8. Exposure Controls/Personal Protection

Airborne Exposure Limits:
- OSHA Permissible Exposure Limit (PEL):
5 mg/m3 (Ceiling) for Manganese
30 mg/m3 (%SiO2+2) for Quartz, total dust
10 mg/m3 (%SiO2+2) for Quartz, respirable fraction
0.5 mg/m3 (TWA) for Barium
0.05 mg/m3 (TWA), 0.03 mg/m3 (Action Level) for Lead
- ACGIH Threshold Limit Value (TLV):
0.2 mg/m3 (TWA) for Manganese
0.05 mg/m3 (TWA), respirable fraction, A2 - Suspected Human Carcinogen.for Quartz

0.5 mg/m3 (TWA), A4 - not classifiable as a human carcinogen, for Barium
0.05 mg/m3 (TWA), A3 - animal carcinogen, for Lead

Ventilation System:

A system of local and/or general exhaust is recommended to keep employee exposures below the Airborne Exposure Limits. Local exhaust ventilation is generally preferred because it can control the emissions of the contaminant at its source, preventing dispersion of it into the general work area. Please refer to the ACGIH document, *Industrial Ventilation, A Manual of Recommended Practices*, most recent edition, for details.

Personal Respirators (NIOSH Approved):

If the exposure limit is exceeded and engineering controls are not feasible, a half facepiece particulate respirator (NIOSH type N95 or better filters) may be worn for up to ten times the exposure limit or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest.. A full-face piece particulate respirator (NIOSH type N100 filters) may be worn up to 50 times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency, or respirator supplier, whichever is lowest. If oil particles (e.g. lubricants, cutting fluids, glycerine, etc.) are present, use a NIOSH type R or P filter. For emergencies or instances where the exposure levels are not known, use a full-facepiece positive-pressure, air-supplied respirator. WARNING: Air-purifying respirators do not protect workers in oxygen-deficient atmospheres. Where respirators are required, you must have a written program covering the basic requirements in the OSHA respirator standard. These include training, fit testing, medical approval, cleaning, maintenance, cartridge change schedules, etc. See 29CFR1910.134 for details.

Skin Protection:

Wear protective gloves and clean body-covering clothing.

Eye Protection:

Use chemical safety goggles. Maintain eye wash fountain and quick-drench facilities in work area.

Other Control Measures:

Clothing contaminated with this material may be an increased fire hazard. Wash contaminated clothing as soon as possible.

---

9. Physical and Chemical Properties

Appearance:

Gray lumps or fine, black to brownish-black powder.

Odor:

Odorless.

Solubility:

Insoluble in water.

Specific Gravity:

5.0

pH:

9 - 10 (10% aqueous slurry)

% Volatiles by volume @ 21C (70F):

0

Boiling Point:

Not applicable.

Melting Point:

> 1539C (> 2802F)

Vapor Density (Air=1):

No information found.

Vapor Pressure (mm Hg):

No information found.

Evaporation Rate (BuAc=1):

No information found.

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10. Stability and Reactivity

Stability:
Stable under ordinary conditions of use and storage.
Hazardous Decomposition Products:
Toxic metal fumes may form when heated to decomposition.
Hazardous Polymerization:
Will not occur.
Incompatibilities:
Easily oxidizable materials, sulfur, sulfides, phosphids, hypophosphites, chlorates, peroxides, aluminum powder, rubidium acetylide, potassium azide, chlorine trifluoride. Reacts with hydrochloric acid to form corrosive chlorine gas. Heating or rubbing this material with organic materials can cause a fire hazard.
Conditions to Avoid:
Heat, flames, ignition sources and incompatibles.

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11. Toxicological Information

Toxicological Data:
Manganese Dioxide: LD50 oral rat > 3478 mg/kg. Investigated as a reproductive effector. Quartz: Investigated as a tumorigen and mutagen. Lead: Investigated as a tumorigen, mutagen, reproductive effector.
Reproductive Toxicity:
For manganese metal:
May damage the reproductive system. Has shown teratogenic effects in laboratory animals. Lead and other smelter emissions are human reproductive hazards. (Chemical Council on Environmental Quality; Chemical Hazards to Human Reproduction, 1981).
Carcinogenicity:
Quartz: NIOSH considers this substance to be a potential occupational carcinogen.
For lead and inorganic lead compounds:
EPA / IRIS classification: Group B2 - Probable human carcinogen, sufficient animal evidence.

--------\Cancer Lists\----------

Ingredient	---NTP Carcinogen--- Known	Anticipated	IARC Category
Manganese Dioxide (1313-13-9)	No	No	None
Quartz (14808-60-7)	Yes	No	1
Barium (7440-39-3)	No	No	None
Nonhazardous Ingredients (Nuisance Particulates)	No	No	None
Lead (7439-92-1)	No	No	2B

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12. Ecological Information

Environmental Fate:
No information found.
Environmental Toxicity:
No information found.

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13. Disposal Considerations

Whatever cannot be saved for recovery or recycling should be managed in an appropriate and approved waste facility. Although not a listed RCRA hazardous waste, this material may exhibit one or more characteristics of a hazardous waste and require appropriate analysis to determine specific disposal requirements. Processing, use or contamination of this product may change the waste management options. State and local disposal regulations may differ from federal disposal regulations. Dispose of container and unused contents in accordance with federal, state and local requirements.

---

14. Transport Information

Not regulated.

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15. Regulatory Information

--------\Chemical Inventory Status - Part 1\--------

Ingredient	TSCA	EC	Japan	Australia
Manganese Dioxide (1313-13-9)	Yes	Yes	Yes	Yes
Quartz (14808-60-7)	Yes	Yes	Yes	Yes
Barium (7440-39-3)	Yes	Yes	No	Yes
Nonhazardous Ingredients (Nuisance Particulates)	Yes	Yes	Yes	Yes
Lead (7439-92-1)	Yes	Yes	Yes	Yes

--------\Chemical Inventory Status - Part 2\--------

Ingredient	Korea	--Canada-- DSL	NDSL	Phil.
Manganese Dioxide (1313-13-9)	Yes	Yes	No	Yes
Quartz (14808-60-7)	Yes	Yes	No	Yes
Barium (7440-39-3)	Yes	No	No	Yes
Nonhazardous Ingredients (Nuisance Particulates)	Yes	Yes	No	Yes
Lead (7439-92-1)	Yes	Yes	No	Yes

--------\Federal, State & International Regulations - Part 1\--------

Ingredient	-SARA 302- RQ	TPQ	------SARA 313---- List	Chemical Catg.
Manganese Dioxide (1313-13-9)	No	No	No	Manganese co
Quartz (14808-60-7)	No	No	No	No
Barium (7440-39-3)	No	No	Yes	No
Nonhazardous Ingredients (Nuisance Particulates)	No	No	No	No
Lead (7439-92-1)	No	No	Yes	No

--------\Federal, State & International Regulations - Part 2\----------
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Ingredient	CERCLA	-RCRA- 261.33	-TSCA- 8(d)
Manganese Dioxide (1313-13-9)	1	No	No
Quartz (14808-60-7)	No	No	No
Barium (7440-39-3)	No	No	No
Nonhazardous Ingredients (Nuisance Particulates)	No	No	No
Lead (7439-92-1)	10	No	No

Chemical Weapons Convention: No TSCA 12(b): No CDTA: No
SARA 311/312: Acute: Yes Chronic: Yes Fire: Yes Pressure: No
Reactivity: No (Mixture / Solid)

WARNING:
THIS PRODUCT CONTAINS CHEMICALS KNOWN TO THE STATE OF CALIFORNIA TO CAUSE CANCER AND BIRTH DEFECTS OR OTHER REPRODUCTIVE HARM.

Australian Hazchem Code: 1WE
Poison Schedule: None allocated.
WHMIS:
This MSDS has been prepared according to the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all of the information required by the CPR.

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16. Other Information

NFPA Ratings: Health: **2** Flammability: **0** Reactivity: **1** Other: **Oxidizer**
Label Hazard Warning:
DANGER! OXIDIZER. CONTACT WITH OTHER MATERIAL MAY CAUSE FIRE. HARMFUL IF SWALLOWED OR INHALED. AFFECTS LUNGS, CENTRAL NERVOUS SYSTEM, BLOOD AND KIDNEYS. MAY CAUSE IRRITATION TO EYES AND RESPIRATORY TRACT. MAY AFFECT THE GUM TISSUE AND REPRODUCTIVE SYSTEM. INHALATION CANCER HAZARD. CONTAINS QUARTZ WHICH CAN CAUSE CANCER. Risk of cancer depends upon duration and level of exposure.
Label Precautions:
Keep from contact with clothing and other combustible materials.
Store in a tightly closed container.
Remove and wash contaminated clothing promptly.
Do not get in eyes, on skin, or on clothing.
Wash thoroughly after handling.
Do not breathe dust.
Use only with adequate ventilation.
Label First Aid:
If inhaled, remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Get medical attention. If swallowed, induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. Get medical attention. In case of eye contact, immediately flush eyes with plenty of water for at least 15 minutes. Get medical attention if irritation develops or persists.
Product Use:
Laboratory Reagent.
Revision Information:
MSDS Section(s) changed since last revision of document include: 3, 14.
Disclaimer:

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Prepared by: Environmental Health & Safety
Phone Number: (314) 654-1600 (U.S.A.)

MSDS Number: **B7222** * * * * * *Effective Date:* ***03/15/02*** * * * * * *Supercedes:* ***09/14/00***

METHYL TERT-BUTYL ETHER

1. Product Identification

Synonyms: 2-Methoxy-2-methylpropane; tert-Butyl methyl ether; Methyl 1,1-dimethyl ethyl ether; MTBE
CAS No.: 1634-04-4
Molecular Weight: 88.15
Chemical Formula: C5H12O
Product Codes:
J.T. Baker: 9034, 9042, 9043
Mallinckrodt: 5398

2. Composition/Information on Ingredients

Ingredient	CAS No	Percent	Hazardous
Methyl tert-butyl Ether	1634-04-4	99 - 100%	Yes

3. Hazards Identification

Emergency Overview

DANGER! EXTREMELY FLAMMABLE LIQUID AND VAPOR. VAPOR MAY CAUSE FLASH FIRE. HARMFUL IF SWALLOWED, INHALED OR ABSORBED THROUGH SKIN. MAY AFFECT CENTRAL NERVOUS SYSTEM, BLOOD, AND KIDNEYS. A CENTRAL NERVOUS SYSTEM DEPRESSANT. CAUSES IRRITATION TO SKIN, EYES AND RESPIRATORY TRACT.

J.T. Baker SAF-T-DATA(tm) Ratings (Provided here for your convenience)

Health Rating: 2 - Moderate
Flammability Rating: 4 - Extreme (Flammable)
Reactivity Rating: 2 - Moderate
Contact Rating: 2 - Moderate
Lab Protective Equip: GOGGLES & SHIELD; LAB COAT & APRON; VENT HOOD; PROPER GLOVES; CLASS B EXTINGUISHER
Storage Color Code: Red (Flammable)

Potential Health Effects

Inhalation:
Inhalation of vapor can irritate respiratory tract. Causes central nervous system effects. Breathing high concentrations in air can cause lightheadedness, dizziness, weakness, nausea, headache.
Ingestion:
May cause nausea, vomiting. Other symptoms similar to inhalation may occur. Laryngeal, ocular, and respiratory muscles are affected in severe poisoning.
Skin Contact:
A mild skin irritant which causes loss of natural oils. May be a route of absorption into the body.
Eye Contact:
Vapors can irritate eyes; splashes may cause damage to eye tissue.
Chronic Exposure:
Symptoms noted above may be produced by cumulative exposure.
Aggravation of Pre-existing Conditions:
Persons with pre-existing skin disorders or eye problems or impaired respiratory function may be more susceptible to the effects of the substance.

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4. First Aid Measures

Inhalation:
Remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Call a physician.
Ingestion:
Induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. Get medical attention.
Skin Contact:
Remove any contaminated clothing. Wash skin with soap and water for at least 15 minutes. Get medical attention if irritation develops or persists.
Eye Contact:
Immediately flush eyes with plenty of water for at least 15 minutes, lifting lower and upper eyelids occasionally. Get medical attention immediately.

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5. Fire Fighting Measures

Fire:
Flash point: -27C (-17F)
Autoignition temperature: 435C (815F)
Flammable limits in air % by volume:
lel: 1.6; uel: 8.4
Extremely Flammable Liquid and Vapor! Vapor may cause flash fire.
Explosion:
Above the flash point, explosive vapor-air mixtures may be formed. Vapors can flow along surfaces to distant ignition source and flash back. Sealed containers may rupture when heated. Sensitive to static discharge.
Fire Extinguishing Media:
Water spray, dry chemical, alcohol foam, or carbon dioxide. Water spray may be used to keep fire exposed containers cool.
Special Information:
In the event of a fire, wear full protective clothing and NIOSH-approved self-contained breathing apparatus with full facepiece operated in the pressure demand or other positive pressure mode.

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6. Accidental Release Measures

Ventilate area of leak or spill. Remove all sources of ignition. Wear appropriate personal protective equipment as specified in Section 8. Isolate hazard area. Keep unnecessary and unprotected personnel from entering. Contain and recover liquid when possible. Use non-sparking tools and equipment. Collect liquid in an appropriate container or absorb with an inert material (e. g., vermiculite, dry sand, earth), and place in a chemical waste container. Do not use combustible materials, such as saw dust. Do not flush to sewer! If a leak or spill has not ignited, use water spray to disperse the vapors, to protect personnel attempting to stop leak, and to flush spills away from exposures. US Regulations (CERCLA) require reporting spills and releases to soil, water and air in excess of reportable quantities. The toll free number for the US Coast Guard National Response Center is (800) 424-8802.

J. T. Baker SOLUSORB® solvent adsorbent is recommended for spills of this product.

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7. Handling and Storage

Protect against physical damage. Store in a cool, dry well-ventilated location, away from any area where the fire hazard may be acute. Outside or detached storage is preferred. Separate from incompatibles. Containers should be bonded and grounded for transfers to avoid static sparks. Storage and use areas should be No Smoking areas. Use non-sparking type tools and equipment, including explosion proof ventilation. Containers of this material may be hazardous when empty since they retain product residues (vapors, liquid); observe all warnings and precautions listed for the product.

---

8. Exposure Controls/Personal Protection

Airborne Exposure Limits:
-ACGIH Threshold Limit Value (TLV): 50 ppm (TWA), A3 - Confirmed animal carcinogen with unknown relevance to humans
Ventilation System:
A system of local and/or general exhaust is recommended to keep employee exposures below the Airborne Exposure Limits. Local exhaust ventilation is generally preferred because it can control the emissions of the contaminant at its source, preventing dispersion of it into the general work area. Please refer to the ACGIH document, *Industrial Ventilation, A Manual of Recommended Practices*, most recent edition, for details. Use explosion-proof equipment.
Personal Respirators (NIOSH Approved):
If the exposure limit is exceeded and engineering controls are not feasible, a half-face organic vapor respirator may be worn for up to ten times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest. A full-face piece organic vapor respirator may be worn up to 50 times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest. For emergencies or instances where the exposure levels are not known, use a full-face piece positive-pressure, air-supplied respirator. WARNING: Air-purifying respirators do not protect workers in oxygen-deficient atmospheres. Where respirators are required, you must have a written program covering the basic requirements in the OSHA respirator standard. These include training, fit testing, medical approval, cleaning, maintenance, cartridge change schedules, etc. See 29CFR1910.134 for details.
Skin Protection:
Wear protective gloves and clean body-covering clothing.
Eye Protection:
Use chemical safety goggles and/or a full face shield where splashing is possible. Maintain eye wash fountain and quick-drench facilities in work area.

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9. Physical and Chemical Properties

Appearance:
Clear, colorless liquid.
Odor:
Characteristic ethereal odor.
Solubility:
4.8 g/100g of water.
Specific Gravity:
0.74
pH:
No information found.
% Volatiles by volume @ 21C (70F):
100
Boiling Point:
55C (131F)
Melting Point:
-110C (-166F)
Vapor Density (Air=1):
No information found.
Vapor Pressure (mm Hg):
245 @ 25C (77F)
Evaporation Rate (BuAc=1):
No information found.

---

10. Stability and Reactivity

Stability:
Stable under ordinary conditions of use and storage. Unstable in acid solutions.
Hazardous Decomposition Products:
Carbon dioxide and carbon monoxide may form when heated to decomposition.
Hazardous Polymerization:
Will not occur.
Incompatibilities:
Oxidizers, acids.
Conditions to Avoid:
Heat, flames, ignition sources and incompatibles.

---

11. Toxicological Information

Methyl tert butyl ether: Oral rat LD50: 4 gm/kg; inhalation rat LC50: 23576 ppm/4H.

--------\Cancer Lists\----------

Ingredient	---NTP Carcinogen--- Known	Anticipated	IARC Category
Methyl tert-butyl Ether (1634-04-4)	No	No	3

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12. Ecological Information

Environmental Fate:
When released into the soil, this material is not expected to biodegrade. When released into the air,

this material is expected to adversely affect the ozone layer. When released into the soil, this material is expected to quickly evaporate. When released to water, this material is expected to quickly evaporate. When released into the water, this material is expected to have a half-life between 1 and 10 days. This material has an estimated bioconcentration factor (BCF) of less than 100. This material is not expected to significantly bioaccumulate. When released into the air, this material is expected to be readily degraded by reaction with photochemically produced hydroxyl radicals. When released into the air, this material is not expected to be degraded by photolysis. When released into the air, this material is expected to have a half-life between 1 and 10 days.
Environmental Toxicity:
No information found.

---

13. Disposal Considerations

Whatever cannot be saved for recovery or recycling should be handled as hazardous waste and sent to a RCRA approved incinerator or disposed in a RCRA approved waste facility. Processing, use or contamination of this product may change the waste management options. State and local disposal regulations may differ from federal disposal regulations. Dispose of container and unused contents in accordance with federal, state and local requirements.

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14. Transport Information

Domestic (Land, D.O.T.)

Proper Shipping Name: METHYL TERT-BUTYL ETHER
Hazard Class: 3
UN/NA: UN2398
Packing Group: II
Information reported for product/size: 335LB

International (Water, I.M.O.)

Proper Shipping Name: METHYL TERT-BUTYL ETHER
Hazard Class: 3
UN/NA: UN2398
Packing Group: II
Information reported for product/size: 335LB

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15. Regulatory Information

--------\Chemical Inventory Status - Part 1\--------------------------------

Ingredient	TSCA	EC	Japan	Australia
Methyl tert-butyl Ether (1634-04-4)	Yes	Yes	Yes	Yes

--------\Chemical Inventory Status - Part 2\--------------------------------

		--Canada--		
Ingredient	Korea	DSL	NDSL	Phil.
Methyl tert-butyl Ether (1634-04-4)	Yes	Yes	No	Yes

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  --------\Federal, State & International Regulations - Part 1\--------------
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                                                -SARA 302-    ------SARA 313----
--
  Ingredient                                    RQ    TPQ     List  Chemical
Catg.
  ----------------------------------------  ---   -----   ----  ------------
--
  Methyl tert-butyl Ether (1634-04-4)       No    No      Yes        No

  --------\Federal, State & International Regulations - Part 2\--------------
--
                                                                -RCRA-    -TSCA-
  Ingredient                                    CERCLA     261.33     8(d)
  ----------------------------------------  ------     ------    ------
  Methyl tert-butyl Ether (1634-04-4)       1000       No         No


Chemical Weapons Convention:  No     TSCA 12(b):  Yes    CDTA:  No
SARA 311/312:  Acute: Yes      Chronic: Yes  Fire: Yes Pressure: No
Reactivity: No          (Pure / Liquid)
```

Australian Hazchem Code: 3[Y]E
Poison Schedule: None allocated.
WHMIS:
This MSDS has been prepared according to the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all of the information required by the CPR.

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16. Other Information

NFPA Ratings: Health: **2** Flammability: **4** Reactivity: **0**
Label Hazard Warning:
DANGER! EXTREMELY FLAMMABLE LIQUID AND VAPOR. VAPOR MAY CAUSE FLASH FIRE. HARMFUL IF SWALLOWED, INHALED OR ABSORBED THROUGH SKIN. MAY AFFECT CENTRAL NERVOUS SYSTEM, BLOOD, AND KIDNEYS. A CENTRAL NERVOUS SYSTEM DEPRESSANT. CAUSES IRRITATION TO SKIN, EYES AND RESPIRATORY TRACT.
Label Precautions:
Keep away from heat, sparks and flame.
Avoid contact with eyes, skin and clothing.
Avoid breathing vapor.
Keep container closed.
Use only with adequate ventilation.
Wash thoroughly after handling.
Label First Aid:
If swallowed, induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. If inhaled, remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. In case of contact, immediately flush eyes or skin with plenty of water for at least 15 minutes. Remove contaminated clothing and shoes. Wash clothing before reuse. In all cases call a physician.
Product Use:
Laboratory Reagent.
Revision Information:

MSDS Section(s) changed since last revision of document include: 8.

Disclaimer:

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Prepared by: Environmental Health & Safety
Phone Number: (314) 654-1600 (U.S.A.)

BANGS LABORATORIES, INC -- POLYSTYRENE OR POLYSTYRENE SURFACTANT FREE

==========

MSDS Safety Information

==========

FSC: 6850
MSDS Date: 05/28/1997
MSDS Num: CJQVN
LIIN: 00N092276
Product ID: POLYSTYRENE OR POLYSTYRENE SURFACTANT FREE
MFN: 01
Responsible Party
Cage: TO292
Name: BANGS LABORATORIES, INC
Address: 9025 TECHNOLOGY DRIVE
City: FISHERS IN 46038
Info Phone Number: 317-570-7020
Emergency Phone Number: 317-570-7020
Review Ind: Y
Published: Y

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Contractor Summary

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Cage: TO292
Name: BANGS LABORATORIES, INC
Address: 9025 TECHNOLOGY DRIVE
City: FISHERS IN 46038
Phone: 317-570-7020

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Item Description Information

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Ingredients

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Cas: 9003-53-6
RTECS #: WL6475000
Name: POLYSTYRENE (PLAIN POLYSTYRENE SUSPENDED IN WATER)

Cas: 7732-18-5
RTECS #: ZC0110000
Name: WATER

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Health Hazards Data

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Route Of Entry Inds - Inhalation: YES
Skin: YES
Ingestion: YES
Carcinogenicity Inds - NTP: NO
IARC: NO
OSHA: NO
Effects of Exposure: EYES: MILD IRRITATION. SKIN CONTACT: CONTACT MAY CAUSE SLIGHT IRRITATION. SHORT EXPOSURE: NO IRRITATION. REPEATED PROLONGED EXPOSURE, ESPECIALY IF CONFINED; MILD IRRITATION, POSSIBLY A MILD SUPERFICIA L BURN. SKIN ABSORPTION: NOT LIKELY TO BE ABSORBED IN TOXIC AMOUNTS. POSSIBLY WEAK SENSITIZER. INGESTION: LOW SINGLE DOSE TOXICITY. INHALATION: NO GUIDE ESTABLISHED. CONSIDERED TO BE LOW IN HAZARD FRO M INHALATION. SYSTEMIC AND

OTHER EFFECTS: NONE KNOWN. HUMAN EFFECTS NOT ESTABLISHED.
Signs And Symptions Of Overexposure: SEE HEALTH HAZARDS.
First Aid: EYES: FLUSH IMMEDIATELY WITH WATER FOR AT LEAST 15 MINUTES IS GOOD SAFETY PRACTICE. MD SHOULD STAIN FOR EVIDENCE OF CORNEAL INJURY. SKIN: WASH OFF IN FLOWING WATER OR SHOWER. WASH CLOTHING BEFORE REUS E. TREAT AS ANY CONTACT DERMATITIS. IF BURN IS PRESENT, TREAT AS ANY THERMAL BURN.
INGESTION:
INDUCE VOMITING IF LARGE AMOUNTS ARE INGESTED. INHALATION: REMOVE TO FRESH AIR IF EFFECTS OCCUR. CONSULT M EDICAL PERSONNEL. SYSTEMIC: NO SPECIFIC ANTIDOTE. TREATMENT BASED ON SOUND JUDGEMENT OF MD & INDIVIDUAL REACTIONS OF PATIENT.

Handling and Disposal

Spill Release Procedures: FLUSH AREA WITH WATER IMMEDIATELY. AVOID UNNECESSARY
EXPOSURE AND CONTACT.
Waste Disposal Methods: DISPOSAL MUST BE I/A/W FEDERAL, STATE & LOCAL REGULATIONS (FP N). MAY PLUG UP SANITARY SEWERS. DIVERT TO POND OR BURN SOLID
WASTE IN AN ADEQUATE INCINERATOR. FLUSH SEWERS WITH LARGE AMOUNTS OF WATER.
Handling And Storage Precautions: STORE AT ROOM TEMPERATURES BETWEEN 4C AND 5C.
MATERIAL MAY DEVELOP BACTERIA ODOR ON LONG TERM STORAGE. NO SAFETY PROBLEMS KNOWN.

Fire and Explosion Hazard Information

Extinguishing Media: WATER FOG - DRIED RESIN ONLY.
Fire Fighting Procedures: USE NIOSH APPROVED SCBA AND FULL PROTECTIVE EQUIPMENT
(FP N).
Unusual Fire/Explosion Hazard: THE DRIED RESIN IS FLAMMABLE SIMILAR TO WOOD. BURNING DRY RESIN EMITS DENSE, BLACK SMOKE. SUSPENDED MATERIAL IS NOT FLAMMABLE.

Control Measures

Respiratory Protection: NONE NORMALLY NEEDED. IN CASES WHERE THERE IS A LIKELIHOOD OF INHALATION EXPOSURE TO DRIED PARTICLES, WEAR A NIOSH APPROVED DUST RESPIRATOR.
Ventilation: GOOD ROOM VENTILATION USUALLY ADEQUATE FOR MOST OPERATIONS.
Protective Gloves: IMPERVIOUS GLOVES (FP N).
Eye Protection: ANSI APPROVED CHEMICAL WORKERS GOGGLES (FP N).
Other Protective Equipment: ANSI APPROVED EYE WASH & DELUGE SHOWER (FP N).

Physical/Chemical Properties

Boiling Point: =100.C, 212.F
Spec Gravity: 0.95-1.05 G/CC
Solubility in Water: EMULSION
Appearance and Odor: MILKY WHITE LIQUID EMULSION.

Reactivity Data

Stability Indicator: YES

Stability Condition To Avoid: MAY COAGULATE IF FROZEN AT 0C/32F. DRIED RESIN IS
COMBUSTIBLE. ADDITION OF CHEMICALS MAY CAUSE COAGULATION.
Hazardous Decomposition Products: IF BURNED, PRODUCES A DENSE BLACK SMOKE AND NOXIOUS GASES (CARBON MONOXIDE AND HYDROCARBONS).
Hazardous Polymerization Indicator: NO

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Toxicological Information
==========
Toxicological Information: DATA NOT YET AVAILABLE.
==========
Ecological Information
==========
Ecological: DATA NOT YET AVAILABLE. WILL COLOR STREAMS AND RIVERS TO A MILKY WHITE. HAS PRACTICALLY NO BIOLOGICAL DEMAND BUT WILL SETTLE OUT AND FORM SLUDGE OR FILM.
==========
MSDS Transport Information
==========
Transport Information: CONTACT BANGS LABORATORIES, INC FOR TRANSPORTATION INFORMATION.
==========
Regulatory Information
==========
==========
Other Information
==========
Other Information: MFR'S FAX NUMBER: 317-570-7034.
==========
HAZCOM Label
==========
Product ID: POLYSTYRENE OR POLYSTYRENE SURFACTANT FREE
Cage: TO292
Assigned IND: Y
Company Name: BANGS LABORATORIES, INC
Street: 9025 TECHNOLOGY DRIVE
City: FISHERS IN
Zipcode: 46038
Health Emergency Phone: 317-570-7020
Label Required IND: Y
Date Of Label Review: 10/25/1999
Status Code: A
Origination Code: F
Chronic Hazard IND: Y
Eye Protection IND: YES
Skin Protection IND: YES
Signal Word: CAUTION
Respiratory Protection IND: YES
Health Hazard: Slight
Contact Hazard: Slight
Fire Hazard: None
Reactivity Hazard: None
Hazard And Precautions: ACUTE: EYES: MILD IRRITATION. SKIN CONTACT: CONTACT MAY
CAUSE SLIGHT IRRITATION. SHORT EXPOSURE: NO IRRITATION. SKIN ABSORPTION: NOT

LIKELY TO BE ABSORBED IN TOXIC AMOUNTS INGESTION: LOW SINGLE DOSE TOXICITY.
INHALATION: NO GUIDE ESTABLISHED. CONSIDERED TO BE LOW IN HAZARD FROM INHALATION. SYSTEMIC AND OTHER EFFECTS: NONE KNOWN. HUMAN EFFECTS NOT ESTABLISHED. CHRONIC: POSSIBLY WEAK SENSITIZER. S KIN: REPEATED PROLONGED EXPOSURE, ESPECIALLY IF CONFINED; MILD IRRITATION, POSSIBLY A MILD SUPERFICIAL BURN.

==========

MSDS Number: **P5631** * * * * * *Effective Date:* ***11/02/01*** * * * * * *Supercedes:* ***11/17/99***

POTASSIUM CHLORIDE

1. Product Identification

Synonyms: Potassium monochloride
CAS No.: 7447-40-7
Molecular Weight: 74.55
Chemical Formula: KCl
Product Codes:
J.T. Baker: 3040, 3045, 3046, 3052, 4001, 4920, 5596
Mallinckrodt: 0865, 0890, 3279, 3610, 3619, 3925, 4251, 4687, 4858, 4910, 5480, 6156, 6205, 6230, 6275, 6307, 6335, 6363, 6788, 6801, 6838, 6841, 6842, 6845, 6849, 6858, 7207, 7535, 7590, 7618, 7769, V483

2. Composition/Information on Ingredients

Ingredient	CAS No	Percent	Hazardous
Potassium Chloride	7447-40-7	100%	Yes

3. Hazards Identification

Emergency Overview

CAUTION! MAY BE HARMFUL IF SWALLOWED. MAY CAUSE IRRITATION TO SKIN, EYES, AND RESPIRATORY TRACT.

J.T. Baker SAF-T-DATA(tm) Ratings (Provided here for your convenience)

Health Rating: 0 - None
Flammability Rating: 0 - None
Reactivity Rating: 0 - None
Contact Rating: 1 - Slight
Lab Protective Equip: GOGGLES; LAB COAT
Storage Color Code: Orange (General Storage)

Potential Health Effects

Inhalation:

Inhalation of high concentrations of dust may cause nasal or lung irritation.

Ingestion:

Large quantities can produce gastrointestinal irritation and vomiting. May produce weakness and circulatory problems. May affect heart. In severe cases, ingestion may be fatal.

Skin Contact:

Contact may cause irritation or rash, particularly with moist skin.

Eye Contact:

Potassium chloride is moderate eye irritant. Redness, tearing, possible abrasion can occur.

Chronic Exposure:

No information found.

Aggravation of Pre-existing Conditions:

Persons with impaired kidney function may be more susceptible to the effects of the substance.

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4. First Aid Measures

Inhalation:

Remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen.

Ingestion:

Induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. Call a physician.

Skin Contact:

Remove any contaminated clothing. Wash skin with soap and water for at least 15 minutes. Get medical attention if irritation develops or persists.

Eye Contact:

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes, lifting upper and lower eyelids occasionally. Call a physician if irritation persists.

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5. Fire Fighting Measures

Fire:

Not considered to be a fire hazard.

Explosion:

Not considered to be an explosion hazard.

Fire Extinguishing Media:

Use any means suitable for extinguishing surrounding fire.

Special Information:

In the event of a fire, wear full protective clothing and NIOSH-approved self-contained breathing apparatus with full facepiece operated in the pressure demand or other positive pressure mode.

---

6. Accidental Release Measures

Ventilate area of leak or spill. Wear appropriate personal protective equipment as specified in Section 8. Spills: Pick up and place in a suitable container for reclamation or disposal, using a method that does not generate dust.

---

7. Handling and Storage

Keep in a tightly closed container, stored in a cool, dry, ventilated area. Protect against physical damage. Containers of this material may be hazardous when empty since they retain product residues (dust, solids); observe all warnings and precautions listed for the product.

8. Exposure Controls/Personal Protection

Airborne Exposure Limits:
- OSHA Permissible Exposure Limit (PEL):
15 mg/m3 total dust, 5 mg/m3 respirable fraction for nuisance dusts.
- ACGIH Threshold Limit Value (TLV):
10 mg/m3 total dust containing no asbestos and < 1% crystalline silica for Particulates Not Otherwise Classified (PNOC).

Ventilation System:
A system of local and/or general exhaust is recommended to keep employee exposures below the Airborne Exposure Limits. Local exhaust ventilation is generally preferred because it can control the emissions of the contaminant at its source, preventing dispersion of it into the general work area. Please refer to the ACGIH document, *Industrial Ventilation, A Manual of Recommended Practices*, most recent edition, for details.

Personal Respirators (NIOSH Approved):
If the exposure limit is exceeded and engineering controls are not feasible, a half facepiece particulate respirator (NIOSH type N95 or better filters) may be worn for up to ten times the exposure limit or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest.. A full-face piece particulate respirator (NIOSH type N100 filters) may be worn up to 50 times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency, or respirator supplier, whichever is lowest. If oil particles (e.g. lubricants, cutting fluids, glycerine, etc.) are present, use a NIOSH type R or P filter. For emergencies or instances where the exposure levels are not known, use a full-facepiece positive-pressure, air-supplied respirator. WARNING: Air-purifying respirators do not protect workers in oxygen-deficient atmospheres.

Skin Protection:
Wear protective gloves and clean body-covering clothing.

Eye Protection:
Use chemical safety goggles and/or full face shield where dusting or splashing of solutions is possible. Maintain eye wash fountain and quick-drench facilities in work area.

9. Physical and Chemical Properties

Appearance:
White crystals or powder.

Odor:
Odorless.

Solubility:
28.1 g/100g of water @ 0C.

Density:
1.987

pH:
ca. 7 Saturated aq. sl. @ 15C

% Volatiles by volume @ 21C (70F):
0

Boiling Point:
1500C (2732F) Sublimes.

Melting Point:
772C (1422F)

Vapor Density (Air=1):
No information found.

Vapor Pressure (mm Hg):
No information found.

Evaporation Rate (BuAc=1):
No information found.

---

10. Stability and Reactivity

Stability:
Stable under ordinary conditions of use and storage.
Hazardous Decomposition Products:
Oxides of the contained metal and halogen, possibly also free, or ionic halogen.
Hazardous Polymerization:
Will not occur.
Incompatibilities:
Bromine trifluoride; potassium permanganate plus sulfuric acid.
Conditions to Avoid:
No information found.

---

11. Toxicological Information

Oral rat LD50: 2600 mg/kg; irritation eye rabbit (standard Draize): 500 mg/24 hr mild; investigated as a mutagen.

--------\Cancer Lists\----------

Ingredient	---NTP Carcinogen--- Known	Anticipated	IARC Category
Potassium Chloride (7447-40-7)	No	No	None

---

12. Ecological Information

Environmental Fate:
No information found.
Environmental Toxicity:
No information found.

---

13. Disposal Considerations

Whatever cannot be saved for recovery or recycling should be managed in an appropriate and approved waste disposal facility. Processing, use or contamination of this product may change the waste management options. State and local disposal regulations may differ from federal disposal regulations. Dispose of container and unused contents in accordance with federal, state and local requirements.

---

14. Transport Information

Not regulated.

---

15. Regulatory Information

```
  --------\Chemical Inventory Status - Part 1\-------------------------------
--
  Ingredient                                      TSCA  EC   Japan
Australia
  ----------------------------------------------  ----  ---  -----  --------
-
  Potassium Chloride (7447-40-7)                   Yes  Yes   Yes      Yes

  --------\Chemical Inventory Status - Part 2\-------------------------------
--
                                                          --Canada--
  Ingredient                                      Korea  DSL   NDSL  Phil.
  ----------------------------------------------  -----  ---   ----  -----
  Potassium Chloride (7447-40-7)                   Yes   Yes   No     Yes

  --------\Federal, State & International Regulations - Part 1\--------------
--
                                          -SARA 302-    ------SARA 313----
--
  Ingredient                              RQ    TPQ     List  Chemical
Catg.
  ----------------------------------------  ---   -----   ----  ------------
--
  Potassium Chloride (7447-40-7)          No    No      No         No

  --------\Federal, State & International Regulations - Part 2\--------------
--
                                                    -RCRA-    -TSCA-
  Ingredient                              CERCLA    261.33     8(d)
  ----------------------------------------  ------    ------    ------
  Potassium Chloride (7447-40-7)          No        No         No


Chemical Weapons Convention:  No     TSCA 12(b):  No     CDTA:  No
SARA 311/312:  Acute: Yes      Chronic: No   Fire: No  Pressure: No
Reactivity: No          (Pure / Solid)
```

Australian Hazchem Code: None allocated.
Poison Schedule: None allocated.
WHMIS:
This MSDS has been prepared according to the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all of the information required by the CPR.

---

16. Other Information

NFPA Ratings: Health: **1** Flammability: **0** Reactivity: **0**
Label Hazard Warning:
CAUTION! MAY BE HARMFUL IF SWALLOWED. MAY CAUSE IRRITATION TO SKIN, EYES, AND RESPIRATORY TRACT.
Label Precautions:
Avoid breathing dust.
Keep container closed.
Use with adequate ventilation.

Avoid contact with eyes, skin and clothing.
Wash thoroughly after handling.

Label First Aid:

If swallowed, induce vomiting immediately as directed by medical personnel. Never give anything by mouth to an unconscious person. In case of contact, immediately flush eyes or skin with plenty of water for at least 15 minutes. If irritation develops call a physician. If inhaled, remove to fresh air. Get medical attention for any breathing difficulty.

Product Use:

Laboratory Reagent.

Revision Information:

MSDS Section(s) changed since last revision of document include: 8.

Disclaimer:

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Mallinckrodt Baker, Inc. provides the information contained herein in good faith but makes no representation as to its comprehensiveness or accuracy. This document is intended only as a guide to the appropriate precautionary handling of the material by a properly trained person using this product. Individuals receiving the information must exercise their independent judgment in determining its appropriateness for a particular purpose. MALLINCKRODT BAKER, INC. MAKES NO REPRESENTATIONS OR WARRANTIES, EITHER EXPRESS OR IMPLIED, INCLUDING WITHOUT LIMITATION ANY WARRANTIES OF MERCHANTABILITY, FITNESS FOR A PARTICULAR PURPOSE WITH RESPECT TO THE INFORMATION SET FORTH HEREIN OR THE PRODUCT TO WHICH THE INFORMATION REFERS. ACCORDINGLY, MALLINCKRODT BAKER, INC. WILL NOT BE RESPONSIBLE FOR DAMAGES RESULTING FROM USE OF OR RELIANCE UPON THIS INFORMATION.

****

Prepared by: Environmental Health & Safety
Phone Number: (314) 654-1600 (U.S.A.)

MSDS Number: **P5884** * * * * * *Effective Date:* ***10/31/00*** * * * * * *Supercedes:* ***11/24/97***

POTASSIUM HYDROXIDE

1. Product Identification

Synonyms: Caustic potash; potassium hydrate
CAS No.: 1310-58-3
Molecular Weight: 56.11
Chemical Formula: KOH
Product Codes:
J.T. Baker: 3140, 3141, 3146, 3150, 5685
Mallinckrodt: 6964, 6976, 6984, 7704, 7815

2. Composition/Information on Ingredients

Ingredient	CAS No	Percent	Hazardous
Potassium Hydroxide	1310-58-3	85 - 90%	Yes
Water	7732-18-5	10 - 15%	No

3. Hazards Identification

Emergency Overview

POISON! DANGER! CORROSIVE. CAUSES SEVERE BURNS TO SKIN, EYES, RESPIRATORY TRACT, AND GASTROINTESTINAL TRACT. MATERIAL IS EXTREMELY DESTRUCTIVE TO ALL BODY TISSUES. MAY BE FATAL IF SWALLOWED. HARMFUL IF INHALED.

J.T. Baker SAF-T-DATA(tm) Ratings (Provided here for your convenience)

Health Rating: 3 - Severe (Poison)
Flammability Rating: 0 - None
Reactivity Rating: 2 - Moderate
Contact Rating: 4 - Extreme (Corrosive)
Lab Protective Equip: GOGGLES; LAB COAT; VENT HOOD; PROPER GLOVES
Storage Color Code: White Stripe (Store Separately)

Potential Health Effects

Inhalation:
Severe irritant. Effects from inhalation of dust or mist vary from mild irritation to serious damage of the upper respiratory tract, depending on the severity of exposure. Symptoms may include coughing, sneezing, damage to the nasal or respiratory tract. High concentrations can cause lung damage.
Ingestion:
Toxic! Swallowing may cause severe burns of mouth, throat and stomach. Other symptoms may include vomiting, diarrhea. Severe scarring of tissue and death may result. Estimated lethal dose: 5 grams.
Skin Contact:
Corrosive! Contact with skin can cause irritation or severe burns and scarring with greater exposures.
Eye Contact:
Highly Corrosive! Causes irritation of eyes with tearing, redness, swelling. Greater exposures cause severe burns with possible blindness resulting.
Chronic Exposure:
Prolonged contact with dilute solutions or dust of potassium hydroxide has a destructive effect on tissue.
Aggravation of Pre-existing Conditions:
Persons with pre-existing skin disorders or eye problems or impaired respiratory function may be more susceptible to the effects of the substance.

---

4. First Aid Measures

Inhalation:
Remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Call a physician.
Ingestion:
If swallowed, DO NOT INDUCE VOMITING. Give large quantities of water. Never give anything by mouth to an unconscious person. Get medical attention immediately.
Skin Contact:
In case of contact, immediately flush skin with plenty of water for at least 15 minutes while removing contaminated clothing and shoes. Wash clothing before reuse. Thoroughly clean shoes before reuse. Get medical attention immediately.
Eye Contact:
Immediately flush eyes with plenty of water for at least 15 minutes, lifting lower and upper eyelids occasionally. Get medical attention immediately.

---

5. Fire Fighting Measures

Fire:
Not combustible, but contact with water or moisture may generate enough heat to ignite combustibles.
Explosion:
Can react with chemically reactive metals such as aluminum, zinc, magnesium, copper, etc. to release hydrogen gas which can form explosive mixtures with air.
Fire Extinguishing Media:
Use any means suitable for extinguishing surrounding fire.
Special Information:
Solution process causes formation of corrosive mists. Hot or molten material can react violently with water. In the event of a fire, wear full protective clothing and NIOSH-approved self-contained breathing apparatus with full facepiece operated in the pressure demand or other positive pressure mode.

6. Accidental Release Measures

Ventilate area of leak or spill. Keep unnecessary and unprotected people away from area of spill. Wear appropriate personal protective equipment as specified in Section 8. Spills: Pick up and place in a suitable container for reclamation or disposal, using a method that does not generate dust.
Do not flush caustic residues to the sewer. Residues from spills can be diluted with water, neutralized with dilute acid such as acetic, hydrochloric or sulfuric. Absorb neutralized caustic residue on clay, vermiculite or other inert substance and package in a suitable container for disposal.
US Regulations (CERCLA) require reporting spills and releases to soil, water and air in excess of reportable quantities. The toll free number for the US Coast Guard National Response Center is (800) 424-8802.

J. T. Baker NEUTRACIT®-2 or BuCAIM® caustic neutralizers are recommended for spills of solutions of this product.

7. Handling and Storage

Keep in a tightly closed container, stored in a cool, dry, ventilated area. Protect against physical damage. Isolate from incompatible substances. Protect from moisture. Addition to water releases heat which can result in violent boiling and spattering. Always add slowly and in small amounts. Never use hot water. Containers of this material may be hazardous when empty since they retain product residues (dust, solids); observe all warnings and precautions listed for the product.

8. Exposure Controls/Personal Protection

Airborne Exposure Limits:
- OSHA Permissible Exposure Limit (PEL):
2 mg/m3 Ceiling
- ACGIH Threshold Limit Value (TLV):
2 mg/m3 Ceiling

Ventilation System:
A system of local and/or general exhaust is recommended to keep employee exposures below the Airborne Exposure Limits. Local exhaust ventilation is generally preferred because it can control the emissions of the contaminant at its source, preventing dispersion of it into the general work area. Please refer to the ACGIH document, *Industrial Ventilation, A Manual of Recommended Practices*, most recent edition, for details.
Personal Respirators (NIOSH Approved):
If the exposure limit is exceeded, a half-face dust/mist respirator may be worn for up to ten times the exposure limit or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest. A full-face piece dust/mist respirator may be worn up to 50 times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency, or respirator supplier, whichever is lowest. For emergencies or instances where the exposure levels are not known, use a full-facepiece positive-pressure, air-supplied respirator. WARNING: Air-purifying respirators do not protect workers in oxygen-deficient atmospheres.
Skin Protection:
Rubber or neoprene gloves and additional protection including impervious boots, apron, or coveralls, as needed in areas of unusual exposure.
Eye Protection:

Use chemical safety goggles and/or a full face shield where splashing is possible. Maintain eye wash fountain and quick-drench facilities in work area.

---

9. Physical and Chemical Properties

Appearance:
White deliquescent solid
Odor:
Odorless.
Solubility:
52.8% in water @ 20C (68F)
Specific Gravity:
2.04
pH:
13.5 (0.1 molar solution)
% Volatiles by volume @ 21C (70F):
0
Boiling Point:
1320C (2408F)
Melting Point:
360C (680F)
Vapor Density (Air=1):
No information found.
Vapor Pressure (mm Hg):
1.0 @ 714C (1317F)
Evaporation Rate (BuAc=1):
No information found.

---

10. Stability and Reactivity

Stability:
Stable under ordinary conditions of use and storage.
Hazardous Decomposition Products:
Carbon monoxide when reacting with carbohydrates, and hydrogen gas when reacting with aluminum, zinc and tin. Thermal oxidation can produce toxic fumes of potassium oxide (K2O).
Hazardous Polymerization:
Will not occur.
Incompatibilities:
Contact with water, acids, flammable liquids and organic halogen compounds, especially trichloroethylene, may cause fire or explosion. Contact with nitromethane and other similar nitro compounds cause formation of shock sensitive salts. Contact with metals such as aluminum, tin and zinc causes formation of flammable hydrogen gas.
Conditions to Avoid:
Heat, moisture, incompatibles.

---

11. Toxicological Information

For potassium hydroxide: Oral rat LD50: 273 mg/kg; Investigated as a mutagen. Skin Irritation Data (std Draize, 50 mg/24 H): Human, Severe; Rabbit, Severe. Eye Irritation Data(Rabbit, non-std test,1 mg/24 H, rinse): Moderate.

```
  --------\Cancer Lists\-------------------------------------------------------
-
                                          ---NTP Carcinogen---
  Ingredient                              Known    Anticipated    IARC
Category
  ----------------------------------      -----    -----------    ------------
-
  Potassium Hydroxide (1310-58-3)          No          No            None
  Water (7732-18-5)                        No          No            None
```

---

12. Ecological Information

Environmental Fate:
No information found.
Environmental Toxicity:
Potassium Hydroxide: TLm: 80 ppm/Mosquito fish/ 24 hr./ Fresh water

---

13. Disposal Considerations

Whatever cannot be saved for recovery or recycling should be handled as hazardous waste and sent to a RCRA approved waste facility. Processing, use or contamination of this product may change the waste management options. State and local disposal regulations may differ from federal disposal regulations. Dispose of container and unused contents in accordance with federal, state and local requirements.

---

14. Transport Information

Domestic (Land, D.O.T.)

Proper Shipping Name: POTASSIUM HYDROXIDE, SOLID
Hazard Class: 8
UN/NA: UN1813
Packing Group: II
Information reported for product/size: 110LB

International (Water, I.M.O.)

Proper Shipping Name: POTASSIUM HYDROXIDE, SOLID
Hazard Class: 8
UN/NA: UN1813
Packing Group: II
Information reported for product/size: 110LB

International (Air, I.C.A.O.)

Proper Shipping Name: POTASSIUM HYDROXIDE, SOLID
Hazard Class: 8
UN/NA: UN1813
Packing Group: II
Information reported for product/size: 110LB

---

15. Regulatory Information

```
  --------\Chemical Inventory Status - Part 1\-------------------------------
--
  Ingredient                                        TSCA  EC   Japan
Australia
  ------------------------------------------------  ----  ---  -----  --------
-
  Potassium Hydroxide (1310-58-3)                    Yes  Yes   Yes      Yes
  Water (7732-18-5)                                  Yes  Yes   Yes      Yes

  --------\Chemical Inventory Status - Part 2\-------------------------------
--
                                                           --Canada--
  Ingredient                                        Korea  DSL   NDSL  Phil.
  ------------------------------------------------  -----  ---   ----  -----
  Potassium Hydroxide (1310-58-3)                    Yes   Yes   No     Yes
  Water (7732-18-5)                                  Yes   Yes   No     Yes

  --------\Federal, State & International Regulations - Part 1\--------------
--
                                          -SARA 302-    ------SARA 313----
--
  Ingredient                              RQ    TPQ     List  Chemical
Catg.
  --------------------------------------  ---   -----   ----  ------------
--
  Potassium Hydroxide (1310-58-3)         No    No      No         No
  Water (7732-18-5)                       No    No      No         No

  --------\Federal, State & International Regulations - Part 2\--------------
--
                                                    -RCRA-    -TSCA-
  Ingredient                              CERCLA    261.33    8(d)
  --------------------------------------  ------    ------    ------
  Potassium Hydroxide (1310-58-3)         1000      No         No
  Water (7732-18-5)                       No        No         No


Chemical Weapons Convention:  No     TSCA 12(b):  No     CDTA:  No
SARA 311/312:  Acute: Yes      Chronic: Yes  Fire: No  Pressure: No
Reactivity: Yes         (Mixture / Solid)
```

Australian Hazchem Code: 2R
Poison Schedule: S6
WHMIS:
This MSDS has been prepared according to the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all of the information required by the CPR.

---

16. Other Information

NFPA Ratings: Health: **3** Flammability: **0** Reactivity: **1**
Label Hazard Warning:
POISON! DANGER! CORROSIVE. CAUSES SEVERE BURNS TO SKIN, EYES, RESPIRATORY TRACT, AND GASTROINTESTINAL TRACT. MATERIAL IS EXTREMELY DESTRUCTIVE TO ALL BODY TISSUES. MAY BE FATAL IF

SWALLOWED. HARMFUL IF INHALED.

Label Precautions:

Do not get in eyes, on skin, or on clothing.
Do not breathe dust.
Keep container closed.
Use only with adequate ventilation.
Wash thoroughly after handling.

Label First Aid:

If swallowed, DO NOT INDUCE VOMITING. Give large quantities of water. Never give anything by mouth to an unconscious person. In case of contact, immediately flush eyes or skin with plenty of water for at least 15 minutes while removing contaminated clothing and shoes. Wash clothing before reuse. If inhaled, remove to fresh air. If not breathing give artificial respiration. If breathing is difficult, give oxygen. In all cases get medical attention immediately.

Product Use:

Laboratory Reagent.

Revision Information:

No changes.

Disclaimer:

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Mallinckrodt Baker, Inc. provides the information contained herein in good faith but makes no representation as to its comprehensiveness or accuracy. This document is intended only as a guide to the appropriate precautionary handling of the material by a properly trained person using this product. Individuals receiving the information must exercise their independent judgment in determining its appropriateness for a particular purpose. MALLINCKRODT BAKER, INC. MAKES NO REPRESENTATIONS OR WARRANTIES, EITHER EXPRESS OR IMPLIED, INCLUDING WITHOUT LIMITATION ANY WARRANTIES OF MERCHANTABILITY, FITNESS FOR A PARTICULAR PURPOSE WITH RESPECT TO THE INFORMATION SET FORTH HEREIN OR THE PRODUCT TO WHICH THE INFORMATION REFERS. ACCORDINGLY, MALLINCKRODT BAKER, INC. WILL NOT BE RESPONSIBLE FOR DAMAGES RESULTING FROM USE OF OR RELIANCE UPON THIS INFORMATION.

****

Prepared by: Environmental Health & Safety
Phone Number: (314) 654-1600 (U.S.A.)

MSDS Number: **S0722** * * * * * *Effective Date:* ***09/14/00*** * * * * * *Supercedes:* ***02/23/99***

SAND, WASHED AND DRIED

1. Product Identification

Synonyms: Agate; Onyx; Quartz; Silica, crystalline quartz; Silicon dioxide
CAS No.: 14808-60-7
Molecular Weight: 60.08
Chemical Formula: SiO2
Product Codes:
J.T. Baker: 3382, 7023
Mallinckrodt: 7062

2. Composition/Information on Ingredients

Ingredient	CAS No	Percent	Hazardous
Quartz	14808-60-7	90 - 100%	Yes

3. Hazards Identification

Emergency Overview

WARNING! HARMFUL IF INHALED. OVEREXPOSURE MAY CAUSE LUNG DAMAGE. MAY CAUSE EYE IRRITATION. INHALATION CANCER HAZARD. CONTAINS QUARTZ WHICH CAN CAUSE CANCER. Risk of cancer depends upon duration and level of exposure.

J.T. Baker SAF-T-DATA(tm) Ratings (Provided here for your convenience)

Health Rating: 3 - Severe (Cancer Causing)

Flammability Rating: 0 - None
Reactivity Rating: 0 - None
Contact Rating: 1 - Slight
Lab Protective Equip: GOGGLES; LAB COAT; VENT HOOD; PROPER GLOVES
Storage Color Code: Orange (General Storage)

Potential Health Effects

Inhalation:
Acute pneumoconiosis from overwhelming exposure to silica dust has occurred. Coughing and irritation of throat are early symptoms.
Ingestion:
No adverse health effects expected.
Skin Contact:
No adverse effects expected.
Eye Contact:
May cause irritation, redness and pain.
Chronic Exposure:
Inhalation of quartz is classified as a human carcinogen. Chronic exposure can cause silicosis, a form of lung scarring that can cause shortness of breath, reduced lung function, and in severe cases, death.
Aggravation of Pre-existing Conditions:
Inhalation may increase the progression of tuberculosis; susceptibility is apparently not increased. Persons with impaired respiratory function may be more susceptible to the effects of this substance. Smoking can increase the risk of lung injury.

---

4. First Aid Measures

Inhalation:
Remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Get medical attention.
Ingestion:
If large amounts were swallowed, give water to drink and get medical advice.
Skin Contact:
Wash exposed area with soap and water. Get medical advice if irritation develops.
Eye Contact:
Wash thoroughly with running water. Get medical advice if irritation develops.

---

5. Fire Fighting Measures

Fire:
Not considered to be a fire hazard.
Explosion:
Not considered to be an explosion hazard.
Fire Extinguishing Media:
Use any means suitable for extinguishing surrounding fire.
Special Information:
In the event of a fire, wear full protective clothing and NIOSH-approved self-contained breathing apparatus with full facepiece operated in the pressure demand or other positive pressure mode.

---

6. Accidental Release Measures

Ventilate area of leak or spill. Wear appropriate personal protective equipment as specified in Section 8. Spills: Sweep up and containerize for reclamation or disposal. Vacuuming or wet sweeping may be used to avoid dust dispersal.

7. Handling and Storage

Keep in a tightly closed container, stored in a cool, dry, ventilated area. Protect against physical damage. Use dustless systems for handling, storage, and clean up so that dust does not exceed the PEL. Use adequate ventilation and dust collection. Practice good housekeeping. Do not allow dust to collect on walls, floors, sills, ledges, machinery, or equipment. Maintain, clean and test respirators in accordance with OSHA regulations. Maintain and test ventilation and dust collection equipment. Wash clothing that has become dusty; do not breathe the dust from clothing. Containers of this material may be hazardous when empty since they retain product residues (dust, solids); observe all warnings and precautions listed for the product.

8. Exposure Controls/Personal Protection

Airborne Exposure Limits:
-OSHA Permissible Exposure Limit (PEL):
Total dust: 30mg/m3/(%SiO2 + 2)
Respirable Fraction: 10 mg/m3/(%SiO2 + 2)

-ACGIH Threshold Limit Value (TLV):
0.05 mg/m3 (TWA) respirable dust, A2 -Suspected Human Carcinogen.

Ventilation System:
A system of local and/or general exhaust is recommended to keep employee exposures below the Airborne Exposure Limits. Local exhaust ventilation is generally preferred because it can control the emissions of the contaminant at its source, preventing dispersion of it into the general work area. Please refer to the ACGIH document, *Industrial Ventilation, A Manual of Recommended Practices*, most recent edition, for details.

Personal Respirators (NIOSH Approved):
If the exposure limit is exceeded and engineering controls are not feasible, a half-face high efficiency particulate respirator (NIOSH type N100 filter) may be worn for up to ten times the exposure limit or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest. A full-face piece high efficiency particulate respirator (NIOSH type N100 filter) may be worn up to 50 times the exposure limit, or the maximum use concentration specified by the appropriate regulatory agency or respirator supplier, whichever is lowest. If oil particles (e.g. lubricants, cutting fluids, glycerine, etc.) are present, use a NIOSH type R or P filter. For emergencies or instances where the exposure levels are not known, use a full-facepiece positive-pressure, air-supplied respirator. WARNING: Air-purifying respirators do not protect workers in oxygen-deficient atmospheres. Where respirators are required, you must have a written program covering the basic requirements in the OSHA respirator standard. These include training, fit testing, medical approval, cleaning, maintenance, cartridge change schedules, etc. See 29CFR1910.134 for details.

Skin Protection:
Wear protective gloves and clean body-covering clothing.

Eye Protection:
Use chemical safety goggles. Maintain eye wash fountain and quick-drench facilities in work area.

9. Physical and Chemical Properties

Appearance:
Fine, off-white granules.
Odor:
Odorless.
Solubility:
Insoluble in water.
Specific Gravity:
2.65
pH:
No information found.
% Volatiles by volume @ 21C (70F):
0
Boiling Point:
2230C (4046F)
Melting Point:
1710C (3110F)
Vapor Density (Air=1):
No information found.
Vapor Pressure (mm Hg):
10 @ 1732C (3150F)
Evaporation Rate (BuAc=1):
Not applicable.

---

10. Stability and Reactivity

Stability:
Stable under ordinary conditions of use and storage.
Hazardous Decomposition Products:
At higher temperatures, can change crystal structure to form tridymite or cristobalite, which have greater health hazards.
Hazardous Polymerization:
Will not occur.
Incompatibilities:
Strong alkalis, hydrofluoric acid, powerful oxidizers and fluorine containing compounds.
Conditions to Avoid:
Dusting and incompatibles.

---

11. Toxicological Information

Toxicological Data:
No LD50/LC50 information found relating to normal routes of occupational exposure.
Investigated as a tumorigen and mutagen.

Carcinogenicity:
Quartz: NIOSH considers this substance to be a potential occupational carcinogen.

--------\Cancer Lists\----------

Ingredient	---NTP Carcinogen--- Known	Anticipated	IARC Category
Quartz (14808-60-7)	Yes	No	1

---

12. Ecological Information

Environmental Fate:
No information found.
Environmental Toxicity:
No information found.

---

13. Disposal Considerations

Whatever cannot be saved for recovery or recycling should be managed in an appropriate and approved waste disposal facility. Processing, use or contamination of this product may change the waste management options. State and local disposal regulations may differ from federal disposal regulations. Dispose of container and unused contents in accordance with federal, state and local requirements.

---

14. Transport Information

Not regulated.

---

15. Regulatory Information

--------\Chemical Inventory Status - Part 1\--------------------------------

Ingredient	TSCA	EC	Japan	Australia
Quartz (14808-60-7)	Yes	Yes	Yes	Yes

--------\Chemical Inventory Status - Part 2\--------------------------------

Ingredient	Korea	--Canada-- DSL	NDSL	Phil.
Quartz (14808-60-7)	Yes	Yes	No	Yes

--------\Federal, State & International Regulations - Part 1\--------------

Ingredient	-SARA 302- RQ	TPQ	------SARA 313---- List	Chemical Catg.
Quartz (14808-60-7)	No	No	No	No

--------\Federal, State & International Regulations - Part 2\--------------

Ingredient	CERCLA	-RCRA- 261.33	-TSCA- 8(d)
Quartz (14808-60-7)	No	No	No

Chemical Weapons Convention: No TSCA 12(b): No CDTA: No

SARA 311/312: Acute: Yes Chronic: Yes Fire: No Pressure: No Reactivity: No (Pure / Solid)

WARNING:
THIS PRODUCT CONTAINS A CHEMICAL(S) KNOWN TO THE STATE OF CALIFORNIA TO CAUSE CANCER.

Australian Hazchem Code: None allocated.
Poison Schedule: None allocated.
WHMIS:
This MSDS has been prepared according to the hazard criteria of the Controlled Products Regulations (CPR) and the MSDS contains all of the information required by the CPR.

---

16. Other Information

NFPA Ratings: Health: **2** Flammability: **0** Reactivity: **0**
Label Hazard Warning:
WARNING! HARMFUL IF INHALED. OVEREXPOSURE MAY CAUSE LUNG DAMAGE. MAY CAUSE EYE IRRITATION. INHALATION CANCER HAZARD. CONTAINS QUARTZ WHICH CAN CAUSE CANCER. Risk of cancer depends upon duration and level of exposure.
Label Precautions:
Do not get in eyes, on skin, or on clothing.
Do not breathe dust.
Keep container closed.
Use only with adequate ventilation.
Minimize dust generation and accumulation.
Wash thoroughly after handling.
Label First Aid:
If inhaled, remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Get medical attention. In case of eye contact, immediately flush eyes with plenty of water for at least 15 minutes. Get medical attention if irritation develops or persists.
Product Use:
Laboratory Reagent.
Revision Information:
MSDS Section(s) changed since last revision of document include: 8, 11.
Disclaimer:
****
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Prepared by: Environmental Health & Safety
Phone Number: (314) 654-1600 (U.S.A.)

JOHNSON MATTHEY ALFA AESAR -- 88220 STRONTIUM OXIDE

MSDS Safety Information

FSC: 6810
NIIN: 01-446-9934
MSDS Date: 08/01/1994
MSDS Num: CFRLZ
Product ID: 88220 STRONTIUM OXIDE
MFN: 01
Responsible Party
Cage: 0MMA6
Name: JOHNSON MATTHEY ALFA AESAR
Address: 30 BOND STREET
City: WARD HILL MA 00000
Info Phone Number: 508-521-6300
Emergency Phone Number: 800-424-9300 CHEMTREC
Preparer's Name: UNKNOWN
Review Ind: Y
Published: Y

Contractor Summary

Cage: 0MMA6
Name: ALFA AESAR (A JOHNSON MATTHEY CO)
Address: 30 BOND STREET
City: WARD HILL MA 01835-0747
Phone: 978-621-6300

Item Description Information

Item Manager: S9G
Item Name: STRONTIUM OXIDE
Specification Number: NONE
Type/Grade/Class: NONE
Unit of Issue: BT
Quantitative Expression: 10000000025GM
UI Container Qty: 0
Type of Container: UNKNOWN

Ingredients

Cas: 1314-11-0
Name: STRONTIUM OXIDE
% Wt: 100
Other REC Limits: NONE RECOMMENDED
OSHA PEL: NOT ESTABLISHED
ACGIH TLV: NOT ESTABLISHED

Health Hazards Data

LD50 LC50 Mixture: NO DATA
Route Of Entry Inds - Inhalation: YES
Skin: YES
Ingestion: YES
Carcinogenicity Inds - NTP: NO
IARC: NO

OSHA: NO
Effects of Exposure: EXPOSURE MAY CAUSE EYE, SKIN & RESPIRATORY TRACT IRRITATION OR BURNS. INGESTION CAUSES IRRITATION OR BURNS OF THE MOUTH, THROAT & GI TRACT.
Explanation Of Carcinogenicity: NO INGREDIENT OF A CONCENTRATION OF 0.1% OR GREATER IS LISTED AS A CARCINOGEN OR SUSPECTED CARCINOGEN.
Signs And Symptions Of Overexposure: SKIN-IRRITATION, BURNS. EYES-IRRITATION, BURNS. INHALED-IRRITATION, BURNS. INGESTED-BURNS/IRRITATION OF MOUTH, THROAT, GI TRACT.
Medical Cond Aggravated By Exposure: NONE KNOWN.
First Aid: EYES-FLUSH WITH WATER FOR 15 MINUTES, LIFT LIDS. GET IMMEDIATE MEDICAL ATTENTION. SKIN-REMOVE CONTAMINATED CLOTHES. FLOOD AREA WITH WATER. GET IMMEDIATE MEDICAL ATTENTION. INHALED-REMOVE TO FRESH AIR. GET IMMEDIATE MEDICAL ATTENTION. INGESTED-GET IMMEDIATE MEDICAL ATTENTION.

Handling and Disposal

Spill Release Procedures: WEARING FULL PROTECTIVE EQUIPMENT, COVER SPILL WITH DRY SAND OR VERMICULITE. MIX WELL AND CAREFULLY TRANSFER TO A CLEAN, DRY CONTAINER.
Neutralizing Agent: NONE SPECIFIED BY MANUFACTURER.
Waste Disposal Methods: DISPOSE OF IN ACCORDANCE WITH LOCAL, STATE AND FEDERAL ENVIRONMENTAL REGULATIONS.
Handling And Storage Precautions: KEEP CONTAINER TIGHTLY CLOSED. STORE IN A COOL, DRY, WELL-VENTILATED AREA. WASH THOROUGHLY AFTER USE.
Other Precautions: NONE SPECIFIED BY MANUFACTURER.

Fire and Explosion Hazard Information

Extinguishing Media: DRY CHEMICAL EXTINGUISHING AGENTS, DRY SAND, DRY DOLOMITE. NO WATER OR CO2 UNLESS MASSIVE FIRE OR ADVANCED.
Fire Fighting Procedures: NORMAL PROCEDURES SUCH AS WEARING SELF-CONTAINED BREATHING APPARATUS AND CHEMICAL RESISTANT CLOTHING.
Unusual Fire/Explosion Hazard: NONE SPECIFIED BY MANUFACTURER.

Control Measures

Respiratory Protection: IF ENGINEERING CONTROLS FAIL OR NON-ROUTINE USE OR AN EMERGENCY OCCURS; WEAR AN MSHA/NIOSH APPROVED RESPIRATOR WITH HEPA CARTRIDGE OR AN AIR-SUPPLIED RESPIRATOR OR SCBA, AS REQUIRED. USE IAW 29 CFR 19 10.134.
Ventilation: GLOVE BAG OR BOX WITH DRY, INERT ATMOSPHERE.
Protective Gloves: RUBBER.
Eye Protection: ANSI APPROVED SAFETY GOGGLES.
Other Protective Equipment: LAB COAT & APRON, FLAME & CHEMICAL RESISTANT COVERALLS, EYE WASH STATION & SAFETY SHOWER, HYGIENIC WASHING FACILITIES.
Work Hygienic Practices: WASH HANDS AFTER HANDLING AND BEFORE EATING, DRINKING, OR SMOKING. LAUNDER CONTAMINATED CLOTHES BEFORE REUSE.

Physical/Chemical Properties

HCC: B2
B.P. Text: 5432F,3000C
M.P/F.P Text: 4406F,2430C
Vapor Pres: 40
Spec Gravity: 4.7
Solubility in Water: SLIGHT
Appearance and Odor: GREY-WHITE POWDER; ODORLESS.
Percent Volatiles by Volume: 0
==========
Reactivity Data
==========
Stability Indicator: YES
Stability Condition To Avoid: THERMAL DECOMPOSITION, INCOMPATIBLES. IN THE PRESENCE OF WATER, MATERIAL MAY EVOLVE ENOUGH HEAT TO IGNITE COMBUSTIBLES.
Materials To Avoid: ACIDS, CARBON DIOXIDE, ALUMINUM, MAGNESIUM, WATER.
Hazardous Decomposition Products: STRONTIUM HYDROXIDE.
Hazardous Polymerization Indicator: NO
Conditions To Avoid Polymerization: WILL NOT OCCUR.
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Toxicological Information
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Ecological Information
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MSDS Transport Information
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Regulatory Information
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Other Information
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Transportation Information
==========
Responsible Party Cage: 0MMA6
Trans ID NO: 137173
Product ID: 88220 STRONTIUM OXIDE
MSDS Prepared Date: 08/01/1994
Review Date: 11/18/1997
MFN: 1
Tech Entry NOS Shipping Nm: CONTAINS STRONTIUM OXIDE
Net Unit Weight: 0.05 LB
Multiple KIT Number: 0
Review IND: Y
Unit Of Issue: BT
Container QTY: 0
Type Of Container: UNKNOWN
Additional Data: PSN IS BEST GUESS BY DLA- STAFF.
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Detail DOT Information
==========
DOT PSN Code: DYB
Symbols: G
DOT Proper Shipping Name: CORROSIVE SOLID, BASIC, ORGANIC, N.O.S.
Hazard Class: 8

UN ID Num: UN3263
DOT Packaging Group: II
Label: CORROSIVE
Packaging Exception: 154
Non Bulk Pack: 212
Bulk Pack: 240
Max Qty Pass: 15 KG
Max Qty Cargo: 50 KG
Vessel Stow Req: B

Detail IMO Information

IMO PSN Code: ETD
IMO Proper Shipping Name: CORROSIVE SOLID, BASIC, INORGANIC, N.O.S. o
IMDG Page Number: 8150-1
UN Number: 3262
UN Hazard Class: 8
IMO Packaging Group: I/II/III
Subsidiary Risk Label: -
EMS Number: 8-15
MED First Aid Guide NUM: 760

Detail IATA Information

IATA PSN Code: HMH
IATA UN ID Num: 3262
IATA Proper Shipping Name: CORROSIVE SOLID, BASIC, INORGANIC, N.O.S. *
IATA UN Class: 8
IATA Label: CORROSIVE
UN Packing Group: II
Packing Note Passenger: 814
Max Quant Pass: 15KG
Max Quant Cargo: 50KG
Packaging Note Cargo: 816

Detail AFI Information

AFI PSN Code: HNC
AFI Symbols: *
AFI Proper Shipping Name: CORROSIVE SOLID, BASIC, INORGANIC, N.O.S.
AFI Hazard Class: 8
AFI UN ID NUM: UN3262
AFI Packing Group: II
Special Provisions: P5
Back Pack Reference: A12.4

HAZCOM Label

Product ID: 88220 STRONTIUM OXIDE
Cage: 0MMA6
Company Name: ALFA AESAR (A JOHNSON MATTHEY CO)
Street: 30 BOND STREET
City: WARD HILL MA
Zipcode: 01835-0747
Health Emergency Phone: 800-424-9300 CHEMTREC
Label Required IND: Y
Date Of Label Review: 11/18/1997

Status Code: C
Label Date: 11/18/1997
Year Procured: 1997
Origination Code: F
Eye Protection IND: YES
Skin Protection IND: YES
Signal Word: DANGER
Respiratory Protection IND: YES
Health Hazard: Moderate
Contact Hazard: Severe
Fire Hazard: None
Reactivity Hazard: Slight
Hazard And Precautions: CORROSIVE! EXPOSURE MAY CAUSE EYE, SKIN & RESPIRATORY TRACT IRRITATION OR BURNS. INGESTION CAUSES IRRITATION OR BURNS OF THE MOUTH, THROAT & GI TRACT. TARGET ORGANS: SKIN, EYES, LUNGS, GI TRACT. FIRST AID: EYES-FLUSH WITH WATER FOR 15 MINUTES, LIFT LIDS. GET IMMEDIATE MEDICAL ATTENTION. SKIN-REMOVE CONTAMINATED CLOTHES. FLOOD AREA WITH WATER. GET IMMEDIATE MEDICAL ATTENTION. INHALED-REMOVE TO FRES H AIR. GET IMMEDIATE MEDICAL ATTENTION. INGESTED-GET IMMEDIATE MEDICAL ATTENTION.
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The Concentration–Response Relation between $PM_{2.5}$ and Daily Deaths

Joel Schwartz,[1,2,3] Francine Laden,[1,3] and Antonella Zanobetti[1]

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Particulate air pollution at commonly occurring concentrations is associated with daily deaths. Recent attention has focused on the shape of the concentration–response curve, particularly at low doses. Several recent articles have reported that particulate matter with aerodynamic diameter ≤ 10 μm (PM_{10}) was associated with daily deaths with no evidence of a threshold. These reports have used smoothing or spline methods in individual cities and pooled the results across multiple cities to obtain estimates that are more robust. To date, fine particulate matter (aerodynamic diameter ≤ 2.5 μm; $PM_{2.5}$), a component of PM_{10}, has not been examined in this regard. We examined this association in a hierarchical model in six U.S. cities. In the first stage, we fit log-linear models including smooth functions of $PM_{2.5}$ in each city, controlling for season, weather, and day of the week. These smooth functions allowed for nonlinearities in the city-specific associations. We combined the estimated curves across cities using a hierarchical model that allows for heterogeneity. We found an essentially linear relationship down to 2 μg/m^3. The same approach was applied to examine the concentration response to traffic particles, controlling for particles from other sources. Once again, the association showed no sign of a threshold. The magnitude of the association suggests that controlling fine particle pollution would result in thousands fewer early deaths per year. *Key words:* **meta-analysis, mortality, particulate air pollution, smoothing, time series, traffic.** *Environ Health Perspect* **110:1025–1029 (2002). [Online 27 August 2002]**
http://ehpnet1.niehs.nih.gov/docs/2002/110p1025-1029schwartz/abstract.html

In the last decade, a series of studies reported associations between daily concentrations of airborne particles and daily deaths (*1–3*). The magnitude of the regression coefficients in those studies indicated that particulate air pollution was associated with between 50 and 100,000 early deaths per year in the United States, and similar numbers were found in Europe. More recently, a number of large, multicity studies (*4–7*) have reported associations between airborne particles, measured in various ways, and daily deaths. The largest study demonstrated that gaseous air pollutants did not confound the association, and that none of the gaseous air pollutants showed an independent effect on daily deaths (*7*). These studies assumed a linear concentration–response relation between airborne particles and daily deaths and did not address the question of what the association looked like for particle constituents, characterized by size, physiochemical composition, or source.

In a recent study of six U.S. cities (*5*), we demonstrated that daily mortality was associated with fine particulate matter (aerodynamic diameter ≤ 2.5 μm; $PM_{2.5}$) and not with coarse particulate matter (aerodynamic diameter between 2.5 and 10 μm; $PM_{2.5–10}$). Each 10 μg/m^3 increase in the 2-day mean concentration of $PM_{2.5}$ was associated with a 1.5% (95% confidence interval, 1.1–1.9%) increase in daily mortality.

Ambient $PM_{2.5}$ consists mainly of combustion particles from motor vehicles and the burning of coal, fuel oil, and wood, but also contains some crustal particles from finely pulverized road dust and soils. These sources produce particles with different characteristics, and the relative toxicity of those sources and characteristics is an area of relative recent but intense interest. In a follow-up study (*8*), we used the elemental composition of size-fractionated particles to identify several distinct source-related fractions of fine particles. We then examined the association of these fractions with daily mortality in each of the six cities and combined the city-specific results in a meta-analysis to derive overall relative risks for each fraction. We found positive associations with particles from traffic, particles from coal, and particles from residual oil combustion when included jointly in the model predicting daily deaths (*8*). The largest effect size was for residual oil particles, followed by traffic particles and then coal particles. Only the latter two associations were statistically significant, however. Again, as traditional, these analyses assumed a linear association between the various particle constituents and daily deaths.

The shape of the concentration–response relationship is critical for public health assessment, and in particular, some have speculated that thresholds might exist.

Recently, three reports have explored this question for particulate air pollution, using multicity studies in the United States. In one study, Daniels et al. (*9*) used data from 20 U.S. cities, five of which had daily measurements of PM_{10}, with the rest having measurements only one day in six. They used regression splines to model the concentration–response curve in each city and combined the results across cities. They found no evidence for a threshold. In fact, the concentration–response relation was quite linear across the entire range of exposure. In another report, Schwartz and Zanobetti (*10*) used data from 10 cities, all of which had daily measurements of PM_{10}, resulting in slightly more days of study than in the first report. They used nonparametric smoothing to model the concentration–response curve between air pollution and daily deaths in each city and combined the results across cities. Again, a linear, no-threshold relationship was seen. Schwartz and Zanobetti also performed simulations to confirm the ability of this approach to detect thresholds and other types of nonlinearity (*10*). Schwartz et al. (*11*), using data from eight Spanish cities, similarly reported a linear association between daily deaths and black smoke, an optical measure of black particles. These results held after adjusting for SO_2. To date, no similar examination of the concentration–response curve has been done for $PM_{2.5}$, or for any source components. Because $PM_{2.5}$ is now the regulated form of particulate air pollution in the United States, we here report results of such an analysis.

Materials and Methods

Air pollution data. As part of the Harvard Six Cities studies (*12*), dichotomous virtual impactor samplers were placed at a central residential monitoring site in six U.S. metropolitan areas: Boston, Massachusetts; Knoxville, Tennessee; St. Louis, Missouri; Stuebenville, Ohio; Madison, Wisconsin; and Topeka, Kansas. Separate filter samples were collected of fine particles ($PM_{2.5}$) and of the coarse mass ($PM_{2.5–10}$) fraction. Integrated 24-hr samples were collected at least every other day from 1979 until the late 1980s, with daily sampling during health survey periods. For fine and coarse particle samples, mass concentration was determined separately by beta-attenuation (*13*). Except for a period

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This work was supported by U.S. EPA Grant R827353 and NIEHS Grant ES 00002.

Received 21 December 2001; accepted 20 March 2002.

between October 1981 and January 1984 in all cities, elemental composition of fine and coarse mass was determined by X-ray fluorescence (*14*). Elemental composition was available on 97% of these samples. In the fine fraction, 15 elements were routinely found above the limit of detection: silicon, sulfur, chlorine, potassium, calcium, vanadium, manganese, aluminum, nickel, zinc, selenium, bromine, lead, copper, and iron.

Source identification. In separate analyses for each city, we used specific rotation factor analysis to identify up to five common factors from the 15 specified elements. We specified a single element as the tracer for each factor and maximized the projection of these elements using the Procrustes rotation, a variant of the oblique rotation method (*15*). The Procrustes method allows us to use known tracers for different sources as targets for the different factors and to maximize their loadings on those factors instead of having factors defined in an entirely data-driven manner. To rescale the factor scores from the normalized scale to the mass scale (in micrograms per cubic meter), we regressed the total daily fine particle concentrations on the daily factor scores for all of the factors in separate regression models for each city and took the product of each factor score with its regression coefficient (*16*). Only sources that were significant predictors of total fine particle mass ($p < 0.10$) were considered in the mortality analyses. Further details have been published previously (*8*).

Meteorologic data. We obtained meteorologic data from the National Center for Atmospheric Research, including hourly measures of temperature, dew point temperature, and precipitation from the National Oceanographic and Atmospheric Administration weather station nearest to each city (*17*). We calculated 24-hr mean values for temperature and dew point temperature.

Mortality data. We defined the six metropolitan areas in this study as the county containing the air pollution monitor and contiguous counties (*5*). We extracted daily deaths from annual detail mortality tapes (National Center for Health Statistics) (*18*) for people who lived and died in the selected counties for the time periods with fine particulate measurements. After excluding all deaths caused by accidents and other external causes [*International Classification of Diseases*, 9th Revision (*ICD-9*) (*19*), clinical modification codes 800–999], we analyzed the remaining total daily deaths.

Poisson regression of mortality. We investigated the association of daily deaths with sources of fine particles separately for each city using Poisson regression in a generalized additive model (GAM) (*20,21*). That is, in each city we assumed

$$\mathrm{Log}\left[E(Y_t)\right] = \beta_o + \sum S_i(X_{it}), \qquad [1]$$

where Y_t is the number of deaths in the city on day t and X_{it} is the value of covariate i on day t. GAMs are distinguished by allowing us to use smooth functions S_i instead of linear terms to control for covariates, such as temperature, that may affect daily deaths in a nonlinear way. Linear functions may be used where appropriate. This approach was introduced for time series of counts in 1994 (*22*) and is now standard (*23,24*).

To control for trend and season, we used a locally weighted linear regression (LOESS) smooth function of date with a span of 0.05 (*25*). For the smooth functions of temperature and dew point temperature, we used LOESS functions with spans of 0.80. Indicator variables for day of the week also were included in the models. This is the identical model used by Schwartz et al. (*5*) and Laden et al. (*8*), and more details are provided there. To these models we added a smooth function of the mean $PM_{2.5}$ concentration on the day of death and the previous day, instead of the linear term previously used by Schwartz et al. (*5*). The smoothing window included 50% of the data, which corresponds to between four and five degrees of freedom for the air pollution relation in each city. Alternatively, we added the estimated mass for each of the source factor scores (in micrograms per cubic meter) simultaneously in the model. That is, the estimate of the mobile source factor is in a model controlling for coal-derived particles, crustal particles, and the other source factors, and vice versa. Because only the particles from traffic showed a strong linear association, and because the exposure ranges for the exposures to coal particles did not overlap sufficiently, we only used a smooth function for the traffic particles and followed Laden et al. (*8*) in treating the particle mass from the other sources as linear terms.

Hierarchical model. To combine the smooth curves across cities, we applied the approach of Schwartz and Zanobetti (*10*), as modified by Schwartz et al. (*11*). In each city, the predicted log relative risk and its pointwise standard error was computed for each 2

Table 1. Mean daily deaths in six U.S. cities and mean concentrations of $PM_{2.5}$ overall, and from the three source categories showing evidence of an association with daily deaths in Laden et al. (*8*).

City	Deaths	$PM_{2.5}$ (μg/m^3)	Traffic (μg/m^3)	Coal (μg/m^3)	Residual oil (μg/m^3)	Dates (month/year)
Boston	59	16.5	4.8	8.3	0.5	5/79–1/86
Knoxville	12	21.1	4.4	6.8	—	1/80–12/87
St. Louis	55	19.2	2.9	5.6	—	9/79–1/87
Steubenville	3	30.5	1.5	19.2	0.9	4/79–9/87
Madison	11	11.3	3.1	4.9	—	3/79–12/97
Topeka	3	12.2	2.1	7.0	—	9/79–10/88

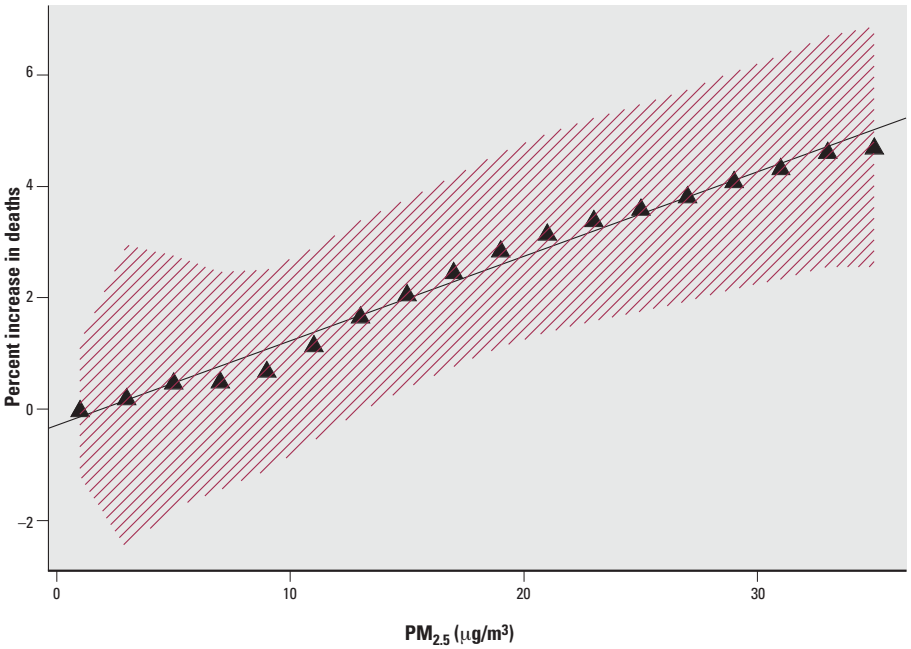

Figure 1. Overall estimated dose–response relation between total $PM_{2.5}$ and daily deaths in six U.S. cities. The estimate is obtained by combining the estimated smoothed curves in each of the cities, after controlling for weather, season, and day of the week. The shaded area indicates the pointwise 95% confidence intervals at each point. The line shown is a least-squares regression line through the estimated points.

μg/m^3 increment in exposure. These estimates are provided by the GAM function in S-plus (MathSoft, Inc., Seattle, WA). To successfully combine data across cities, we need to use a range of exposures that is common to all cities. Because high concentrations of $PM_{2.5}$ were rare, the curves were combined only in the range of 0–35 μg/m^3. The first phase of the analysis produced estimated effect sizes (log relative risks) $\hat{Y}_{ij}$ in each city i for each exposure category j. A pointwise standard error of the estimate is also estimated by GAM. To produce the combined curve, we regressed these estimates against indicator variables for each level, using inverse variance weighting and allowing for a random variance component to capture heterogeneity in the association across cities. That is, we assumed

$$\hat{Y}_{ij} \sim N\left(\beta_1 d_1 + \beta_2 d_2 + \cdots + \beta_k d_k, V_{ij} + \delta\right), \quad [2]$$

where d_j are dummy variables for the j exposure levels, V_{ij} is the estimated variance in city i at level j, and δ is the estimated random variance component.

We used the iterative meta-regression approach of Berkey et al. (*26*) to obtain a maximum likelihood estimate of the random variance component.

The nonparametric smooth functions we use to estimate the shape of the concentration response relation use four to five degrees of freedom, and it is not clear that the source-specific relations can support so many degrees of freedom, which would entail a total of 20 degrees of freedom for all the $PM_{2.5}$ sources. In our previous report (*8*), the relation between $PM_{2.5}$ from traffic and daily deaths was estimated with considerably greater precision than for particles from other sources, most of which were not significant. Further, the range of overlap in exposures across cities was lower for coal, crustal, and residual oil factors. Therefore, in our source-specific models, we only modeled the traffic source particles using a nonparametric smooth, while controlling for $PM_{2.5}$ from the other sources using linear terms, as in Laden et al. (*8*). We then combined the estimated concentration–response relations for traffic particles similarly to what we did for $PM_{2.5}$ from the other sources.

Results

Table 1 shows the daily deaths, $PM_{2.5}$ levels, and estimated concentrations of $PM_{2.5}$ from each source. Figure 1 shows the meta-smooth dose–response relation between $PM_{2.5}$ and daily deaths in the six cities. There is no evidence of a threshold, and the relation occurs well below the U.S. Environmental Protection Agency standard of 65 μg/m^3 (*27*). The line shows the least-squares fit of a linear relation through the estimated points.

The next results come from the source component models. These models had a smooth function of $PM_{2.5}$ from traffic and linear functions of $PM_{2.5}$ from the other sources in each city. Figure 2 shows the results when we combined the estimated dose–response curves for traffic particles across the six cities. Again, there is no evidence of a threshold, and the association is essentially linear. If anything, the slope is steeper at lower concentrations. To test the robustness of the association with traffic particles to our method of controlling for particles from other sources, we re-estimated the relationship controlling for smooth functions of the estimated particle mass from other sources, rather than the linear terms. This association is shown in Figure 3 and differs little from that shown in Figure 2. We also fit linear regressions through the points shown on Figures 1 and 2. We obtained a slope of 1.5% increase in deaths per 10 μg/m^3 increase in $PM_{2.5}$ and 3% increase in deaths per 10 μg/m^3 increase in particles from traffic, which is the same as the results reported by Laden et al. (*8*). These lines are shown on the figures. This supports the assumption of a linear relationship.

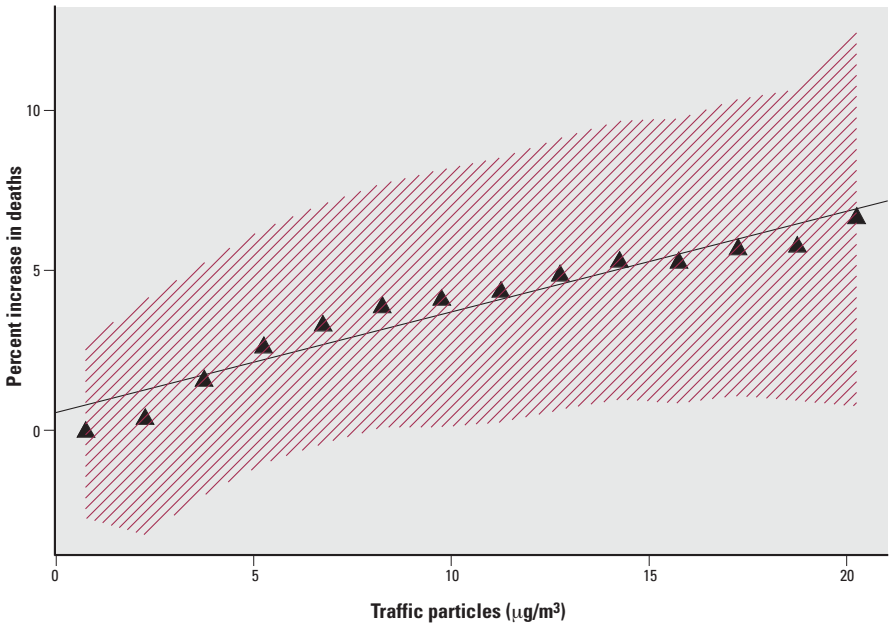

Figure 2. Overall estimated dose–response relation between $PM_{2.5}$ from traffic and daily deaths in six U.S. cities. The estimate is obtained by combining the estimated smoothed curve in each of the cities, after controlling for weather, season, and day of the week and for $PM_{2.5}$ from crustal sources, coal combustion, residual oil, salt, and metal processes as linear terms. The line shown is a least-squares regression line through the estimated points.

Discussion

We have explored the concentration–response relation between $PM_{2.5}$ and daily deaths in six U.S. cities and combined the results to obtain greater stability, while accounting for heterogeneity in response. The population mean curve shows no evidence of a threshold down to the lowest levels of $PM_{2.5}$. In fact, the curve is quite linear over the exposure range from 0 to 35 μg/m^3. This is consistent with previous results using a similar methodology but with PM_{10} (*10*) and black smoke (*11*) as the exposure metric. In addition, a different methodology, using regression splines, was applied by Daniels et al. (*9*) to PM_{10} data in different cities. They combined these spline models across 20 cities. Again, the association appeared to be quite linear without any evidence of a threshold. A spline model had previously been applied by Schwartz (*22*) to the $PM_{2.5}$ data from Boston, with a similar finding. Indeed, the original study of these data by Schwartz, Dockery, and Neas (*5*) found a significant association when limited to days below 30 μg/m^3, with a slightly larger slope. The consistency of the results on two continents, and using different techniques, suggests that this finding is robust. The concentration–response curve seen here for $PM_{2.5}$ is steeper than that previously reported (per μg/m^3) for PM_{10} (*10*). This is consistent with the previous report from this study (*5*) that coarse mass (the difference between PM_{10} and $PM_{2.5}$) is not associated with daily deaths. We note that Schwartz and Zanobetti (*10*) demonstrated in simulation studies that measurement error was not likely to distort the shape of the association. Similarly, recent studies of "harvesting" have shown that effect sizes increase rather than decrease when longer lags are taken into account; for example, high

days producing harvesting that mutes the effect on the next high day is unlikely to have distorted the shape of the association.

These results are also biologically plausible. Schwartz (*28*) pointed out that if thresholds exist in individuals, but there is a distribution of those thresholds among individuals, and if multiple genetic and predisposing illnesses each contributed to the distribution of those thresholds, then by the central limit theorem, the distribution of thresholds should approach a normal distribution. Hence, the population concentration–response curve should approach a cumulative normal curve. But the low-dose end of the cumulative normal curve is linear. To see this, consider that typical death rates in U.S. cities are 8/1,000 per year, or 2×10^{-6} per day. The normal range of variation in daily deaths in U.S. cities is a factor of two or less. Hence, the normal range of daily death probabilities in response to all risk factors is from 1 to 3×10^{-6}. Figure 4 shows the cumulative normal curve in that range of probabilities, which is quite linear. Because we are clearly in the low-dose regime, in the sense that the exposures to particles are well below the threshold for mortality for most people, this linearity is exactly what would be expected.

Figure 1 also indicates that the association reported here has public health significance. The difference between mean $PM_{2.5}$ concentrations of 10 μg/m^3 and 20 μg/m^3, which is a difference found between U.S. cities, is associated with about a 1.5% increase in deaths. In a metropolitan area of a million inhabitants, this would amount to about 130 additional early deaths per year, and in the country as a whole, these results indicate that a reduction of 10 μg/m^3 would be expected to result in about 36,000 fewer early deaths per year. Although this study does not indicate the extent to which these deaths are brought forward, other studies of the harvesting issue (*29–32*) suggest that they are considerable.

The association of daily deaths with traffic particles also has no threshold and is somewhat steeper than the association with all $PM_{2.5}$. This is consistent with the results of Laden et al. (*8*), except that they used linear terms instead of smooth functions. This study confirms that this association extends to low levels. This result has considerable public policy relevance. Recently, automotive companies have proposed using diesel engines to achieve higher fuel economy in the future. However, diesel engines produce substantially greater emissions of particles and particle precursors such as NO_x. The present results indicate that such an expansion of diesel engine use in the United States before diesel engines can meet the same particle emission levels as gasoline engines may result in important public health problems. A 1 μg/m^3 increase in the concentration of traffic particles in the United States, for example, could be associated with about 7,000 additional early deaths per year in the United States.

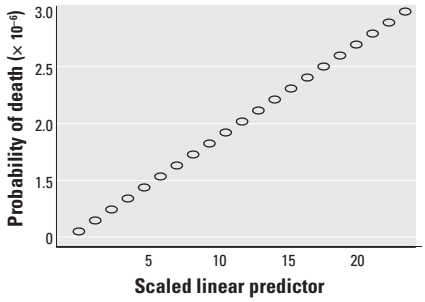

Figure 4. Cumulative normal curve versus a standardized predictor (the sum of the effects of all risk factors) over the range of exposures that correspond to daily death rates of between 1 and 3 per million, which is the observed range of variation in U.S. cities. It is quite linear in the predictor.

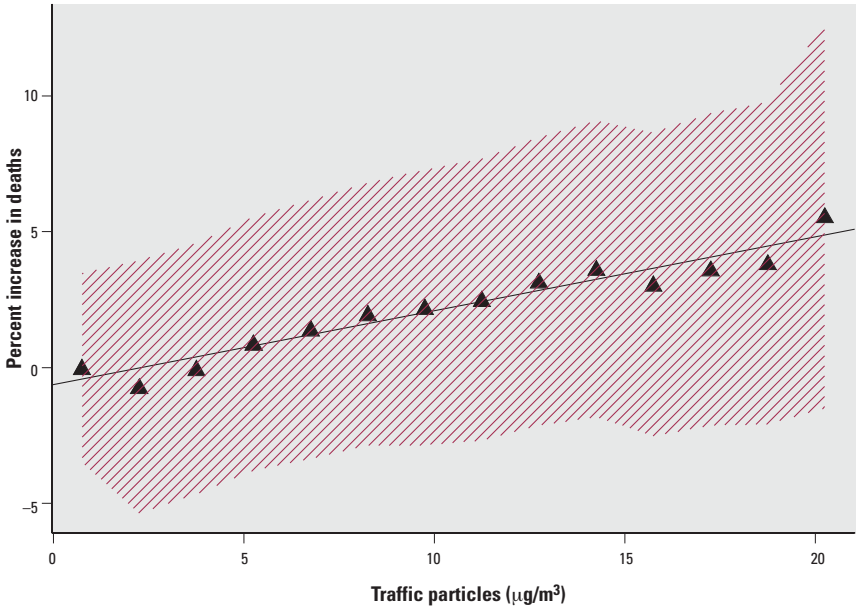

Figure 3. Overall estimated dose–response relation between $PM_{2.5}$ from traffic and daily deaths in six U.S. cities. The estimate is obtained by combining the estimated smoothed curve in each of the cities, after controlling for weather, season, and day of the week. Instead of linear terms for particles from other sources, in this analysis we controlled for smoothed terms for $PM_{2.5}$ from crustal sources, coal combustion, residual oil, and salt. The line shown is a least-squares regression line through the estimated points.

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A Section 508–conformant HTML version of this article is available at http://dx.doi.org/10.1289/ehp.1409111.

Low-Concentration $PM_{2.5}$ and Mortality: Estimating Acute and Chronic Effects in a Population-Based Study

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Background: Both short- and long-term exposures to fine particulate matter (≤ 2.5 μm; $PM_{2.5}$) are associated with mortality. However, whether the associations exist at levels below the new U.S. Environmental Protection Agency (EPA) standards (12 μg/m^3 of annual average $PM_{2.5}$, 35 μg/m^3 daily) is unclear. In addition, it is not clear whether results from previous time series studies (fit in larger cities) and cohort studies (fit in convenience samples) are generalizable.

Objectives: We estimated the effects of low-concentration $PM_{2.5}$ on mortality.

Methods: High resolution (1 km × 1 km) daily $PM_{2.5}$ predictions, derived from satellite aerosol optical depth retrievals, were used. Poisson regressions were applied to a Medicare population (≥ 65 years of age) in New England to simultaneously estimate the acute and chronic effects of exposure to $PM_{2.5}$, with mutual adjustment for short- and long-term exposure, as well as for area-based confounders. Models were also restricted to annual concentrations < 10 μg/m^3 or daily concentrations < 30 μg/m^3.

Results: $PM_{2.5}$ was associated with increased mortality. In the study cohort, 2.14% (95% CI: 1.38, 2.89%) and 7.52% (95% CI: 1.95, 13.40%) increases were estimated for each 10-μg/m^3 increase in short- (2 day) and long-term (1 year) exposure, respectively. The associations held for analyses restricted to low-concentration $PM_{2.5}$ exposure, and the corresponding estimates were 2.14% (95% CI: 1.34, 2.95%) and 9.28% (95% CI: 0.76, 18.52%). Penalized spline models of long-term exposure indicated a larger effect for mortality in association with exposures ≥ 6 μg/m^3 versus those < 6 μg/m^3. In contrast, the association between short-term exposure and mortality appeared to be linear across the entire exposure distribution.

Conclusions: Using a mutually adjusted model, we estimated significant acute and chronic effects of $PM_{2.5}$ exposure below the current U.S. EPA standards. These findings suggest that improving air quality with even lower $PM_{2.5}$ than currently allowed by U.S. EPA standards may benefit public health.

Citation: Shi L, Zanobetti A, Kloog I, Coull BA, Koutrakis P, Melly SJ, Schwartz JD. 2016. Low-concentration $PM_{2.5}$ and mortality: estimating acute and chronic effects in a population-based study. Environ Health Perspect 124:46–52; http://dx.doi.org/10.1289/ehp.1409111

Introduction

Many studies have found associations between fine particulate matter [PM with aerodynamic diameter ≤ 2.5 μm ($PM_{2.5}$)] and increased mortality (Dockery et al. 1993; Franklin et al. 2007; Pope et al. 2002; Schwartz 1994; Zanobetti and Schwartz 2009). Biological evidence has been established for plausible mechanisms between $PM_{2.5}$ and mortality, such as increased risk of ventricular arrhythmia and thrombotic processes, increased system inflammation and oxidative stress, increased blood pressure, decreased plaque stability, and reduced lung function, among others (Brook et al. 2009; Gauderman et al. 2004; Gurgueira et al. 2002; Suwa et al. 2002; Yue et al. 2007). Based on evidence from epidemiological and toxicological studies (Chen and Nadziejko 2005; Furuyama et al. 2006; Ohtoshi et al. 1998), National Ambient Air Quality Standards (NAAQS) were implemented for fine particulate matter. For example, the U.S. Environmental Protection Agency (EPA) revised the fine particle NAAQS in 1997, 2006, and 2012 in order to protect public health (U.S. EPA 1997, 2006, 2013). Further changes in the standards require additional studies to elucidate whether health effects occur at levels below the current annual and daily U.S. EPA NAAQS of 12 and 35 μg/m^3, respectively. The Clean Air Act Amendments of 1990 require the U.S. EPA to review national air quality standards every 5 years to determine whether they should be retained or revised; thus, whether health effects can be observed below the current standards is of great interest and importance.

Previous studies have generally focused on either long-term (Hart et al. 2011; Jerrett et al. 2005; Puett et al. 2009; Schwartz 2000) or short-term (Dominici et al. 2006; Katsouyanni et al. 1997; Samoli et al. 2008; Schwartz and Dockery 1992) exposures across the entire range of $PM_{2.5}$ concentrations. In the case of time series analyses of short-term exposures, the need to ensure the relevance of the monitoring data as well as the need to have a study population of a size for sufficent power has limited analyses to large cities; hence, exurbs, small cities, and rural areas are not generally represented in the literature, which may compromise the generalizability of the results. In addition, there is spatial variability in $PM_{2.5}$ concentrations within cities that time series studies generally do not take into account, which can introduce exposure measurement error (Laden et al. 2006; Lepeule et al. 2012).

Chronic effects studies began using comparisons across cities of mortality experiences of cohorts living in various communities and the monitored air pollutant concentrations in those communities (Dockery et al. 1993; Pope et al. 1995). Again, these studies suffered from exposure error due to failure to capture within-city spatial variability in exposure. Because the geographic exposure gradient is the exposure contrast in these studies, the failure to capture within-city contrasts leads to classical measurement error with expected downward bias. Studies with, for example, land use regression estimates of exposure have generally reported larger effect sizes (Miller et al. 2007; Puett et al. 2009). Previous cohort studies have not controlled for the acute effects of particles when estimating chronic effects, raising the question of whether there are independent chronic effects that represent more than the cumulative effects of acute responses.

In general, existing study cohorts are not representative of the overall population. For example, the American Cancer Society (ACS) cohort has a higher level of education than the U.S. population as a whole (Stellman

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We thank P. Liu and Y. Wang for fruitful discussions.

This study was funded by the National Institute of Environmental Health Sciences/National Institutes of Health grant ES000002. This publication was also made possible by U.S. Environmental Protection Agency (EPA) grant RD-83479801.

The contents of this publication are solely the responsibility of the grantee and do not necessarily represent the official views of the U.S. EPA. Further, the U.S. EPA does not endorse the purchase of any commercial products or services mentioned in the publication.

The authors declare they have no actual or potential competing financial interests.

Received: 22 August 2014; Accepted: 13 May 2015; Advance Publication: 3 June 2015; Final Publication: 1 January 2016.

and Garfinkel 1986). Hence, few population-based cohort studies have been conducted until recently (Kloog et al. 2013).

Several time series studies examined the concentration–response relationship between $PM_{2.5}$ and mortality below concentrations of 100 μg/m^3; these studies generally reported a linear concentration–response relationship (Samoli et al. 2008; Schwartz and Zanobetti 2000). However, there have been few studies focusing on exposures below the current daily U.S. EPA standard of 35 μg/m^3.

Many studies have examined the shape of the concentration–response curve for long-term exposure versus short-term exposure, but in general, they have not covered population-based cohorts, or have only included very low exposures (Schwartz et al. 2008; Crouse et al. 2012).

We recently presented a new hybrid method of assessing temporally and spatially resolved $PM_{2.5}$ exposure for epidemiological studies by combining 1 km × 1 km resolution satellite-retrieved aerosol optical depth (AOD) measurements with traditional land use terms, meteorological variables, and their interactions (Kloog et al. 2014a). This approach allows for predicting daily $PM_{2.5}$ concentrations at a 1 km × 1 km spatial resolution throughout the New England area of the northeastern United States. We also validated our model's performance in rural areas: 10-fold cross-validation (CV) of our model in rural areas (using the IMPROVE stations) resulted in a CV R^2 of 0.92. Further details have been published (Kloog et al. 2014a).

The present study aimed to simultaneously estimate acute and chronic health effects of $PM_{2.5}$ in a population-based Medicare cohort (≥ 65 years of age) encompassing the New England region. We used high-spatial-resolution exposure estimates based on satellite measurements that are available across the region and not just in limited locations. To make this study relevant to future assessments of current U.S. EPA standards, we repeated the analysis after restricting the data to long-term exposures (365-day moving average) < 10 μg/m^3 and repeated the time series analysis of short-term exposures after restricting the data to 2-day average exposures < 30 μg/m^3.

Methods

Study domain. The spatial domain of our study included the New England area, comprising the states of Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, and Vermont (Figure 1A).

Exposure data. A 3-stage statistical modeling approach for predicting daily $PM_{2.5}$ was previously reported incorporating AOD and land use data for the New England region (Kloog et al. 2011). Previous studies have shown that using actual physical measurements in our prediction models improved predictive accuracy over that of comparable land use or spatial smoothing models (Kloog et al. 2011). With AOD retrieved by the multi-angle implementation of atmospheric correction (MAIAC) algorithm, a similar approach was applied for estimating daily $PM_{2.5}$ exposures in New England at a spatial resolution of 1 km × 1 km (Kloog et al. 2014a). In this study, the same $PM_{2.5}$ exposure predictions were employed.

Briefly, we calibrated the AOD–$PM_{2.5}$ relationship on each day of the study period (2003–2008) using data from grid cells with both ground $PM_{2.5}$ monitors and AOD measurements (stage 1), and we used inverse probability weighting to address selection bias due to nonrandom missingness patterns in the AOD measurements. We then used the AOD–$PM_{2.5}$ relationship to predict $PM_{2.5}$ concentrations for grid cells that lacked monitors but had available AOD measurement data (stage 2). Finally, we used a generalized additive mixed model (GAMM) with spatial smoothing and a random intercept for each 1 km × 1 km grid cell to impute data for grid cells/days for which AOD measurements were not available (stage 3). The performance of the estimated $PM_{2.5}$ was validated by 10-fold cross-validation. High out-of-sample R^2 (R^2 = 0.89, year-to-year variation 0.88–0.90 for the years 2003–2008) was found for days with available AOD data. Excellent performance held even in cells/days with no available AOD (R^2 = 0.89, year-to-year variation 0.87–0.91 for the years 2003–2008). The 1-km model had better spatial (0.87)

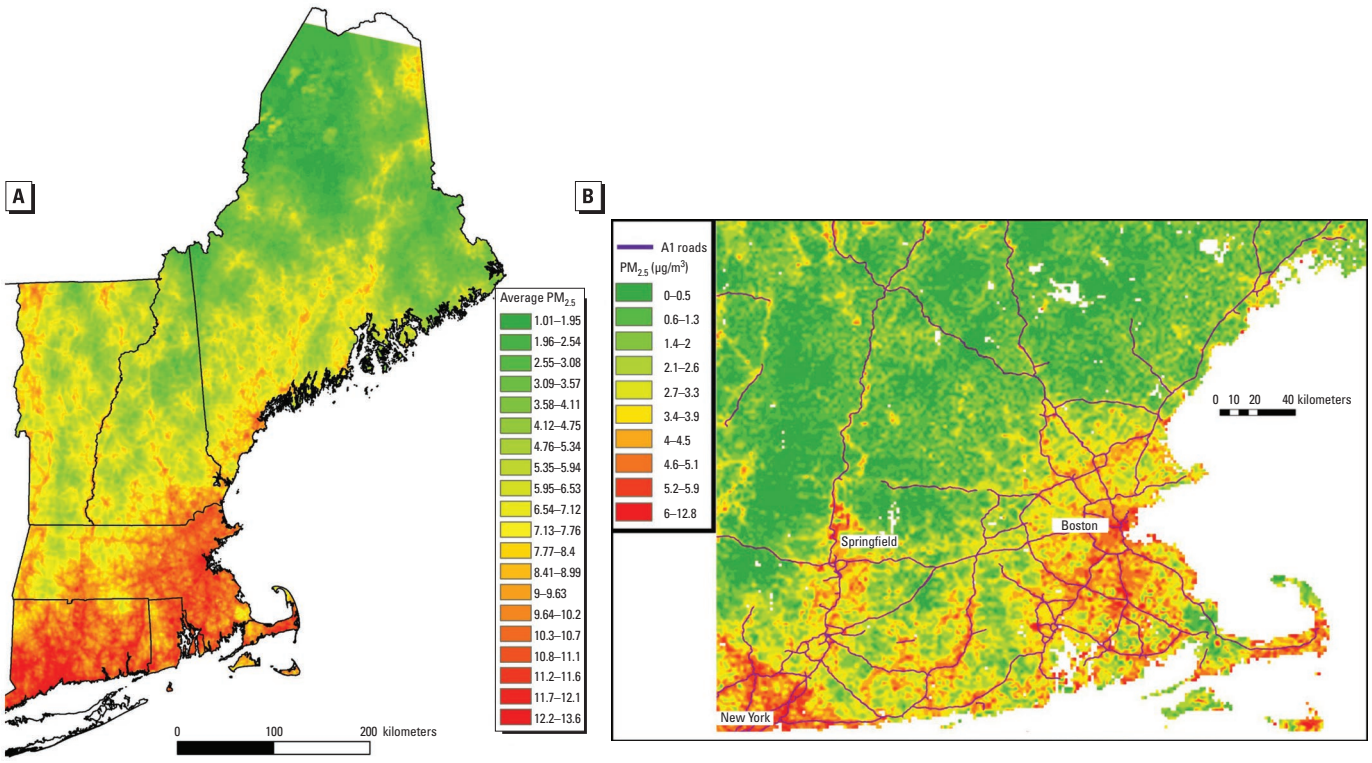

Figure 1. (*A*) Mean $PM_{2.5}$ concentrations in 2004 at a high resolution (1 km × 1 km) across New England predicted by the AOD models. (*B*) Predicted $PM_{2.5}$ concentrations at a 1 km × 1 km grid for 15 November 2003.

and temporal (0.87) out-of-sample R^2 than the previous 10-km model (0.78 and 0.84, respectively). Details of the $PM_{2.5}$ prediction models are in Kloog et al. (2014a).

Figure 1A shows an example of mean $PM_{2.5}$ concentrations in 2004 at a 1 km × 1 km spatial resolution across New England. By averaging the estimated daily exposures at each location, we generated long-term exposures.

Figure 1B (a subset of the study area) shows that spatial variability existed even for daily data and was not identical to the long-term pattern shown in Figure 1A. That is, there was space–time variation in the $PM_{2.5}$ exposure captured in this analysis, but not in previous time-series analyses.

Because the deaths were coded at the ZIP code level, both long- and short-term predictions were matched to ZIP codes by using ArcGIS (ESRI, Redlands, CA) and SAS (SAS Institute Inc., Cary, NC) to link the ZIP code centroid to the nearest $PM_{2.5}$ grid.

Traditionally, studies of acute air pollution effects have controlled for temperature using values taken from the nearest airport. This approach is not feasible for the entire region because many residences are distant from airports. In addition, there is spatiotemporal variation in temperature. We have applied a similar 3-stage statistical modeling approach to estimate daily ambient temperature at 1 km × 1 km resolution in New England using satellite-derived surface temperature (Kloog et al. 2014b). To our knowledge, such fine control for temperature has not previously been used in air pollution epidemiology.

Mortality data. Individual mortality records were obtained from the U.S. Medicare program for all residents ≥ 65 years of age for all available years during 2003–2008 (CMS 2013b). The Medicare cohort was used because of the availability of ZIP code of residence data, whereas National Center for Health Statistics mortality data are only available at the county level. Additionally, previous studies found that elderly people are highly susceptible to the effects of particulate matter (Pope 2000). The Medicare beneficiary denominator file from the Centers for Medicare and Medicaid services (CMS 2013a) lists all beneficiaries enrolled in the Medicare fee-for-service (FFS) program and contains information on beneficiaries' eligibility and enrollment in Medicare and the date of death. The Medicare Provider Analysis and Review (MEDPAR) file includes information on age, sex, race, ZIP code of residence, and one record for each hospital admission (CMS 2013c).

Daily mortality was first aggregated by ZIP code and then matched with the corresponding $PM_{2.5}$ exposure. We summarized the mortality data by ZIP code and day because that was the finest resolution we could obtain for addresses. Because the mortality data sets did not include changes of residence, we assumed that the subjects lived at their current address over the entire study period.

Covariates. We used daily 1-km temperature data estimated from surface temperature measured by satellites (Kloog et al. 2014b). All socioeconomic variables were obtained through the U.S. Census Bureau 2000 Census Summary File 3, which includes social, economic, and housing characteristics (U.S. Census Bureau 2000). ZIP code tabulation area–level socioeconomic variables, including race, education, and median household income, were used. The county-level percentage of people who currently smoke every day, obtained from the CDC Behavioral Risk Factor Surveillance survey for the entire country, was also adjusted (CDC 2013). Dummy variables were used to control for day of the week.

Statistical models. Conventionally, the acute effects of air pollution are estimated by Poisson log-linear models, and the chronic effects of air pollution are estimated by Cox proportional hazard models (Kloog et al. 2013; Laden et al. 2006). Laird and Olivier (1981) noted the equivalence of the likelihood of a proportional hazard model with piecewise constant hazard for each year of follow-up and a Poisson regression with a dummy variable for each year of follow-up. We have taken advantage of this equivalence to generalize from dummy variables for each year to a spline of time to represent the baseline hazard and to aggregate subjects into counts per person time at risk, and we obtained a mixed Poisson regression model (Kloog et al. 2012). This approach allows the rate of death as a function of both long- and short-term exposures to be modeled simultaneously. By doing so, we achieved the equivalence of a separate time series analysis for each ZIP code, greatly reducing the exposure error in that part of the model, while simultaneously conducting a survival analysis on the participants, and we were also able to estimate the independent effects of both exposures.

Most time series studies have reported stronger associations with acute exposures when exposures were defined as the mean $PM_{2.5}$ on the day of death and the previous day (lag01) than when they were defined as the mean $PM_{2.5}$ on the current day only, or for exposures with longer lags (Schwartz et al. 1996; Schwartz 2004). We used the lag01 average for our main analysis but performed a sensitivity analysis on that choice. Long-term exposure was calculated as the 365-day moving average ending on the date of death so that our results were comparable with those of previous studies (Lepeule et al. 2012; Schwartz et al. 2008). Short-term exposure was defined as the difference between the 2-day average and the long-term average, ensuring that acute and chronic effects were independent. We subtracted the long-term average from the short-term average to avoid collinearity issues and to ensure that differences between ZIP codes in $PM_{2.5}$ at a given time did not contribute to the short-term effect estimate. Thus, the short-term effect could not be confounded by variables that differed across ZIP codes.

Specifically, we fit a Poisson survival analysis with a logarithmic link function and a log (population) offset term and modeled the expected daily death counts (μ_{it}) in the ith ZIP code on the tth day as follows:

$$\log(\mu_{it}) = \lambda_i + \beta_1 PM_{it} + \beta_2 \Delta PM_{it} + \lambda(t) + \text{temporal covariates} + \text{spatial covariates} + \text{offset}, \quad [1]$$

where λ_i is a random intercept for each ZIP code, PM_{it} is the 365-day moving average ending on day t in ZIP code i, ΔPM_{it} is the deviation of the 2-day average from its long-term average (PM_{it}) in ZIP code i, $\lambda(t)$ is a smooth function of time, temporal covariates are temperature and day of the week, and spatial covariates are socioeconomic factors defined at the ZIP code level (percent of people without high school education, percent of white people, median household income) and smoking data at the county level. Additionally, a quasi-Poisson model was used to control for possible overdispersion (Ver Hoef and Boveng 2007).

We estimated $\lambda(t)$ with a natural cubic spline with 5 degrees of freedom (df) per year to control for time and season trends. The specific temporal and spatial covariates that we used were a natural cubic spline for temperature with 3 df in total; a categorical variable for day of the week; linear variables for percent of people without high school education, percent of white people, median household income, and percent of people who currently smoke every day.

The number of deaths per ZIP code area over the study period (2003–2008) averaged 319 with a standard deviation of 430. Because the outcome was counts, we could not adjust for age and sex as in a Cox model. Instead, we adjusted for variables that varied by ZIP code. The analyses were repeated without mutual adjustment for short- and long-term $PM_{2.5}$.

We modeled the association between all-cause mortality and $PM_{2.5}$ at low doses in which the person-time at risk in each year of follow-up in each ZIP code was used as the offset. We also conducted effect modification by population size by choosing the median (4,628) of the ZIP code–level total population as the cutoff between urban and rural areas.

Estimating the effects of low-level $PM_{2.5}$. For full cohort analyses with 10,938,852 person-years of follow-up, all observed deaths were used. To estimate effects at low

levels of exposure, we performed restricted analyses: we conducted one analysis restricted to annual exposures < 10 μg/m^3, below the current annual $PM_{2.5}$ NAAQS of 12 μg/m^3, and another restricted to observations with short-term exposure < 30 μg/m^3, below the current daily $PM_{2.5}$ NAAQS of 35 μg/m^3. After these exclusions, the chronic analyses were restricted to 268,050 deaths out of 551,024 deaths in total, and the acute analyses were restricted to 422,637 deaths.

Assessing the dose–response relationship. For both the acute and chronic analyses, we fit penalized regression splines in the restricted analyses to estimate the shape of the dose–response curve below current U.S. EPA standards. The degrees of freedom of the penalized splines for $PM_{2.5}$ were estimated by generalized cross-validation (GCV).

Results

Table 1 presents a summary of the predicted exposures for both short- and long-term $PM_{2.5}$ exposure across all grid cells in the study area.

Table 2 presents the estimated percent change in all-cause mortality with 95% CIs for a 10-μg/m^3 increase in both short- and long-term $PM_{2.5}$ in the restricted and full cohort. In the restricted population, we found an estimated 9.28% increase in mortality (95% CI: 0.76, 18.52%) for every 10-μg/m^3 increase in long-term $PM_{2.5}$ exposure. A 2.14% increase in mortality (95% CI: 1.34, 2.95%) was observed for every 10-μg/m^3 increase in short-term $PM_{2.5}$ exposure. For long-term exposure, the effect estimates were smaller when higher pollution days were included (7.52%; 95% CI: 1.95, 13.40%), suggesting larger effects between low-concentration long-term $PM_{2.5}$ and mortality.

Without mutual adjustment, lower estimates were found for both acute and chronic effects than for those with mutual adjustment. In full-cohort analyses, a 2.08% (95% CI: 1.32, 2.84%) and a 6.46% (95% CI: 0.93, 12.30%) increase in mortality was found for each 10-μg/m^3 increase in short- and long-term $PM_{2.5}$, respectively. In restricted analyses, the corresponding effect estimates were 2.07% (95% CI: 1.27, 2.89%) and 7.16% (95% CI: –1.23, 16.27%), respectively.

Our results were robust to the choice of lag period for acute exposure. We analyzed different averaging periods (Figure 2): for example, lag0 (day of death exposure) and lag04 (a moving average of day of death exposure and previous 4-day exposure). For the acute effects, we found a significant but smaller association for lag0 $PM_{2.5}$ (1.71%; 95% CI: 1.09, 2.34%) and lag04 $PM_{2.5}$ (1.76%; 95% CI: 0.72, 2.81%) than for lag01 analysis. The lag period used for short-term exposure did not affect estimates of chronic effects. For example, estimated increases in mortality with a 10-μg/m^3 increase in long-term $PM_{2.5}$ were 7.35% (95% CI: 1.79, 13.21%) and 7.25% (95% CI: 1.69, 13.12%) when short-term $PM_{2.5}$ was classified using lag0 or lag04, respectively.

We also examined effect modification by population size. In the full cohort, a significant interaction was found for chronic effects (p < 0.01), with a larger effect of 12.56% (95% CI: 5.71, 19.85%) in urban areas compared with 3.21% (95% CI: –2.92, 9.72%) in rural areas. Such a significant interaction, however, was not observed in the restricted analysis (p = 0.16). Estimates were 14.27% (95% CI: 3.19, 26.53%) and 5.48% (95% CI: –4.21, 16.16%) in urban and rural areas, respectively. For short-term exposure, population size did not modify the acute effects in either the full cohort or the restricted analysis (p = 0.74 and 0.46, respectively).

In our penalized spline model for long-term exposure below the cutoff of 10 μg/m^3 (Figure 3A), we found a nonlinear relationship between long-term $PM_{2.5}$ and mortality. The association was linear with evidence of a smaller effect < 6 μg/m^3. However, a large confidence interval was observed; hence, we could not be confident whether the slope of the dose–response curve changed for long-term exposures < 6 μg/m^3. When examining the shape of the dose–response curve for chronic effects, both a linear term for short-term exposure (the difference) and a penalized spline for long-term average exposure were included in the model, resulting in a penalized spline with a df of 1.71. In contrast, we only included the 2-day average in the penalized spline model of acute effects in order to provide an interpretable dose–response relationship (Figure 3B). The results of this analysis indicated a linear association across the exposure distribution, but we could not be certain about the shape of the slope for acute effects < 3 μg/m^3.

Discussion

When we applied the predicted daily $PM_{2.5}$ with 1-km spatial resolution from our novel hybrid models, we observed that both short- and long-term $PM_{2.5}$ exposure were significantly associated with all-cause mortality among residents of New England ≥ 65 years of age, even when restricted to ZIP codes and times with annual exposures < 10 μg/m^3 or with daily exposure < 30 μg/m^3. Hence, the association of particle exposure with mortality exists for concentrations below the current standards established by the United States, the World Health Organization (WHO) (10 μg/m^3 of annual average $PM_{2.5}$, 25 μg/m^3 daily), and the European Union (EU) (25 μg/m^3 of annual average $PM_{2.5}$) (EU 2013; WHO 2013). Notably, this analysis includes all areas in New England and all Medicare enrollees ≥ 65 years of age in this region, and it provides chronic effect estimates that are independent of acute effects. Based

Table 1. Descriptive statistics for $PM_{2.5}$ exposure and temperature in New England, 2003–2008.

Covariate	Mean	SD	Minimum	Median	Maximum	Range	Q1	Q3	IQR
Lag01 $PM_{2.5}$ (μg/m^3)	8.21	5.10	0.00	7.10	53.98	53.98	4.60	10.65	6.05
1-year $PM_{2.5}$ (μg/m^3)	8.12	2.28	0.08	8.15	20.22	20.14	6.22	10.00	3.78
Temperature (°C)	9.24	6.50	–36.79	9.81	41.51	78.30	4.90	14.39	9.49

Table 2. Percent increase in mortality (95% CI) for a 10-μg/m^3 increase for both short-term and long-term $PM_{2.5}$.

$PM_{2.5}$ exposure	Model	Percent increase	p-Value
With mutual adjustment			
Short-term $PM_{2.5}$	Low daily exposure[a]	2.14 ± 0.81	< 0.001
	Full cohort	2.14 ± 0.75	< 0.001
Long-term $PM_{2.5}$	Low chronic exposure[b]	9.28 ± 8.88	0.032
	Full cohort	7.52 ± 5.73	0.007
Without mutual adjustment			
Short-term $PM_{2.5}$	Low daily exposure[a]	2.07 ± 0.80	< 0.001
	Full cohort	2.08 ± 0.76	< 0.001
Long-term $PM_{2.5}$	Low chronic exposure[b]	7.16 ± 8.75	0.109
	Full cohort	6.46 ± 5.69	0.026

The full cohort analysis had 551,024 deaths.
[a]The analysis was restricted only to person time with daily $PM_{2.5}$ < 30 μg/m^3 (422,637 deaths). [b]The analysis was restricted only to person time with chronic $PM_{2.5}$ < 10 μg/m^3 (268,050 deaths).

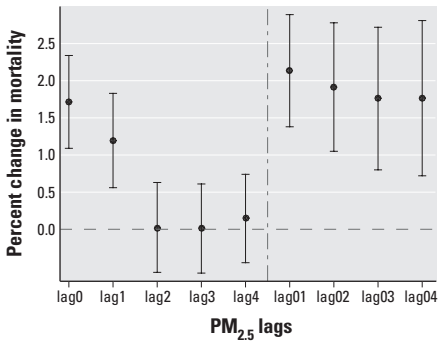

Figure 2. Percent change in mortality per 10-μg/m^3 increase in short-term $PM_{2.5}$ with different lags with mutual adjustment. Error bars indicate the 95% CIs.

on a penalized spline model, the positive dose–response relationship between chronic exposure and mortality appears to be linear for $PM_{2.5}$ concentrations ≥ 6 μg/m^3, with a positive (though smaller and less precise) dose–response slope continuing below this level. This lack of power is likely due to the small exposed population in areas with annual $PM_{2.5}$ < 6 μg/m^3, which were quite rural.

For acute effects, we found a 2.14% (95% CI: 1.38, 2.89%) increase in all-cause mortality per 10-μg/m^3 increment in $PM_{2.5}$ for the full cohort of our study, which is higher than the effect size of most studies using city averages obtained from monitors. For instance, in a U.S. national study by Zanobetti and Schwartz (2009), the effect size was 0.98% (95% CI: 0.75, 1.22%). Similar results were also obtained in a systematic review, where researchers determined that the overall summary estimate was 1.04% (95% CI: 0.52, 1.56%) per 10-μg/m^3 increment in $PM_{2.5}$ (Atkinson et al. 2014). The exposure data used in most previous studies had low spatial resolution (citywide average, not ZIP code), which introduced exposure measurement error and likely resulted in a downward bias in estimates; our results (for the acute effect) are consistent with such a phenomenon. Our restricted study estimated a 2.14% (95% CI: 1.34, 2.95%) increase in all-cause mortality per 10-μg/m^3 increment in $PM_{2.5}$, which was close to the effect size of the full cohort study, possibly because the sample size of the restricted study for acute effects was close to that of the full cohort. Furthermore, the U.S. EPA daily standard (35 μg/m^3) was almost never exceeded in this study. In addition, lower effect estimates for short-term exposure were observed with mutual adjustment for both full cohort and restricted analyses. This finding has important implications for the interpretation of previous studies without such mutual adjustment.

For chronic effects, the effect estimate in our full cohort study was consistent with findings of previous studies with comparable sample sizes (Hoek et al. 2013; Laden et al. 2006; Lepeule et al. 2012). For example, an ACS study comprising 500,000 adults from 51 U.S. cities reported a 6% (95% CI: 2, 11%) increase in all-cause mortality for each 10-μg/m^3 increment in $PM_{2.5}$ (Pope et al. 2002). A study of 13.2 million elderly Medicare recipients across the eastern United States found a 6.8% (95% CI: 4.9, 8.7%) increase in all-cause mortality for each 10-μg/m^3 increment in $PM_{2.5}$ (Zeger et al. 2008). When we restricted our analysis to annual concentrations < 10 μg/m^3, a larger slope of 9.28% (95% CI: 0.76, 18.52%) increase per 10 μg/m^3 was observed. Our findings suggest a larger effect at low concentrations among those ≥ 65 years of age, which may also reflect particle composition. The sources and composition of the particles may differ between low-pollution days and high-pollution days, which are likely more affected by secondary aerosols. Compared with the effect estimate for the full cohort, the effect estimate from the restricted analysis was closer to estimates published in the literature that reported larger effect estimates, such as those reported by the ESCAPE (European Study of Cohorts for Air Pollution Effects) study, the Harvard Six Cities study, and the Women's Health Initiative study (Beelen et al. 2014; Puett et al. 2008). Smaller effect estimates were also observed for chronic effects without mutual adjustment.

To the best of our knowledge, this study is the first of its kind to restrict exposure and to explore the dose–response relationship between $PM_{2.5}$ below the current U.S. EPA standards (12 μg/m^3 of annual average $PM_{2.5}$, 35 μg/m^3 daily) and mortality. Moreover, the use of the Medicare cohort means that we studied the entire population of Medicare enrollees ≥ 65 years of age and not a convenience sample. In addition, temperature was controlled on a 1 km × 1 km fine geographic scale. The acute and chronic effects observed in analyses restricted to low $PM_{2.5}$ exposure were similar to or even higher than those of the full cohort analyses. These results indicate that the adverse health effects of $PM_{2.5}$ are at least retained, if not strengthened, at low levels of exposure. However, the findings from the penalized spline model did not support a strong association at the lowest range of $PM_{2.5}$ concentrations. This finding provides epidemiological evidence for the reevaluation of U.S. EPA guidelines and standards, although more evidence is needed to confirm the association < 6 μg/m^3.

The Poisson survival analysis applied in this study provided a novel method of simultaneously assessing acute and chronic effects. As shown in our analysis, the chronic effect estimate was much larger than the acute effect estimate after controlling for the acute estimate, indicating that there were chronic effects of $PM_{2.5}$, which cannot be solely explained by the short-term exposure.

Another key component of this study is that the application of high spatial (1 km × 1 km) and temporal (daily) resolution of $PM_{2.5}$ concentrations reduced exposure error to a certain extent. The out-of-sample R^2 was higher than that for the predictions with 10 km × 10 km spatial resolution.

A potential limitation is the limited availability of individual-level confounders, such as smoking status, which could bias the health effect estimates. We were able to control for ZIP code–level education, median income, race, and county-level smoking data. However, Brochu et al. (2011) reported that census tract–level socioeconomic indicators were uncorrelated with $PM_{2.5}$ on the subregional and local scale, providing some assurance that confounding by socioeconomic status may not be much of an issue. The results reported by Brochu et al. (2011) suggest that those variables may not confound the association, but the inability to control for them remains an issue. Another limitation is that we did not examine other pollutants such as ozone (O_3) or nitrogen dioxide (NO_2) owing to a lack of data at the same spatial level as that of $PM_{2.5}$.

Conclusions

In conclusion, the acute and chronic effects of low-concentration $PM_{2.5}$ were examined for a Medicare population using a comprehensive exposure data set obtained from a satellite-based prediction model. Our findings show that both short- and long-term exposure to $PM_{2.5}$ were associated with all-cause mortality, even for exposure levels not exceeding the newly revised U.S. EPA standards, suggesting that adverse health effects occur at low levels of fine particles. The policy implication of these findings is that improving the air quality at even lower levels of $PM_{2.5}$ than presently allowed by the U.S. EPA standards can yield health benefits.

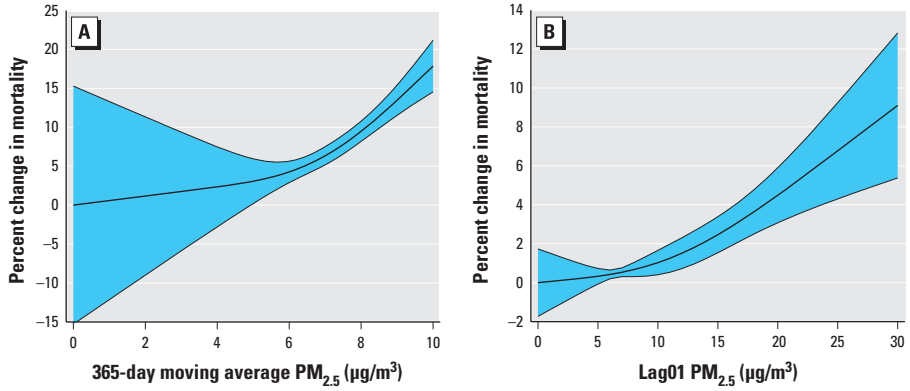

Figure 3. The dose–response relationship between long-term $PM_{2.5}$ and mortality at low doses with mutual adjustment (*A*) and the dose–response relationship between short-term $PM_{2.5}$ and mortality at low doses without mutual adjustment (*B*). Shaded areas indicate the 95% CIs.

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Ambient Air Pollution and Pregnancy Outcomes: A Review of the Literature

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Over the last decade or so, a large number of studies have investigated the possible adverse effects of ambient air pollution on birth outcomes. We reviewed these studies, which were identified by a systematic search of the main scientific databases. Virtually all reviewed studies were population based, with information on exposure to air pollution derived from routine monitoring sources. Overall, there is evidence implicating air pollution in adverse effects on different birth outcomes, but the strength of the evidence differs between outcomes. The evidence is sufficient to infer a causal relationship between particulate air pollution and respiratory deaths in the postneonatal period. For air pollution and birth weight the evidence suggests causality, but further studies are needed to confirm an effect and its size and to clarify the most vulnerable period of pregnancy and the role of different pollutants. For preterm births and intrauterine growth retardation (IUGR) the evidence as yet is insufficient to infer causality, but the available evidence justifies further studies. Molecular epidemiologic studies suggest possible biologic mechanisms for the effect on birth weight, premature birth, and IUGR and support the view that the relation between pollution and these birth outcomes is genuine. For birth defects, the evidence base so far is insufficient to draw conclusions. In terms of exposure to specific pollutants, particulates seem the most important for infant deaths, and the effect on IUGR seems linked to polycyclic aromatic hydrocarbons, but the existing evidence does not allow precise identification of the different pollutants or the timing of exposure that can result in adverse pregnancy outcomes. *Key words:* **air pollution, intrauterine growth retardation, low birth weight, molecular epidemiology, PAHs, particulate matter, PM_{10}, premature birth, reproductive effects, SO_2.** *Environ Health Perspect* **113:375–382 (2005). doi:10.1289/ehp.6362 available via** *http://dx.doi.org/* **[Online 4 January 2005]**

There is extensive evidence that ambient air pollution affects human health (e.g., Brunekreef and Holgate 2002; Künzli et al. 2000; Pope et al. 2002). Most studies have focused on the effects of air pollution on adult mortality and respiratory morbidity (Dockery et al. 1993; Schwartz and Marcus 1990). However, some age groups appear to be more susceptible than others. For example, it has been shown that the effects are larger in the elderly than in the general adult population (Saldiva et al. 1995). Studies on childhood health risks, such as respiratory symptoms or hospital admissions for asthma, suggest that the opposite end of the age spectrum is also more vulnerable to air pollution than is the general population (Dockery and Pope 1994; Heinrich et al. 1999; Schwartz et al. 1994). In addition to these "traditional" end points in children, there is now emerging evidence that air pollution is also associated with elevated risk of adverse pregnancy outcomes (Glinianaia et al. 2004; Maisonet et al. 2004).

The study of birth outcomes is an important emerging field of environmental epidemiology. Birth outcomes are important in their own right because they are important indicators of the health of the newborns and infants. In addition, low birth weight (LBW), intrauterine growth retardation (IUGR), and impaired growth in the first years of life are known to influence the subsequent health status of individuals, including increased mortality and morbidity in childhood and an elevated risk of hypertension, coronary heart disease, and non-insulin-dependent diabetes in adulthood (Barker 1995; Osmond and Baker 2000).

It is increasingly apparent that there is a critical period of development when the timing of exposure and the dose absorption rate can be even more important for the biologic effects than is the overall dose (Axelrod et al. 2001). Fetuses, in particular, are considered to be highly susceptible to a variety of toxicants because of their exposure pattern and physiologic immaturity (Perera et al. 1999; Šrám 1999). Their developing organ systems can be more vulnerable to environmental toxicants during critical windows (sensitive periods of development) because of higher rates of cell proliferation or changing metabolic capabilities (Calabrese 1986). Therefore, prenatal exposure to environmental pollution can result in some adverse reproductive outcomes, similar to the association between maternal active and passive smoking and impaired reproductive outcomes (Misra and Nguyen 1999; Salihu et al. 2004). The specific mechanisms that may account for the link between ambient air pollution and adverse reproductive outcomes are also reviewed in this article.

The objective of this review is to examine the evidence linking adverse birth outcomes with ambient air pollution. For the purpose of this review, birth outcomes have been divided into five groups: *a*) mortality of fetuses and infants, *b*) LBW, *c*) premature (preterm) births, *d*) IUGR, and *e*) birth defects. In this article we review the evidence on each of these separately. For each of the outcomes, we assess the three critical issues in interpreting epidemiologic studies (random error, selection or measurement bias, and confounding); issues related to all reviewed outcomes (e.g., publication bias or biologic plausibility) are considered together at the end of the article. By weighting the evidence, we attempt to draw balanced conclusions about the relations between air pollution and birth outcomes.

Materials and Methods

We searched all publications included in the electronic databases PubMed (from 1966; National Library of Medicine, Bethesda, MD, USA) and the Science Citation Index and Social Science Citation Index of the Institute of Scientific Information, available on the Web of Knowledge (from 1981; Thompson Scientific, Philadelphia, PA, USA). We searched for combinations of either of the key words "air pollution" or "pollution" with any of the following: "infant mortality," "postneonatal mortality," "postneonatal mortality," "birth weight," "birthweight," "intrauterine growth retardation," "IUGR," "premature birth," "prematurity," "fetal growth," and "foetal growth." We also searched the reference lists of identified papers for additional publications. We excluded abstracts of conference presentations because they did not contain sufficient information (but relevant conference abstracts that were subsequently published as full papers were included).

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We thank T. Damstra (World Health Organization, Research Triangle Park, NC, USA) for her critical comments.

This work was supported by the grants from the European Commission (QLRT-2001-02198, ChildrenGenonetwork) and the Ministry of Environment of the Czech Republic (VaV 740/5/03).

The authors declare they have no competing financial interests.

Received 31 March 2003; accepted 4 January 2005.

Results

Air pollution and childhood mortality. The possible impact of air pollution on children's health was first connected to early child mortality. One of the earliest reports was based on an ecologic study of counties of England and Wales in 1958–1964, with air pollution estimated from indices of domestic and industrial pollution (Collins et al. 1971). The study found significant correlations between air pollution and infant mortality, and infant respiratory mortality in particular. The Nashville Air Pollution Study, conducted in the 1950s, indicated that dust fall, a measure of air pollution estimated for each census tract, was related to neonatal deaths with signs of prematurity, but the results were inconclusive (Sprague and Hagstrom 1969). Another early signal that air pollution may be associated with deaths in infancy came from the extensive analyses of air pollution and mortality in 117 U.S. metropolitan areas in the 1960s (Lave and Seskin 1977). Particulates and, to a lesser degree, sulfate concentrations were positively associated with infant mortality; a 10% increase in pollution was associated with a 1% increase in infant mortality.

Almost two decades passed before a new generation of studies addressed this question in more detail. These newer studies, summarized in Table 1, confirmed the early results. A small ecologic study in the Rio de Janeiro, Brazil, metropolitan area reported a positive association between annual levels of particulates and infant mortality from pneumonia (Penna and Duchiade 1991).

Bobak and Leon (1992) studied infant mortality in an ecologic study in the Czech Republic. The study found an association between sulfur dioxide and total suspended particles (TSP), and infant mortality, after controlling for a number of potential confounding variables (at the ecologic level). The effects were specific to respiratory mortality in the postneonatal period. These results were later confirmed in a nationwide case–control study based on the Czech national death and birth registers; this design allowed controlling for social and biologic covariates at the individual level (Bobak and Leon 1999a). The study found a strong effect of SO_2 and TSP on postneonatal mortality from respiratory causes: the relative risks (RRs) per 50 μg/m^3 increase in pollutant concentration were 1.95 [95% confidence interval (CI), 1.09–3.50] for SO_2 and 1.74 (95% CI, 1.01–2.98) for TSP.

Woodruff et al. (1997) analyzed the association between early postneonatal mortality and PM_{10} (particulate matter < 10 μm) levels in about 4 million babies born from 1989 through 1991 in the United States. Infants were categorized as having high, medium, or low exposures based on tertiles of PM_{10}. After adjustment for other covariates, for total postneonatal mortality for the high-exposure (> 40 μg/m^3) versus low-exposure (< 28 μg/m^3) groups was 1.10 (95% CI, 1.04–1.16). In infants of normal birth weight, high PM_{10} exposure was associated with respiratory deaths (RR, 1.40; 95% CI, 1.05–1.85) and sudden infant death syndrome (RR, 1.26; 95% CI, 1.14–1.39). The results were similar in term and LBW infants.

Pereira et al. (1998) investigated the associations between daily counts of intrauterine mortality in the city of São Paulo, Brazil, in 1991–1992 and daily measurements of several pollutants: nitrogen dioxide, SO_2, carbon monoxide, ozone, and PM_{10}. The association was strongest for NO_2 (coefficient R = 0.0013/μg/m^3; $p < 0.01$). A significant association was also observed with exposure combining the pollutants NO_2, SO_2, and CO together ($p < 0.01$).

Loomis et al. (1999) conducted a time-series study of infant mortality in the southwestern part of Mexico City in 1993–1995. Exposure included NO_2, SO_2, O_3, and particulate matter < 2.5 μm ($PM_{2.5}$). A 10 μg/m^3 increase in the mean level of fine particles during the preceding 3 days was associated with a 6.9% (95% CI, 2.5–11.3%) excess increase in infant death.

Dolk et al. (2000) examined perinatal and infant outcomes in populations residing near 22 coke works in Great Britain. Data on specific pollutants were not provided; the exposure was based on the proximity to pollution sources. The ratios of observed to expected cases for residence in proximity of the coke works were 0.94 (95% CI, 0.78–1.12) for stillbirth, 0.95 (95% CI, 0.83–1.09) for infant mortality, 0.86 (95% CI, 0.72–1.03) for neonatal mortality, 1.10 (95% CI, 0.90–1.33) for postneonatal mortality, 0.79 (95% CI, 0.30–1.46) for respiratory postneonatal mortality, and 1.07 (95% CI, 0.77–1.43) for sudden infant death syndrome in the postneonatal period. This study, however, had a limited statistical power owing to the relatively small size of the study.

A time-series analysis of daily deaths in Seoul, South Korea, found a relatively specific association between PM_{10} and total and respiratory mortality in the postneonatal period; the RRs per 10 μg/m^3 were 1.03 (1.02–1.04) and 1.18 (1.14–1.21), respectively (Ha et al. 2003).

The consistency of these studies, conducted in a range of different populations and using both spatial and time-series study designs, is remarkable. The three largest studies produced very similar estimates of RR (Bobak and Leon 1992, 1999a; Woodruff et al. 1997). Perhaps the only alternative explanation that may affect the interpretation

Table 1. Air pollution and child mortality.

Mortality	Pollutant	Results	Reference
Postneonatal respiratory mortality	TSP	AOR = 2.41 (95% CI, 1.10–5.28) comparing highest vs. lowest quintile AOR = 3.91 (95% CI, 0.90–3.50) for 50 μg/m^3 increase	Bobak and Leon 1992
Postneonatal respiratory mortality	TSP	AOR = 1.95 (95% CI, 1.90–3.50) for 50 μg/m^3 increase	Bobak and Leon 1999a
	SO_2	AOR = 1.74 (95% CI, 1.01–2.98) for 50 μg/m^3 increase	
	NO_x	AOR = 1.66 (95% CI, 0.98–2.81) for 50 μg/m^3 increase	
Postneonatal infant mortality	PM_{10}	AOR = 1.10 (95% CI, 1.04–1.16) comparing high vs. low exposure	Woodruff et al. 1997
Respiratory death groups	PM_{10}	AOR = 1.40 (95% CI, 1.05–1.85) comparing high vs. low exposure with normal birth weight	
Sudden infant death	PM_{10}	AOR = 1.26 (95% CI, 1.14–1.39) comparing high vs. low exposure groups	
Intrauterine mortality	NO_2	Strong association (coefficient = 0.0013 μg/m^3, $p < 0.01$)	Pereira et al. 1998
	SO_2	NE	
	CO	NE Significant association using pollution index $NO_x + SO_2 + CO$	
	O_3	NE	
	PM_{10}	NE	
Infant mortality	NO_2	NE	Loomis et al. 1999
	SO_2	NE	
	CO	NE	
	O_3	NE	
	PM_{10}	6.9% excess (95% CI, 2.5–11.3%) for 10 μg/m^3 increase	
Perinatal and infant mortality		NE between residence near coke works	Dolk et al. 2000

Abbreviations: AOR, adjusted odds ratio; NE, no effect; PM_{10}, particulate matter < 10 μm; TSP, total suspended particulate.

of these studies is confounding by maternal smoking. It is likely that maternal smoking is associated with children's risk of respiratory death, and none of the studies was able to control for maternal smoking on the individual level. However, at least three observations argue against this possibility. First, all recent studies controlled for socioeconomic factors and other potential confounders. Because smoking in industrialized countries is strongly socially patterned, adjustment for socioeconomic factors should at least partly adjust for smoking. This would be reflected by adjusted estimates being substantially smaller than unadjusted ones. However, in most instances the differences between the crude and adjusted effect estimates were minimal. This does not suggest a presence of residual confounding.

Second, the results of spatial and time-series studies were similar. It is highly unlikely that the social composition or maternal smoking in the studied populations would change substantially over the relatively short periods covered by the time-series studies. In our view, the time-series design practically precludes a presence of confounding by socioeconomic factors or maternal smoking.

Finally, the studies were conducted in very different populations, ranging from China to the United States and from Brazil to the Czech Republic; it is unlikely that the distribution of socioeconomic disadvantage or maternal smoking with respect to air pollution would be similar enough in these different countries to produce the same pattern of results. We therefore conclude that the evidence is sufficient to infer causal relationship between particulate air pollution and respiratory deaths in the postneonatal period.

Air pollution and birth weight. The potential effects of air pollutants on birth weight were first examined in a small case–control study by Alderman et al. (1987); the study did not find any relationship between neighborhood ambient levels of CO during the third trimester of pregnancy and LBW. Over the last decade, this question has been investigated in a number of studies (summarized in Table 2).

Wang et al. (1997) examined the effects of SO_2 and TSP on birth weight in a time-series study in four relatively highly polluted residential areas of Beijing, China. A spectrum of potential confounding factors was adjusted for in multivariate analysis. A graded dose–effect relationship was found between maternal exposure to SO_2 and TSP during the third trimester and infant birth weight. The mean birth weight was reduced by 7.3 and 6.9 g for each 100-μg/m^3 increase in SO_2 and TSP, respectively. The RRs of LBW associated with a 100-μg/m^3 increase in SO_2 and TSP were 1.11 (95% CI, 1.06–1.16) and 1.10 (95% CI, 1.05–1.14), respectively. The authors speculated that SO_2 and particles, or some complex mixtures associated with these pollutants, during late gestation contributed to the LBW risk in the studied population.

Bobak and Leon (1999b) conducted an ecologic study of LBW and levels of nitrous oxides (NO_x), SO_2, and TSP in 45 districts of the Czech Republic in 1986–1988. After controlling for socioeconomic factors, the RRs of LBW associated with an increase of 50 μg/m^3 in the annual mean concentrations were 1.04 (95% CI, 0.96–1.12) for TSP, 1.10 (95% CI, 1.02–1.17) for SO_2, and 1.07 (95% CI, 0.98–1.16) for NO_x. When all pollutants were included in one model, only SO_2 remained related to LBW [odds ratio (OR), 1.10; 95% CI, 1.01–1.20].

In a subsequent study, Bobak (2000) analyzed individual-level data on all single live births in the Czech Republic that occurred in 1991 in the 67 districts where at least one pollutant (NO_x, SO_2, or TSP) was monitored. The risk of LBW was analyzed separately for each trimester of pregnancy. The association between LBW and pollution was strongest for pollutant levels during the first trimester of pregnancy. The RRs of LBW per 50 μg/m^3 increase in the mean concentration of SO_2 and TSP during the first trimester were 1.20 (95% CI, 1.11–1.30) and 1.15 (95% CI, 1.07–1.24), respectively.

In a population-based study in Southern California, Ritz and Yu (1999) examined the influence of pollution levels during the third trimester on LBW risk in a cohort of 126,000 term births. The exposure to O_3, NO_2, and PM_{10} in the last trimester was estimated from continuous monitoring data. After adjustment for potential confounders, the risk of LBW was associated with maternal exposure to > 5.5 ppm CO during the third trimester (RR, 1.22; 95% CI, 1.03–1.44). The association

Table 2. Air pollution and birth weight.

Outcome	Pollutant	Results	Reference
LBW	SO_2	AOR = 1.21 (95% CI, 1.06–1.16) for 100 μg/m^3 increase	Wang et al. 1997
	TSP	AOR = 1.10 (95% CI, 1.05–1.14) for 100 μg/m^3 increase	
LBW	TSP	OR = 1.04 (95% CI, 0.96–1.12) for 50 μg/m^3 increase	Bobak and Leon 1999b
	SO_2	OR = 1.10 (95% CI, 1.02–1.17) for 50 μg/m^3 increase	
	NO_x	OR = 1.07 (95% CI, 0.98–1.16) for 50 μg/m^3 increase	
LBW	NO_x	NE	Bobak 2000
	SO_2	AOR = 1.20 (95% CI, 1.11–1.30) for 50 μg/m^3 increase in the first trimester	
	TSP	AOR = 1.15 (95% CI, 1.07–1.24) for 50 μg/m^3 increase in the first trimester	
LBW	O_3	NE	Ritz and Yu 1999
	NO_2	NE	
	PM_{10}	NE	
	CO	OR = 1.22 (95% CI, 1.03–1.44) for CO > 5.5 ppm in the first trimester	
VLBW	TSP + SO_2	AOR = 2.88 (95% CI, 1.16–7.13) comparing highest vs. lowest exposure groups (56.7 vs .9.9 μg/m^3)	Rogers et al. 2000
LBW	CO	AOR = 1.43 (95% CI, 1.18–1.74) for 1 ppm increase in first trimester	Maisonet et al. 2001
		AOR = 1.75 (95% CI, 1.50–2.04) for 1 ppm increase in first trimester in African Americans	
	SO_2	AOR = 1.18/1.20 (95% CI, 1.02–1.36) ppm increase in all trimesters in whites	
LBW	SO_2 + NO_2 + PM_{10}	AOR = 1.77 (95% CI, 1.00–3.12) comparing petrochemical and control municipalities	Lin et al. 2001b
LBW	CO	AOR = 1.08 (95% CI, 1.04–1.12) in the first trimester	Ha et al. 2001
	NO_2	AOR = 1.07 (95% CI, 1.03–1.11) in the first trimester	
	SO_2	AOR = 1.06 (95% CI, 1.02–1.10) in the first trimester	
	TSP	AOR = 1.04 (95% CI, 1.00–1.08) in the first trimester	
LBW	POM	OR = 1.31 (95% CI, 1.21–1.43) comparing highest vs. lowest exposure groups	Vassilev et al. 2001b

Abbreviations: AOR, adjusted odds ratio; NE, no effect; VLBW, very low birth weight (< 1,500 g).

between LBW risk and pollution exposure during earlier gestational stages was not significant.

In a population-based case–control study in Georgia (USA), Rogers et al. (2000) analyzed the combined effect on very low birth weight (VLBW) (< 1,500 g) of SO_2 and TSP levels, using annual exposure estimates. The risk of VLBW was increased in babies of mothers who were exposed to concentrations of the combined pollutants above the 95th percentile of the exposure distribution (56.8 μg/m^3); the RR was 2.88 (95% CI, 1.16–7.13).

Maisonet et al. (2001) examined the association between term LBW and ambient levels of SO_2, PM_{10}, and CO in six large cities in the northeastern United States. Their results suggested that the effects of ambient CO and SO_2 on the risk of term LBW may differ by ethnic group. In Caucasians (n ~ 36,000), the risk of LBW associated with a 10-ppm increase in SO_2 was 1.18 (95% CI, 1.12–1.23) in the first trimester, 1.18 (95% CI, 1.02–1.35) in the second, and 1.20 (95% CI, 1.06–1.36) in the third. By contrast, in African Americans (n ~ 47,000) LBW was associated with CO; a 1-ppm increase in CO concentration was associated with an RR of 1.43 (95% CI, 1.18–1.74) in the first trimester and 1.75 (95% CI, 1.50–2.04) in the third trimester. No effects were seen in Hispanics (n ~ 13,000), although this may have been due to a lower statistical power in this group.

Lin et al. (2001b) compared the rates of adverse pregnancy outcomes in an area polluted by the petrochemical industry and in a control area in Taiwan. The exposed and control areas differed substantially in the levels of air pollution; for example, the differences in the mean concentrations of PM_{10} was 26.7 μg/m^3. The RR of term LBW, when the petrochemical municipality was compared with the control municipality, was 1.77 (95% CI, 1.00–3.12).

Ha et al. (2001) examined full-term births from 1996 through 1997 in Seoul, South Korea, to determine the association between LBW and exposure to CO, SO_2, NO_2, TSP, and O_3 in the first and third trimesters. They found that ambient CO, SO_2, NO_2, and TSP concentrations during the first trimester of pregnancy were associated with LBW; the RRs were 1.08 (95% CI, 1.04–1.12) for CO, 1.06 (95% CI, 1.02–1.10) for SO_2, 1.07 (95% CI, 1.03–1.11) for NO_2, and 1.04 (95% CI, 1.00–1.08) for TSP.

Vassilev et al. (2001b) used U.S. Environmental Protection Agency (EPA) Cumulative Exposure Project data to investigate the association between outdoor airborne polycyclic organic matter (POM) and adverse reproductive outcomes in New Jersey for newborns born in 1991–1992. The RR of LBW in term babies, comparing the highest and the lowest exposure groups, was 1.31 (95% CI, 1.21–1.43).

Bobak et al. (2001) tested the hypothesis that air pollution is related to LBW on data from a British 1946 cohort. They found a strong association between birth weight and air pollution index based on coal consumption. After controlling for a number of potential confounding variables, babies born in the most polluted areas (annual mean concentration of smoke > 281 μg/m^3) were on average 82 g (95% CI, 24–140) lighter than those born in the areas with the cleanest air (mean smoke concentration < 67 μg/m^3).

Chen et al. (2002) examined the association between PM_{10}, CO, and O_3 and birth weight in northern Nevada (USA) from 1991 through 1999. The results suggested that a 10-μg/m^3 increase in the mean PM_{10} concentrations during the third trimester of pregnancy was associated a reduction in birth weight of 11 g (95% CI, 2.3–19.8).

Wilhelm and Ritz (2003) studied the effect on LBW of residential proximity to heavy traffic in Los Angeles County, California (USA) in 1994–1996. The risk of term LBW increased by 19% for each 1 ppm increase in the mean annual concentration of background CO. In addition, an elevated risk was observed for women whose third trimester fell during the fall/winter months (RR, 1.39; 95% CI, 1.16–1.67); this is probably due to the more stagnant air conditions during the winter period. Overall, the study reported an approximately 10–20% increase in the risk of term LBW in infants born to women exposed to high levels of traffic-related air pollution.

A time-series study in Vancouver, Canada, found that LBW was associated with SO_2 in the first month of pregnancy (OR per 5 ppb increase, 1.11; 95% CI, 1.01–1.22); NO_2, CO, and O_3 were not independently associated with LBW (Liu et al. 2003). Particles were not measured.

A time-series study in Sao Paulo, Brazil, found that birth weight was inversely related to CO in the first trimester; after controlling for potential confounders, a 1-ppm increase in the mean CO concentration in the first trimester was associated with a 23-g (95% CI, 5–41 g) reduction in birth weight (Gouveia et al. 2004).

The results of studies of outdoor exposures are complemented by studies of indoor and personal exposures (not included in Table 2). Boy et al. (2002) compared the association between birth weight and the type of fuel (open fires with wood smoke, chimney stove, and electricity/gas) used in kitchens by mothers in rural Guatemala during pregnancy. The use of open fire produced average levels of 24-hr PM_{10} of about 1,000 μg/m^3. Babies of mothers using wood fuel and open fires were on average 63 g (95% CI, 0.4–126 g) lighter than those of women using electricity/gas. Perera et al. (2003) evaluated the effects of prenatal exposure to airborne carcinogenic polycyclic aromatic hydrocarbons (PAHs) monitored during pregnancy by personal air sampling in a sample of 263 nonsmoking African-American and Dominican women in New York. The mean total PAH exposure was 3.7 ng/m^3 (range, 0.4–36.5 ng/m^3). Among African Americans, high prenatal exposure to PAHs was associated with lower birth weight (p = 0.003) and smaller head circumference (p = 0.01). No such effects were observed among Dominican women.

Several methodologic issues should be considered in the interpretation of these studies. First, could chance (random error) play a role here? In several of the studies reviewed above, there is a potential problem of multiple comparisons. The more comparisons that are made, the higher the probability that some of them will be "statistically significant." In some instances, a more stringent use of statistical testing would be useful. Especially in studies where exposures to different pollutants at different pregnancy periods were analyzed, some of the associations could be chance findings. In addition, exposures in different pregnancy periods and concentrations of different pollutants are mutually correlated, and efforts to separate their effects are not reliable.

Second, as with infant mortality, confounding by socioeconomic factors and maternal smoking could affect the results. Overall, however, this seems unlikely for the same reasons as those listed above in "Air pollution and childhood mortality." In addition, some of the studies were able to control for social conditions and maternal smoking at the individual level, and the results were essentially identical.

In terms of the magnitude of the effect, the results were consistent in suggesting that the effects are relatively small. For comparison, it has been estimated that active smoking in pregnancy leads to a reduction in birth weight by approximately 150–200 g (Adriaanse et al. 1996), and exposure to environmental tobacco smoke in pregnancy results in birth weight reduction by approximately 20–30 g (Windham et al. 1999). There were also substantial inconsistencies in the results with respect to the importance of individual pollutants and the timing of critical exposure. The extent of the inconsistencies was such that the studies were not "combinable" into a formal meta-analysis to produce pooled effect estimates, although it is possible that the mix of pollutants differs between different settings and that this underlies the discrepancies in results.

The evidence suggests causality of the effect of air pollution on birth weight. However, given the potential problem with multiple comparisons and the heterogeneity of results, further studies are needed to confirm that the

effect is indeed causal, to clarify the most vulnerable periods of pregnancy and the role of individual pollutants, and to examine whether the impaired reproductive outcomes have any long-term consequences on child health.

Air pollution and premature births. Perhaps the first study that suggested a possible association between air pollution and preterm births was the Nashville Air Pollution Study; the results suggested that dust fall (a measure of particulate pollution) was associated with neonatal deaths among premature births (Sprague and Hagstrom 1969). However, the study did not address the question of preterm births specifically, and there were concerns about confounding by socioeconomic variables. It was only in the 1990s when this issue was investigated in more detail (Table 3).

The first "modern" investigation of the possible influence of air pollution on premature birth was a time-series study in Beijing, China, conducted by Xu et al. (1995). The study found an inverse relationship between gestational age and the concentration of SO_2 and TSP; the RRs of premature birth associated with a 100-μg/m^3 increase in the mean SO_2 and TSP concentrations during pregnancy, after controlling for potential confounders, were 1.21 (95% CI, 1.01–1.45) and 1.10 (95% CI, 1.01–1.20), respectively. Trimester-specific effects were not studied.

Bobak (2000) examined the relation between premature birth and ambient NO_x, SO_2, and TSP during each trimester. The association was strongest for SO_2, weaker for TSP, and only marginal for NO_x. For exposures during the first trimester, the RRs associated with a 50-ng/m^3 increase in pollutant concentrations were 1.27 (95% CI, 1.16–1.39) and 1.18 (95% CI, 1.05–1.31) for SO_2 and TSP, respectively. The effects of pollutants on premature births in the later two trimesters were weak.

The possible impact of CO, NO_2, O_3, and PM_{10} on premature birth was studied by Ritz et al. (2000) in Southern California. After adjustment for a number of biologic, social, and ethnic covariates, premature births were associated with CO and PM_{10} in the first gestational month and during late pregnancy. The RR associated with PM_{10} during the first gestational month was 1.16 (95% CI, 1.06–1.26); exposure in the last 6 weeks of gestation was associated with an RR of 1.20 (95% CI, 1.09–1.33). The association of premature birth with CO levels was not consistent throughout the study area.

In a study in a petrochemically polluted area in Taiwan, Lin et al. (2001a) found an RR of preterm birth in the polluted area, compared with the clean area, of 1.41 (95% CI, 1.08–1.82), after controlling for potential confounders.

The Vancouver time-series study found that the risk of preterm birth was associated with SO_2 and CO during the last month of pregnancy; the ORs were 1.09 (1.01–1.19 per 5-ppb increase) and 1.08 (1.01–1.15 per 1-ppm increase), respectively (Liu et al. 2003).

The interpretation of the studies of preterm birth is complicated by similar issues as in the case of birth weight: the issue of multiple comparisons, and the inconsistency of the results in terms of the role of individual pollutants and the timing of exposure. In addition, there have been fewer studies of premature birth than of birth weight. We therefore conclude that the evidence, as yet, is insufficient to infer causality, but further studies are justified.

Air pollution and IUGR. IUGR is defined as birth weight below the 10th percentile of birth weight for gestational age and sex. IUGR is an interesting end point that may predict functional changes in adulthood, such as hypertension and coronary heart disease. The studies of the relationship between IUGR and air pollution are summarized in Table 4.

Dejmek et al. (1999) examined the impact of PM_{10} and $PM_{2.5}$ on IUGR in a highly polluted area of northern Bohemia (Teplice District). The mean concentration of pollutants in each month of gestation for each mother were estimated from continuous monitoring data. A significantly increased risk of giving birth to a child with IUGR was established for mothers who were exposed to PM_{10} levels > 40 μg/m^3 or $PM_{2.5}$ > 27 μg/m^3 during the first month of gestation. The adjusted odds ratio (AOR) associated with a 10-μg/m^3 increase in PM_{10} was 1.25 (95% CI, 1.08–1.56); a similar, although weaker, association was seen for $PM_{2.5}$. There was no association between IUGR and particulate levels in later gestational months or with SO_2, NO_x, or O_3 (Dejmek et al. 1996).

The question of IUGR was addressed again in a reanalysis of an extended data set (Dejmek et al. 2000). Compared with exposure to the mean PM_{10} of < 40 μg/m^3 during the first month of gestation, the AOR was 1.44 (95% CI, 1.03–2.02) for the medium-exposure group (PM_{10} 40 to < 50 μg/m^3) and 2.14 (95% CI, 1.42–3.23) for PM_{10} of ≥ 50 μg/m^3. Using a continuous exposure, the AOR of IUGR was 1.19 (CI, 1.06–1.33) per 10-μg/m^3 increase of PM_{10} in the first gestational month.

In further analyses of this cohort, Dejmek et al. (2000) investigated the association between carcinogenic PAHs and IUGR in

Table 3. Air pollution and premature births.

Pollutant	Results	Reference
SO_2	AOR = 1.21 (95% CI, 1.01–1.45) for 100 μg/m^3 increase	Xu et al. 1995
TSP	AOR = 1.10 (95% CI, 1.01–1.20) for 100 μg/m^3 increase	
SO_2	AOR = 1.27 (95% CI, 1.16–1.39) for 50 μg/m^3 increase in the 1st trimester	Bobak 2000
TSP	AOR = 1.18 (95% CI, 1.05–1.31) for 50 μg/m^3 increase in the 1st trimester	
CO	NE	Ritz et al. 2000
NO_2	NE	
O_3	NE	
PM_{10}	RR = 1.16 (95% 1.06–1.26) for 50 μg/m^3 increase in the 1st trimester	
$SO_2 + NO_2 + PM_{10}$	AOR = 1.41 (91% CI, 1.08–1.82) comparing petrochemical and control municipalities	Lin et al. 2001a

Abbreviations: AOR, adjusted odds ratio; NE, no effect.

Table 4. Air pollution and IUGR.

Outcome	Pollutant	Results	Reference
IUGR	SO_2	NE	Dejmek et al. 1996
	NO_x	NE	
	O_3	NE	
IUGR	PM_{10}	AOR = 2.64 (95% CI, 1.48–4.71) comparing PM_{10} > 50 μg/m^3 with PM_{10} < 40 μg/m^3 in the first month of pregnancy	Dejmek et al. 1999
	$PM_{2.5}$	AOR = 2.11 (95% CI, 1.20–3.70) comparing $PM_{2.5}$ > 37 μg/m^3 with $PM_{2.5}$ < 27 μg/m^3 in the first month of pregnancy	
IUGR	PM_{10}	AOR = 2.14 (95% CI, 1.42–3.23) comparing PM_{10} > 50 μg/m^3 with PM_{10} < 40 μg/m^3 in the first month of pregnancy	Dejmek et al. 2000
	$PM_{2.5}$	AOR = 1.96 (95% CI, 1.02–3.11) comparing $PM_{2.5}$ > 37 μg/m^3 with $PM_{2.5}$ < 27 μg/m^3 in the first month of pregnancy	
	carc-PAHs	AOR = 2.15 (95% CI, 1.27–3.63) comparing carc-PAHs > 30 μg/m^3 with carc-PAHs < 15 μg/m^3 in the first month of pregnancy	
SGA	POM	AOR = 1.22 (95% CI, 1.16–1.27) comparing highest vs. lowest exposure groups for the term birth	Vassilev et al. 2001a

Abbreviations: AOR, adjusted odds ratio; carc-PAHs, carcinogenic-PAHs; NE, no effect; SGA, small for gestational age.

two Czech districts: Teplice and Prachatice. In the Teplice data, there was a highly significant increase of IUGR with exposures to carcinogenic PAHs (benz[*a*]anthracene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*g,h,i*]perylene, benzo[*a*]pyrene, chrysene, dibenz[*a,h*]anthracene, and indeno[1,2,3-*c,d*]pyrene) above 15 ng/m^3. Again, the effect was specific for the first gestational month. The AORs were 1.59 (95% CI, 1.06–2.39) for medium levels of carcinogenic PAHs and 2.15 (95% CI, 1.27–3.63) for high exposure levels. Using a continuous measure of exposure, a 10 ng/m^3 increase in carcinogenic PAH level was associated with an AOR of 1.22 (95% CI, 1.07–1.39). Although there was no effect of PM_{10} on IUGR found in Prachatice, the association between carcinogenic PAHs and IUGR was close to that found in Teplice. Again, the only consistent association between carcinogenic PAHs and IUGR was observed in the first gestational month: compared with the lowest category of exposure to carcinogenic PAHs, the AOR of IUGR was 1.63 (95% CI, 0.87–3.06) in the medium category and 2.39 (95% CI, 1.01–5.65) in the highest category.

The analysis of the Czech national birth register linked with air pollution data did not reveal any significant association between IUGR and ambient levels of NO_x, SO_2, and TSP (Bobak 2000). The discrepancy between the Czech studies is probably related to exposure measurement. PAHs appear to be the critical exposure for IUGR, but PAHs were not measured by the national monitoring system used for exposure estimation by Bobak (2000).

Vassilev et al. (2001a) examined the association of POM in outdoor air with "small for gestational age" (SGA) births (the definition of SGA is identical to that of IUGR). Information from birth certificates in New Jersey (USA) from 1991 through 1992 was combined with data on air toxicity derived from the U.S. EPA Cumulative Exposure Project, using the predicted POM concentrations from annual exposure estimates. The AOR for term SGA in the highest exposure tertile (0.61–2.83 μg/m^3, which includes about 89% of the state's births) was 1.22 (95% CI, 1.16–1.27), suggesting that residential exposure to airborne POM is associated with an increased prevalence of IUGR.

In the Vancouver study, using the time-series approach, SO_2, NO_2, and CO in the first month of pregnancy were associated with IUGR; the ORs were 1.07 (95% CI, 1.01–1.13) per 5-ppb increase, 1.05 (95% CI, 1.01–1.10) per 10-ppb increase, and 1.06 (95% CI, 1.01–1.10) per 1-ppm increase, respectively (Liu et al. 2003). Data on exposures to particles or PAHs were not available in that study.

As with studies of birth weight and preterm births, the reviewed studies of IUGR produced inconsistent results, and the interpretation is complicated by multiple comparisons (Bobak 2000; Liu et al. 2003) and mutual correlations of exposures. The results by Dejmek et al. (1999, 2000) and Liu et al. (2003) suggest that the first month was the most sensitive period for the effect of air pollutants, but further studies should clarify this issue. Data by Dejmek et al. (2000) and Vassilev et al. (2001a) imply a critical role of PAHs. It is possible that carcinogenic PAHs are responsible for the biologic activity of complex mixtures adsorbed to respirable air particles that can result in IUGR. With the increase in traffic, the significance of PAHs in Europe is growing, but their monitoring remains scarce. At present, the evidence is insufficient to infer causality, but further studies are required.

Air pollution and birth defects. At present, the evidence on the relation between outdoor air pollution and birth defects is limited to only one report. Ritz et al. (2002) evaluated the effect of CO, NO_2, O_3, and PM_{10} on the occurrence of birth defects in Southern California for the period 1987–1993. The average monthly exposure for each pollutant throughout pregnancy was calculated. Dose–response patterns were observed for CO exposure in the second month of gestation and ventricular septal defects (AOR for the highest vs. lowest quartile of exposure, 2.95; 95% CI, 1.44–6.05) and for exposure to O_3 in the second month and aortic artery and valve defects (AOR, 2.68; 95% CI, 1.19–6.05).

Given the lack of studies on air pollution and birth defects, the evidence base available so far is insufficient to draw conclusions about causality. Further studies are required to support these results by Ritz et al. (2002).

Discussion

The studies reviewed above indicate that ambient air pollution is inversely associated with a number of birth outcomes. This is a relatively new area of environmental epidemiology; most reports have emerged over the last 10 years. A critical assessment of the evidence is therefore timely. Issues pertinent to different studies were considered separately above. Here, we consider questions common to all reproductive outcomes: publication bias, measurement of exposure, and the biologic plausibility of the effects on birth weight, IUGR, and preterm births.

Publication bias. Negative studies are less likely to be published, and studies published in non-English journals are less likely to be included in reviews. We included all studies we were able to identify. We cannot exclude the possibility that some negative studies, especially in the earlier period, remain unpublished. However, given the recent interest in this topic, it is likely that most studies over the last decade have been published or at least presented at conferences.

Measurement of exposure. Most studies relied on routine monitoring of air pollution in large areas. Extrapolation from citywide or areawide measurements to individual exposures can be problematic. In this context, molecular epidemiologic studies are particularly valuable for the interpretation of the epidemiologic data. The molecular epidemiologic studies used biomarkers of exposure, mainly as the DNA adducts measured by ^{32}P-postlabeling and PAH–DNA adducts assessed by enzyme-linked immunosorbent assays (Šrám and Binková 2000). Overall, these studies suggest that DNA adduct levels in maternal blood and placentas are higher in areas with higher pollution levels (Šrám et al. 1999; Whyatt et al. 1998), and significant district and seasonal differences in DNA adducts were found in subgroups with the *GSTM1* null genotype (Topinka et al. 1997a, 1997b). The increase in the levels of DNA adducts related to pollution is similar to, but smaller in magnitude than, the differences between smoking and nonsmoking mothers. All this indicates that ambient air pollution levels do translate to higher individual exposures, even for unborn babies. This provides support for the validity of the epidemiologic studies reviewed above.

DNA adducts in placentas and the impact of PAHs on IUGR are consistent with findings of *in vitro* studies that exposure to extracts of urban air PM increased DNA adducts and embryotoxicity (Binková et al. 1999, 2003). These findings indicate that particle-bound carcinogenic PAH concentrations may be taken as an index of the biologically active components in samples of particulates in air.

Biologic plausibility. The molecular epidemiologic studies suggest biologic mechanisms for the effect of air pollution on birth outcomes. It has been shown that the levels of DNA adducts are positively related to risk of IUGR (Dejmek at al 2000; Šrám et al. 1999), birth weight, birth length, and head circumference (Perera et al. 1998, 1999), and hypoxanthine-guanine phosphoribosyltransferase (*HPRT*) locus mutation frequency in infants (Perera et al. 2002).

PAHs and/or their metabolites may bind to the aryl hydrocarbon receptor (AhR) and accumulate in the nucleus of cells, resulting in increased rates of mutagenesis. Because PAHs bind to the AhR, it may result in antiestrogenic activity through increased metabolism and the depletion of endogenous estrogens (Carpenter et al. 2002), thus disrupting the endocrine system by altering steroid function. Bui et al. (1986) hypothesized that benzo[*a*]pyrene exposure may interfere with uterine growth during pregnancy because of its antiestrogenic effects, thereby disrupting the endocrine system. Fetal toxicity

may be further caused by DNA damage resulting in activation of apoptotic pathways (Nicol et al. 1995) or binding to receptors for placental growth factors resulting in decreased exchange of oxygen and nutrients (Dejmek et al. 2000).

The finding of higher DNA adduct levels in the infant compared with the mother suggests an increased susceptibility of the developing fetus to DNA damage (Perera et al. 1999). With respect to IUGR, it appears that the increased risk is principally due to exposure to carcinogenic PAHs. This finding is consistent with the idea of a primary role for carcinogenic PAHs in fetal growth modulation (Guyda 1991; MacKenzie and Angevine 1981; Rigdon and Rennels 1964; Zhang et al. 1995). Perera et al. (2003) labeled PAHs as significant independent determinants of birth outcomes. In addition, there appears to be an interaction between exposure to PAHs and genotypes that produce DNA adducts (Whyatt et al. 2001).

Although the specific steps of these pathways need to be further clarified, the molecular epidemiology studies and the similarity of effects of air pollution to those of smoking (Adriaanse et al. 1996; Windham et al. 1999) support the biologic plausibility of the effects.

Conclusions

Overall, there is evidence implicating air pollution in adverse effects on different birth outcomes, but the strength of the evidence differs between outcomes. The evidence is sufficient to infer a causal relationship between particulate air pollution and respiratory deaths in the postneonatal period. For air pollution and birth weight, the evidence is suggestive of causality, although further studies are needed. For preterm births and IUGR, the evidence as yet is insufficient to infer causality, but the available evidence justifies further studies. Molecular epidemiologic studies suggest possible biologic mechanisms for the effect on birth weight, premature birth, and IUGR and support the view that the relation between pollution and these birth outcomes is genuine. For birth defects, the evidence base so far is insufficient to draw conclusions. In terms of exposure to specific pollutants, particulates seem the most important for infant deaths, and the effect on IUGR seems linked to PAHs, but the existing evidence does not allow precise identification of the different pollutants and the timing of exposure that can result in adverse pregnancy outcomes.

On the basis of this review, we suggest several priorities for future research. First, it remains to be confirmed that the effects on birth weight, prematurity, and IUGR are genuine and causal. Second, it is important to identify the most vulnerable period of exposure in pregnancy. Third, the contribution of different pollutants needs to be established. Fourth, the biologic pathways require further clarification. And finally, with the increasing attention to the life course, it would be interesting to examine whether early exposures and impaired reproductive outcome have any long-term consequences in later life.

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OPEN

National Prevalence and Effects of Multiple Chemical Sensitivities

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Objective: The aim of this study was to assess the prevalence of multiple chemical sensitivities (MCS), its co-occurrence with asthma and fragrance sensitivity, and effects from exposure to fragranced consumer products. **Methods:** A nationally representative cross-sectional population-based sample of adult Americans ($n = 1137$) was surveyed in June 2016. **Results:** Among the population, 12.8% report medically diagnosed MCS and 25.9% report chemical sensitivity. Of those with MCS, 86.2% experience health problems, such as migraine headaches, when exposed to fragranced consumer products; 71.0% are asthmatic; 70.3% cannot access places that use fragranced products such as air fresheners; and 60.7% lost workdays or a job in the past year due to fragranced products in the workplace. **Conclusion:** Prevalence of diagnosed MCS has increased over 300%, and self-reported chemical sensitivity over 200%, in the past decade. Reducing exposure to fragranced products could help reduce adverse health and societal effects.

Keywords: asthma, chemical sensitivity, fragrance, MCS, multiple chemical sensitivities

BACKGROUND

Multiple chemical sensitivities (MCS) is a medical condition characterized by adverse health effects from exposure to common chemicals and pollutants, from products such as pesticides, new carpet and paint, renovation materials, diesel exhaust, cleaning supplies, perfume, scented laundry products, and air fresheners.[1,2] MCS can cause a range of acute, chronic, multiorgan, and disabling health effects, such as headaches, dizziness, cognitive impairment, breathing difficulties, heart palpitations, nausea, mucous membrane irritation, and asthma attacks.[3] Individuals with MCS may not receive a diagnosis but nonetheless exhibit the condition of chemical sensitivity. Previous studies have found that MCS often co-occurs with asthma,[4] as well as fragrance sensitivity,[5] characterized by adverse health effects from exposure to fragranced consumer products.[6]

While MCS is perhaps the most common term, the condition is also known by other terms, such as chemical intolerance or environmental illness (specific to chemical exposures).[3] MCS follows a two-step process of (i) initiation of the disease, often from exposure to petrochemical products, and then (ii) triggering of symptoms when exposed to problematic chemicals, often at low levels.[3,7] While significant efforts have been devoted to developing case definitions and diagnostic criteria,[3,8,9] a single internationally agreed-upon standard for prevalence studies is not yet established. Nonetheless, prior population-based studies of MCS, using specific and often different definitions and criteria, offer useful data on the extent and severity of the condition.

In the USA, two previous nationally representative studies, conducted in 2002 to 2003[4] and 2005 to 2006,[5] investigated the prevalence of MCS by using the key question developed by the California Department of Health Services (CDHS)[10]: ''Compared to other people, do you consider yourself allergic or unusually sensitive to everyday chemicals like those in household cleaning products, paints, perfumes, detergents, insect spray and things like that?'' This criterion reflects self-reported chemical sensitivity. To ascertain a medical diagnosis of MCS, the survey asked, ''Has a doctor or health care professional ever told you that you have multiple chemical sensitivities?'' This criterion reflects medically diagnosed MCS. These two USA studies found (respectively) a prevalence of 11.1% and 11.6% self-reported chemical sensitivity and 2.5% and 3.9% medically diagnosed MCS.

At the state and regional level in the USA, using the CDHS criteria, a survey of 4046 Californians in 1995[10] found a prevalence of 15.9% self-reported chemical sensitivity and 6.3% medically diagnosed MCS. A survey of 1583 metropolitan Atlantans in 1999 to 2000,[1] also using the CDHS criteria, found a prevalence of 12.6% self-reported chemical sensitivity and 3.1% diagnosed MCS. A survey of 1027 individuals in North Carolina in 1993,[7] using a question similar to CDHS, found a prevalence of 33% chemical sensitivity.

In Sweden, using the chemical sensitivity scale for sensory hyperreactivity (CSS-SHR),[11] an investigation of 1387 adults in Skövde found a prevalence of 33% of self-reported general odor intolerance, or being bothered by strong or pungent odors, such as perfume, cleaning agents, or flower scents.[12] Also in Sweden, a survey of 3406 adult respondents from Västerbotten found 12.2% reported chemical intolerance to odorous pungent chemicals, such as perfumes and cleaning agents, and 3.3% were physician-diagnosed with chemical intolerance.

In Australia, a population-based survey of 4009 adults in South Australia in 2001 to 2002,[13] using a variation of the CDHS question, found a prevalence of 15.9% of self-reported chemical sensitivity and 1% medically diagnosed MCS. In Japan, a national survey of 7245 adults,[14] using the Quick Environmental Exposure and Sensitivity Inventory (QEESI) questionnaire,[9] found a prevalence of 7.5% for chemical intolerance. In Korea, a survey of 379 adults, also using the QEESI, found a prevalence of 16.4% for chemical intolerance.[15]

While these studies provide useful context, we lack recent nationally representative data in the USA. A primary objective of this study is to provide a current estimate of the prevalence of MCS in the American population. Also, given previous studies indicating connections, a second objective is to investigate the co-occurrence of MCS with asthma and with fragrance sensitivity. Finally, because fragranced products are a common trigger, a third objective is to investigate the effects of exposure to fragranced products for individuals with MCS, which points to ways to reduce potential adverse effects.

METHODS

To assess the prevalence and effects of MCS, an online survey was conducted with a random national cross-sectional sample of the

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No external sources of funding were received for this work.
The authors have no conflicts of interest.

Supplemental digital contents are available for this article. Direct URL citation appears in the printed text and is provided in the HTML and PDF versions of this article on the journal's Web site (www.joem.org).

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DOI: 10.1097/JOM.0000000000001272

adult US population, representative of age, gender, and region ($n = 1137$, 95% confidence level, 3% margin of error), drawn from a large national panel (over 5,000,000 people) held by Survey Sampling International. The survey instrument was developed and tested over a 2-year period before full implementation in June 2016. Response rate was 95%, and all responses were anonymous. (Details on survey methodology, questions, and data are provided in the files ''Survey Methodology'' and ''Survey Data'' as Supplemental Digital Content, http://links.lww.com/JOM/A412 and http://links.lww.com/JOM/A413.)

To promote comparability, the survey replicated questions from previous large US national, state, and regional MCS prevalence studies.[1,4,5,7,10] In accordance, to ascertain medically diagnosed MCS, the survey asked, ''Has a doctor or health care professional ever told you that you have multiple chemical sensitivities?'' To ascertain self-reported chemical sensitivity, the survey asked, ''Compared to other people, do you consider yourself allergic or unusually sensitive to everyday chemicals like those in household cleaning products, paints, perfumes, detergents, insect spray and things like that?''

To ascertain asthma, the survey asked ''Has a doctor or health care professional ever told you that you have asthma or an asthma-like condition?'' and then further asked to specify whether asthma or an asthma-like condition. The term ''asthmatic'' will be used herein to encompass individuals with either asthma or an asthma-like condition or both.

To ascertain fragrance sensitivity, the survey investigated health effects associated with exposures to fragranced consumer products. A ''fragranced consumer product,'' or ''fragranced product'' for brevity, is a chemically formulated product with the addition of a fragrance or scent.[6] An individual was considered to characterize fragrance sensitivity if they experienced one or more types of health problems from one or more types of fragranced products and exposure contexts.

Fragranced product types were categorized as follows: air fresheners and deodorizers, personal care products, cleaning supplies, laundry products, household products, fragrance, and other. Health effects were categorized as follows: migraine headaches; asthma attacks; neurological problems; respiratory problems; skin problems; cognitive problems; mucosal symptoms; immune system problems; gastrointestinal problems; cardiovascular problems; musculoskeletal problems; and other. (Additional details on specific product types and health effects within each category, along with response data, are provided in the file ''Survey Data'' as Supplemental Digital Content, http://links.lww.com/JOM/A413.)

Specific exposure contexts were air fresheners or deodorizers used in public restrooms and other environments, scented laundry products coming from a dryer vent, being in a room after it was cleaned with scented cleaning products, being near someone wearing a fragranced product, entering a business with the scent of fragranced products, fragranced soap used in public restrooms, and ability to access environments that used fragranced products. The survey also investigated effects of fragrance exposure in the workplace, access to public places that used fragranced products, and preferences for fragrance-free environments and policies. Data on fragranced product exposures and effects were derived from a survey of the general population,[6] while the present study focuses specifically on effects on the subpopulations of individuals with MCS or chemical sensitivity.

RESULTS

A national prevalence of 12.8% medically diagnosed MCS, 25.9% self-reported chemical sensitivity, and 27.5% either or both, was assessed by the survey. (See Table 1.) Compared with previous studies,[4,5] the prevalence of diagnosed MCS has increased over three times (2.5%, 3.9% to 12.8%) and self-reported chemical sensitivity has increased over two times (11.1%, 11.6% to 25.9%) in a little over 10 years.

In addition, 71.0% of those with MCS are asthmatic: diagnosed with asthma (40.0%), an asthma-like condition (34.5%), or both. Also, 59.2% with chemical sensitivity are asthmatic: diagnosed with asthma (35.0%), an asthma-like condition (26.2%), or both (See Table 1). Compared with previous studies,[4,5] the co-occurrence of asthma with diagnosed MCS (42.3%, 39.0% vs

TABLE 1. Prevalence and Co-Occurrence of MCS and Chemical Sensitivity With Asthma and Fragrance Sensitivity

	Gen Pop	MCS Diag	ChemSens	MCS/ChemSens
Total (*N*)	1,137	145	294	313
(% relative to general population)	100.0%	12.8%	25.9%	27.5%
	N	***N***	***N***	***N***
	% of Column Total	**% of Column Total**	**% of Column Total**	**% of Column Total**
MCS diagnosed	145	145	126	145
	12.8%	100.0%	42.9%	46.3%
Chemically sensitive	294	126	294	294
	25.9%	86.9%	100.0%	93.9%
MCS diagnosed or chemically sensitive (or both)	313	145	294	313
	27.5%	100.0%	100.0%	100.0%
Asthma diagnosed	173	58	103	105
	15.2%	40.0%	35.0%	33.5%
Asthma-like condition diagnosed	142	50	77	80
	12.5%	34.5%	26.2%	25.6%
Asthmatic (asthma or asthma-like condition or both)	305	103	174	179
	26.8%	71.0%	59.2%	57.2%
Fragrance sensitive	394	125	238	247
	34.7%	86.2%	81.0%	78.9%

ChemSens, self-reported chemical sensitivity; Gen Pop, general population (including subpopulations of MCS and ChemSens); MCS Diag, medically diagnosed with MCS; MCS/ChemSens, medically diagnosed with MCS, or self-reported chemical sensitivity, or both.

40.0%) and with chemical sensitivity (34.2%, 34.9% vs 35.0%) is relatively similar.

Fragranced consumer products were found to trigger a range of adverse health and societal effects. When exposed to fragranced consumer products, 86.2% of those with MCS experience one or more types of health problems, including respiratory difficulties (50.3%), migraine headaches (46.9%), mucosal symptoms (46.9%), skin problems (37.9%), and asthma attacks (31.7%). Similarly, 81.2% of those with chemical sensitivity report one or more types of health problems when exposed to fragranced products (see Tables 1 and 2).

Specific exposures triggering health problems include air fresheners and deodorizers (67.6%), scented laundry products coming from a dryer vent (57.9%), being in a room recently cleaned with scented products (67.6%), being near someone wearing a fragranced product (65.5%), and in general fragranced consumer products (73.1%) (see Table 3, and the file ''Survey Data'' as Supplemental Digital Content, http://links.lww.com/JOM/A413).

For 76.0% of people with MCS, the severity of these health problems was potentially disabling according to the criterion of the Americans with Disabilities Act Amendments Act of 2008 (ADAAA), asked by the question: ''Do any of these health problems substantially limit one or more major life activities, such as seeing, hearing, eating, sleeping, walking, standing, lifting, bending, speaking, breathing, learning, reading, concentrating, thinking, communicating, or working, for you personally?''[16] (See Table 4.)

Fragranced products also restrict access in society: 58.6% of individuals with MCS are unable to use public restrooms that have an air freshener, deodorizer, or scented product; 55.2% are unable to wash their hands in a public place if the soap is fragranced; 63.4% enter a business but then want to leave as quickly as possible due to a fragranced product; and 70.3% have been prevented from going someplace because of the presence of a fragranced product that would make them sick. (See Table 4.)

Significantly, 60.7% of those with MCS lost workdays or a job in the past year due to illness from fragranced product exposure in the workplace. Further, 71% of those with MCS would support a fragrance-free policy in the workplace, and 82.1% would prefer that health care facilities and professionals were fragrance-free. (See Table 4.)

Demographic proportions of diagnosed MCS are 57.9% male and 42.1% female, compared with the general population of 46.2% male and 53.8% female. Thus, diagnosed MCS has a male bias (+11.7%). Previous national prevalence studies in the US found instead a slight female bias. Relative to gender and age, the highest bias (percentage MCS greater than general population) is male 25 to 34 (+12.7%). (See Table 5.)

DISCUSSION

Results of this study provide evidence that MCS is widespread and increasing in the US population: an estimated 25.6 million adults are diagnosed with MCS, and an estimated 51.8 million adults report chemical sensitivity.[17] Using the same

TABLE 2. Health Problems (Frequency and Type) Reported from Exposure to Fragranced Consumer Products

	Gen Pop	MCS Diag	ChemSens	MCS/ChemSens
Total (*N*)	1,137	145	294	313
(% relative to general population)	100.0%	12.8%	25.9%	27.5%
	N	***N***	***N***	***N***
	% of Column Total	**% of Column Total**	**% of Column Total**	**% of Column Total**
Total fragrance sensitive (*N*) (reporting one or more health problems)	394	125	238	247
(% relative to Subpopulation)	34.7%	86.2%	81.0%	78.9%
Type of health problem				
* *Migraine headaches*	179	68	124	128
	15.7%	46.9%	42.2%	40.9%
* *Asthma attacks*	91	46	75	75
	8.0%	31.7%	25.5%	24.0%
* *Neurological problems* (eg, dizziness, seizures, head pain, fainting, loss of coordination)	82	38	62	63
	7.2%	26.2%	21.1%	20.1%
* *Respiratory problems* (eg, difficulty breathing, coughing, shortness of breath)	211	73	147	148
	18.6%	50.3%	50.0%	47.3%
* *Skin problems* (eg, rashes, hives, red skin, tingling skin, dermatitis)	121	55	84	88
	10.6%	37.9%	28.6%	28.1%
* *Cognitive problems* (eg, difficulties thinking, concentrating, or remembering)	66	35	56	57
	5.8%	24.1%	19.0%	18.2%
* *Mucosal symptoms* (eg, watery or red eyes, nasal congestion, sneezing)	184	68	120	124
	16.2%	46.9%	40.8%	39.6%
* *Immune system problems* (eg, swollen lymph glands, fever, fatigue)	45	31	39	39
	4.0%	21.4%	13.3%	12.5%
* *Gastrointestinal problems* (eg, nausea, bloating, cramping, diarrhea)	63	32	53	53
	5.5%	22.1%	18.0%	16.9%
* *Cardiovascular problems* (eg, fast or irregular heartbeat, jitteriness, chest discomfort)	50	28	37	38
	4.4%	19.3%	12.6%	12.1%
* *Musculoskeletal problems* (eg, muscle or joint pain, cramps, weakness)	43	28	35	36
	3.8%	19.3%	11.9%	11.5%
* *Other*	19	2	6	6
	1.7%	1.4%	2.0%	1.9%

TABLE 3. Health Problems (Frequency and Type) Reported from Exposure to Fragranced Consumer Products

	Gen Pop	MCS Diag	ChemSens	MCS/ChemSens
	N	*N*	*N*	*N*
	% of Column Total	% of Column Total	% of Column Total	% of Column Total
Total	1,137	145	294	313
	100.0%	100.0%	100.0%	100.0%
Fragrance sensitive	394	125	238	247
	34.7%	86.2%	81.0%	78.9%
Health problems from exposure to				
Air fresheners or deodorizers	232	98	162	168
	20.4%	67.6%	55.1%	53.7%
Scented laundry products from a dryer vent	142	84	107	112
	12.5%	57.9%	36.4%	35.8%
Room cleaned with scented products	224	98	166	171
	19.7%	67.6%	56.5%	54.6%
Someone wearing a fragranced product	268	95	178	183
	23.6%	65.5%	60.5%	58.5%
Any type of fragranced consumer product	253	106	192	196
	22.3%	73.1%	65.3%	62.6%

criteria to assess MCS and chemical sensitivity as prior US national prevalence studies, this represents an increase of 300% in diagnosed MCS and 200% in self-reported chemical sensitivity in a little more than 10 years.

Among individuals diagnosed with MCS, 71.0% report being diagnosed also with asthma or an asthma-like condition. Thus, individuals with MCS are proportionally more likely to be asthmatic than individuals without MCS (prevalence odds ratio 9.6; 95% confidence interval 6.5 to 14.2).

In addition, among individuals with MCS, 86.2% report adverse health effects from exposure to fragranced consumer products. Thus, individuals with MCS are proportionally more likely to be fragrance sensitive than individuals without MCS (prevalence odds ratio 16.8; 95% confidence interval 10.3 to 27.5).

As a consequence, individuals with MCS are prevented from accessing restrooms, businesses, workplaces, and public places due to risk of adverse health effects—some potentially disabling—from fragranced consumer products. Notably, exposure to fragranced consumer products is associated with lost workdays or a job, in the past year, for 11.0% of the adult population with MCS or chemical sensitivity, representing an estimated 22 million Americans. While researchers continue to investigate which chemicals or mixtures of chemicals in fragranced consumer products could be associated with adverse effects,[18] a practical step in the meantime would be to reduce exposure to the products. For instance, 71.0% of those with MCS would support fragrance-free policies in the workplace, and 82.1% would prefer fragrance-free health care facilities and professionals, as would a majority of the US general population.[6]

Limitations of the study include the following: (a) data were based on self-reports, although a standard and accepted method for epidemiological research, and consistent with prior prevalence studies of MCS; (b) only adults (ages 18 to 65) were surveyed; (c) all possible fragranced products and health effects were not

TABLE 4. Societal Effects of Fragranced Consumer Products for Individuals with MCS

	Gen Pop	MCS Diag	ChemSens	MCS/ChemSens
	N	*N*	*N*	*N*
	% of Column Total	% of Column Total	% of Column Total	% of Column Total
Total	1,137	145	294	313
	100.0%	100.0%	100.0%	100.0%
Fragrance sensitive	394	125	238	247
	34.7%	86.2%	81.0%	78.9%
Disabling health effects from fragranced consumer products	195	95	160	164
	49.5%	76.0%	67.2%	66.4%
Unable to use restrooms in public place because of air freshener, deodorizer, or scented product	199	85	132	138
	17.5%	58.6%	44.9%	44.1%
Unable to wash hands because of fragranced soap	160	80	118	122
	14.1%	55.2%	40.1%	39.0%
Want to leave a business quickly because of fragranced product	229	92	160	164
	20.1%	63.4%	54.4%	52.4%
Prevented from going someplace because of fragranced product	258	102	168	179
	22.7%	70.3%	57.1%	57.2%
Lost workdays or job in past year due to fragranced product exposure in workplace	172	88	119	125
	15.1%	60.7%	40.5%	39.9%
Supportive of fragrance-free policy in the workplace	604	103	212	221
	53.1%	71.0%	72.1%	70.6%
Prefer fragrance-free health care facilities and professionals	623	119	236	248
	54.8%	82.1%	80.3%	79.2%

TABLE 5. Demographic Information

	Gen Pop	MCS Diag	ChemSens	ChemSens/MCS
	N	*N*	*N*	*N*
	% of Column Total	% of Column Total	% of Column Total	% of Column Total
Total	1,137	145	294	313
	100.0%	100.0%	100.0%	100.0%
Male/Female				
All males	525	84	133	145
	46.2%	57.9%	45.2%	46.3%
All females	612	61	161	168
	53.8%	42.1%	54.8%	53.7%
Gender vs age				
Male 18–24	47	7	10	12
	4.1%	4.8%	3.4%	3.8%
Male 25–34	130	35	42	47
	11.4%	24.1%	14.3%	15.0%
Male 35–44	136	30	44	48
	12.0%	20.7%	15.0%	15.3%
Male 45–54	108	4	20	20
	9.5%	2.8%	6.8%	6.4%
Male 55–65	104	8	17	18
	9.1%	5.5%	5.8%	5.8%
Female 18–24	78	8	19	21
	6.9%	5.5%	6.5%	6.7%
Female 25–34	135	16	34	35
	11.9%	11.0%	11.6%	11.2%
Female 35–44	155	16	45	47
	13.6%	11.0%	15.3%	15.0%
Female 45–54	144	13	41	42
	12.7%	9.0%	13.9%	13.4%
Female 55–65	100	8	22	23
	8.8%	5.5%	7.5%	7.3%

included, although the low percentages for responses in the ''other'' category indicates the survey captured the primary products and effects; and (d) MCS and chemical sensitivity lack standard diagnostic criteria, although the survey replicated questions from prior large-scale USA prevalence studies to promote comparability.

CONCLUSION

The prevalence of MCS has increased across the American population, and it frequently co-occurs with asthma and fragrance sensitivity. Moreover, fragranced consumer products, such as air fresheners and scented cleaning products, trigger significant adverse health and societal effects among individuals with MCS. Reducing exposure to fragranced products, such as through fragrance-free policies, would be an important practical step to reduce the adverse effects.

ACKNOWLEDGEMENTS

I thank Amy Davis, John Barrie, Robert Damiano, and Survey Sampling International for their valuable contributions. This article is written as a tribute to my departed colleague, Dr. Stanley Caress.

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Environment International 133 (2019) 105151

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Associations of wildfire smoke $PM_{2.5}$ exposure with cardiorespiratory events in Colorado 2011–2014

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ARTICLE INFO

Handling Editor: Hanna Boogaard

Keywords:
Particulate matter
Wildfire
Air quality
Epidemiology
Climate change
Respiratory
Cardiovascular

ABSTRACT

Background: Substantial increases in wildfire activity have been recorded in recent decades. Wildfires influence the chemical composition and concentration of particulate matter ≤2.5 μm in aerodynamic diameter ($PM_{2.5}$). However, relatively few epidemiologic studies focus on the health impacts of wildfire smoke $PM_{2.5}$ compared with the number of studies focusing on total $PM_{2.5}$ exposure.

Objectives: We estimated the associations between cardiorespiratory acute events and exposure to smoke $PM_{2.5}$ in Colorado using a novel exposure model to separate smoke $PM_{2.5}$ from background ambient $PM_{2.5}$ levels.

Methods: We obtained emergency department visits and hospitalizations for acute cardiorespiratory outcomes from Colorado for May–August 2011–2014, geocoded to a 4 km geographic grid. Combining ground measurements, chemical transport models, and remote sensing data, we estimated smoke $PM_{2.5}$ and non-smoke $PM_{2.5}$ on a 1 km spatial grid and aggregated to match the resolution of the health data. Time-stratified, case-crossover models were fit using conditional logistic regression to estimate associations between fire smoke $PM_{2.5}$ and non-smoke $PM_{2.5}$ for overall and age-stratified outcomes using 2-day averaging windows for cardiovascular disease and 3-day windows for respiratory disease.

Results: Per 1 $\mu g/m^3$ increase in fire smoke $PM_{2.5}$, statistically significant associations were observed for asthma (OR = 1.081 (1.058, 1.105)) and combined respiratory disease (OR = 1.021 (1.012, 1.031)). No significant relationships were evident for cardiovascular diseases and smoke $PM_{2.5}$. Associations with non-smoke $PM_{2.5}$ were null for all outcomes. Positive age-specific associations related to smoke $PM_{2.5}$ were observed for asthma and combined respiratory disease in children, and for asthma, bronchitis, COPD, and combined respiratory disease in adults. No significant associations were found in older adults.

Discussion: This is the first multi-year, high-resolution epidemiologic study to incorporate statistical and chemical transport modeling methods to estimate $PM_{2.5}$ exposure due to wildfires. Our results allow for a more precise assessment of the population health impact of wildfire-related $PM_{2.5}$ exposure in a changing climate.

1. Introduction

Climate change, defined as the long-term change in global and regional weather patterns, has been extensively documented since the mid-to-late 20th century (Boudes, 2011; Incropera, 2016; The Environmental Pollution Panel, 1965; United States Environmental Protection Agency, 2017b). Despite politically charged debates regarding the cause of the change, it is clear that climate change and its resulting extreme weather events could severely impact the health and well-being of populations across the globe (Berrang-Ford et al., 2015; Kjellstrom et al., 2016; Thornton et al., 2014; Wu et al., 2016). One area that reflects the synergistic impact of climate change and human activity is the occurrence of wildfires. Notably, the Western US has seen consistent and rapid increases in wildfire activity since the 1980s. This

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https://doi.org/10.1016/j.envint.2019.105151
Received 23 May 2019; Received in revised form 26 August 2019; Accepted 2 September 2019

increase has been characterized by rises in the frequency, severity, size, and total burned area associated with wildfires (Liu et al., 2013; Westerling, 2016; Westerling et al., 2006). Fire effects are often seen at great distances from the events due to large smoke plumes, sometimes extending across multiple counties or states. States in the Rocky Mountain region continue to exhibit climatic factors conducive to fire activity—including high temperatures, low soil moisture, decreased rainfall, and increased solar radiation (Crockett and Westerling, 2018; Dawson et al., 2014; Griffin and Anchukaitis, 2014; Leung and Gustafson, 2005; Penrod et al., 2014). Conditions may become more suitable to large wildfires over time due to climate change (Keeley and Syphard, 2016; Keywood et al., 2013; Stavros et al., 2014). Consequently, wildfires place significant burdens on the human, economic, and environmental systems in areas surrounding and downwind from the burn zone. This is of particular concern given the impact that wildfire events can have on regional air quality and, subsequently, human health (Liu et al., 2015; Reid, Jerrett et al., 2016).

Wildfire smoke can produce significantly higher exposures to harmful compounds than are normally found in non-fire urban settings (Alves et al., 2011; Kim et al., 2018; Na and Cocker, 2008). Fine particulate matter ($PM_{2.5}$, airborne particles $< 2.5\,\mu m$ in aerodynamic diameter) is of particular concern due to its ability to travel deep into the human respiratory system and enter the blood stream (Dockery and Pope, 1994; Hong et al., 2017; Kim et al., 2015; Liu et al., 2015; Park and Wexler, 2008; Reid, Jerrett et al., 2016; United States Environmental Protection Agency, 2017a). Smoke particles differ in both size and composition from particles found in typical ambient PM from non-wildfire sources. It has been shown that organic compounds, such as methanol or formaldehyde, make up a significantly higher proportion of smoke $PM_{2.5}$ when compared with ambient PM (X.X. Liu, 2017; Na and Cocker, 2008). These distinctions could have differing effects on human health outcomes and may vary by fuel source. This has been shown in both in vivo and in vitro studies using human cells and mice (Kim et al., 2019; Shin et al., 2017; Xu et al., 2019). While much is left to be understood about the toxicological differences, current literature has begun to elucidate potential differences between smoke and ambient PM sources. It is, therefore, important to differentiate between smoke and non-smoke $PM_{2.5}$ when assessing the health impact of wildfires.

While numerous epidemiological studies have established the associations between ambient $PM_{2.5}$ and human health (Brook et al., 2010; Di et al., 2017; Pope and Dockery, 2006), relatively few studies have focused specifically on wildfire smoke (Rappold et al., 2017). For example, Reid et al. published a study showing a significant results for asthma during fire events (previous 2-day moving average) for a $5\,\mu g/m^3$ change in $PM_{2.5}$ concentration (Reid, Jerrett et al., 2016). While Reid et al. included satellite and chemical transport data, they were limited to the use of fire day and fire distance parameters to account for smoke PM instead of directly estimating smoke PM concentrations. Additionally, many studies are restricted to the use of ambient urban air pollution measurements, coupled with fire day indicators, to represent fire-related exposures. In addition, current guidelines for public health response to wildfire events rely heavily on changes of ambient total PM measurements due to a lack of information in wildfire-specific air quality (Lipsett et al., 2016). A few studies have distinguished among sources on larger scales (Hutchinson et al., 2018; J.C. Liu, 2017; Thelen et al., 2013). For example, Liu et al. derived metrics of smoke waves for distinguishing fire activity and evaluated the health impacts of smoke $PM_{2.5}$ (J.C. Liu, 2017). Their chemical transport model simulations, however, were on a spatial grid of 0.5×0.67 degrees, which may be too coarse to capture finer-scale spatial gradients of exposure, see Supplemental Fig. 1.

Though there is consistent evidence for associations between wildfire events and disease, questions remain regarding the relationship between wildfire smoke $PM_{2.5}$ and both respiratory and cardiovascular outcomes given the difficulty in estimating smoke $PM_{2.5}$ exposure. Developing robust methods for understanding this complex relationship is vital to understand the potential future impacts of climate and wildfire events on human health. Building upon previous studies, the goal of our study is to estimate the associations for multiple respiratory and cardiovascular acute health events in relation to wildfire smoke $PM_{2.5}$ in Colorado during the fire seasons of 2011–2014 using novel, high-resolution methods to separate wildfire smoke $PM_{2.5}$ from background ambient $PM_{2.5}$.

2. Methods

2.1. Health data

We obtained individual-level health data on daily hospitalizations and emergency department (ED) visits at all public and private hospitals for the fire seasons (May–August) of 2011–2014 from the Colorado Department of Public Health and Environment. Information included in the patient records are dates of admission, residential address, age, sex, payer information and International Classification of Diseases version 9 (ICD9) codes for primary and secondary diagnoses. Patients admitted to the hospital through the ED were only counted once, and those with elective hospitalizations were excluded from analysis.

We analyzed multiple endpoints for primary cardiovascular and respiratory diagnoses. Respiratory outcomes include asthma (ICD9: 493), bronchitis (ICD9: 490), chronic obstructive pulmonary disease (ICD9: 491, 492, and 496), upper respiratory infection (ICD9: 460–465 and 466.0), and combined respiratory disease (ICD9: 460–465, 466.0, 466.1, 466.11, 466.19, 480–486, 487, 488, 490, 491, 492, 496, and 493). Cardiovascular outcomes include ischemic heart disease (ICD9: 410–414), acute myocardial infarction (ICD9: 410), congestive heart failure (ICD9: 428), dysrhythmia (ICD9: 427), peripheral/cerebrovascular disease (ICD9: 433–437, 440, 443, 444, 451–453), and combined cardiovascular disease (ICD9: 410–414. 427, 428, 433–437, 440, 443, 444, 451–453). Due to inadequate numbers, events in children were not analyzed for COPD or any cardiovascular outcomes.

2.2. $PM2_{.5}$ and meteorological data

We sought to separate smoke $PM_{2.5}$ from ambient sources. To accomplish this, daily mean $PM_{2.5}$ concentrations were adopted and improved from our previous study by adding new data (Geng et al., 2018). Briefly, mean concentrations were estimated using a two-model approach to combine information from high-resolution satellite AOD derived from the Multi-angle Implementation of Atmospheric Correction (MAIAC) algorithm, model simulations from the Community Multiscale Air Quality Modeling System (CMAQ), and ground measurements obtained from the U.S. Environmental Protection Agency (USEPA) for fire seasons in the state of Colorado (April–September 2011–2014). The first model (i.e. AOD model) utilized random forest modeling to incorporate MAIAC AOD, smoke mask, meteorological fields and land-use variables. The second model (i.e. CMAQ model) utilized statistical downscaling to calibrate the CMAQ $PM_{2.5}$ simulations. Additional exposure modeling specifics can be found in Supplemental 2 and Supplemental Fig. 2. The output exposure data have full coverage in space and time and are able to capture the large fire events at a resolution of $1\,km \times 1\,km$ (CV $R^2 = 0.81$ and RMSE $= 1.85\,\mu g/m^3$). Compared to Geng et al. (2018), major improvements include new observation data from the National Park Service to capture $PM_{2.5}$ enhancement near wildfires, allowing for a better representation of high values found during fire events (Supplemental 2 and Supplemental Fig. 2) (Benedict et al., 2017; Martin et al., 2013). Additionally, a random forest approach was utilized instead of the original statistical downscaler for the AOD model. This improved the R^2 of the AOD model from 0.65 to 0.92 and the gap-filled R^2 from 0.66 to 0.81 (Geng et al., 2018). $PM_{2.5}$ exposure values were then aggregated to a $4\,km \times 4\,km$ grid to match the resolution of the health data.

Fire count data were obtained using the MODIS fire count product to specify fire days for each grid cell (NASA, 2018). Wildfire and prescribed fire emissions were obtained from the US EPA emissions inventory for the study period. To calculate the wildfire smoke $PM_{2.5}$ fractions, we used two CMAQ model scenarios–with and without smoke and dust particles. The differences between these scenarios were then divided by the total $PM_{2.5}$ scenario to calculate the smoke $PM_{2.5}$ fractions. The smoke $PM_{2.5}$ fractions were then multiplied by the total satellite-based $PM_{2.5}$ exposure to get the smoke $PM_{2.5}$ concentrations.

2.3. Epidemiological modeling methods

We estimated associations between short-term changes in air quality and ED visits and hospital admissions using a case-crossover study design (Maclure, 1991). Each individual's event day (i.e., date of ED visit or hospitalization) was matched with up to four non-event days, with matching based on grid location, day of week, and calendar month (Levy et al., 2001). Exposure and meteorology were assigned to each event day and corresponding non-event days based on the 4 km × 4 km grid cell in which the patient's address is located. The 4 km grid was chosen a priori through collective agreement between the researchers and the Colorado State Health Department. This resolution was deemed the finest resolution we could use while still conserving confidentiality. We then used conditional logistic regression to estimate the associations between ED visits and hospitalizations for each outcome and exposure to non-smoke $PM_{2.5}$ and smoke $PM_{2.5}$. The final models for respiratory outcomes are shown in model specification 1 & 2 below:

$$\textit{logit } P(Y) = \beta(total_{3day}\ PM_{2.5}) + \beta(temp_{3day}) + ns(doy) \quad (1)$$

$$\textit{logit } P(Y) = \beta(smoke_{3day}\ PM_{2.5}) + \beta(nonsmoke_{3day}\ PM_{2.5}) + \beta(temp_{3day}) + ns(doy) \quad (2)$$

where $total_{3day}\ PM_{2.5}$ represents the 3-day moving average for total $PM_{2.5}$ (i.e., smoke + non-smoke), $temp_{3day}$ is the 3-day moving average temperature, *ns(doy)* is a spline for day of year (two internal nodes per year), $smoke_{3day}\ PM_{2.5}$ represents the three-day moving average smoke $PM_{2.5}$; and $nonsmoke_{3day}\ PM_{2.5}$ denotes three-day moving average $PM_{2.5}$ not related to wildfires. Cardiovascular outcome models were conducted using the same models shown in model specifications 1 and 2, but with 2-day averaging windows. Exposure windows of 3-day average PM for respiratory outcomes and 2-day average PM for cardiovascular outcomes were decided a priori based on published studies and consensus information found in the latest Integrated Science Assessment from the USEPA (Analitis et al., 2012; Delfino et al., 2009; Kunzli et al., 2006; J.C. Liu, 2017; Rappold et al., 2011; Reid, Jerrett et al., 2016; Strickland et al., 2010; USEPA, 2019). Sensitivity analyses were conducted using lag 0, lag 0–1 and seven-day exposure windows for respiratory outcomes and lag 0 and three-day exposure windows for cardiovascular outcomes.

Other potential confounders were assessed (relative humidity, boundary layer height, heat index, wind speed). However, these parameters did not influence the results and were omitted in the final model. Analyses to examine the presence of potential effect modification were completed using sex and age-stratification. Age-stratified categories included children (0–18 years), adults (19–64 years), and older adult (65+ years). We conducted all analyses in R 3.4.3 (2017) and SAS© 9.4.

3. Results

3.1. Exposure modeling and smoke contribution to $PM_{2.5}$ levels

A time series plot for modeled statewide daily mean $PM_{2.5}$ concentrations is shown in Fig. 1. Modeled total $PM_{2.5}$ values ranged from close to 0 to 47.48 μg/m^3, with an overall mean value of 4.67 μg/m^3. The exposure model was also used to separate smoke $PM_{2.5}$ from non-smoke $PM_{2.5}$. This separation is based on the CMAQ fraction, with total $PM_{2.5}$ equal to the sum of non-smoke $PM_{2.5}$ and smoke $PM_{2.5}$. Ratios of smoke $PM_{2.5}$ to total $PM_{2.5}$ ranged from 0 to 99.56% (mean = 0.006%), with smoke $PM_{2.5}$ levels ranging from 0 to 37.34 μg/m^3. The statewide daily mean smoke vs. total $PM_{2.5}$ ratio is also shown for the entire study period (See Fig. 2). As shown, concentrations varied year-to-year and between stations. This is likely due to the spatial variability of wildfires and varied smoke plume behavior due to factors such as prevailing wind speed and direction. To illustrate PM2.5 concentrations and ratios attributable to fire, Fig. 3 shows the domain-wide average total PM2.5 on fire days (smoke PM2.5 > 1%) compared with the domain-wide average ratio of smoke PM2.5. For the entire study period, total PM2.5 averaged 7.87 μg/m3 with average fire PM2.5 ratios at 28%. Fig. 4 shows locations on a fire day near two major fires that occurred during our study period. As shown in Fig. 4A, high levels of smoke PM can be seen despite more moderate total PM2.5 concentrations. Fig. 4B depicts a fire day with much higher total PM2.5 concentrations and the subsequent contributions of smoke PM. Additional analysis showed relatively little correlation between smoke $PM_{2.5}$ and non-smoke $PM_{2.5}$ (Pearson correlation coefficient $r = 0.11$, $p < 0.0001$). The peaks of highest smoke $PM_{2.5}$ ratios tended to correspond with active fire days. Fig. 5 illustrates the modeled total $PM_{2.5}$ and smoke $PM_{2.5}$ ratio for June 22, 2013, a peak fire day during the West Fork Fire Complex. As depicted, when compared to satellite imaging, the modeled smoke $PM_{2.5}$ appears to capture the apparent visible smoke plume adequately.

3.2. Epidemiological modeling

After excluding duplicate events and events with non-geocoded addresses, 44,262 of 490,368 (9%) of cases were excluded from the analysis. A total of 446,106 ED visit and hospitalization events were analyzed from the Colorado Department of Public Health and Environment. Of those included, there were 204,823 male and 241,283 female cases. The lowest case count occurred in 2011 (n = 102,318), with the highest number of cases in 2014 (n = 129,477). While many reasons could exist, the large increase seen in 2014 could be explained by changes in health seeking behavior due to wider Medicaid coverage resulting from the implementation of the Affordable Care Act (Singer et al., 2019). Other summary statistics on age groups and events per year are found in Table 1.

Using conditional logistic regression models, we estimated the odds ratio for exposure to smoke $PM_{2.5}$ and individual health outcomes. As shown in Fig. 6 and Supplemental Table 1, we observed significant positive associations between 1 μg/m^3 increases in 3-day moving average fire exposures and both asthma (OR 1.081, 95% CI (1.058, 1.105)) and combined respiratory disease (OR 1.021, 95% CI (1.012, 1.031)) in a model that adjusted for $PM_{2.5}$ from other sources. There were no significant positive associations linked to cardiovascular outcomes and 2-day smoke $PM_{2.5}$ exposures (see Fig. 7 and Supplemental Table 2). However, some inverse associations were shown to be protective for cardiovascular outcomes. This could possibly be due to random error, or it may be that individuals with pre-existing cardiovascular disease stay indoors on days with fire activity.

The models were also run using total $PM_{2.5}$ for both cardiovascular and respiratory outcomes. Overall, the majority of the respiratory odds ratios for 3-day average total $PM_{2.5}$ were either null or trending to positive (Supplemental Table 3).The odds ratios for ischemic heart disease, acute myocardial infarction, and dysrhythmia also suggest a trend toward a positive association (see Supplemental Table 4). The cardiovascular results for total $PM_{2.5}$ included significant negative results for congestive heart failure, peripheral/cerebrovascular disease, and cardiovascular disease.

We conducted sensitivity analyses for additional exposure windows. Using lag 0 for both respiratory and cardiovascular outcomes, similar

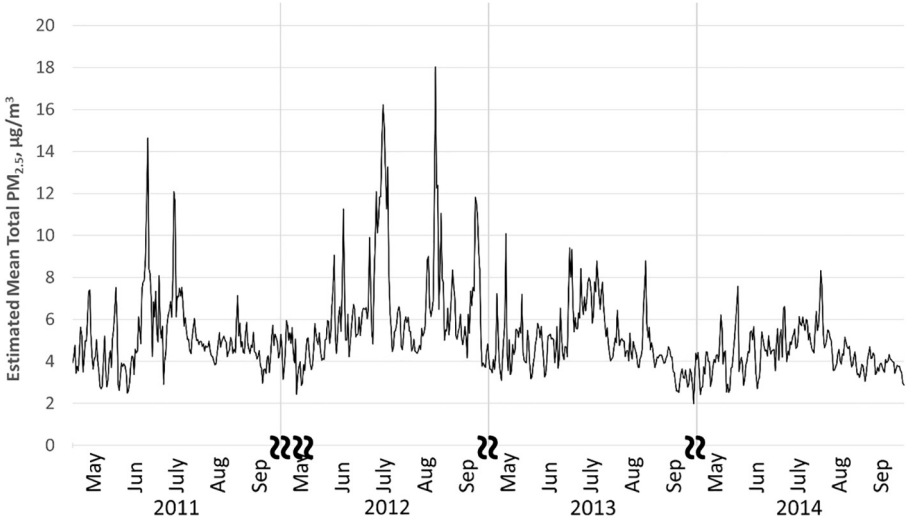

Fig. 1. Daily mean modeled $PM_{2.5}$ from for fire seasons 2011–2014 in Colorado. State-averaged time series data for fire seasons (May–August) 2011–2014 show total modeled $PM_{2.5}$ levels by day, month, and year.

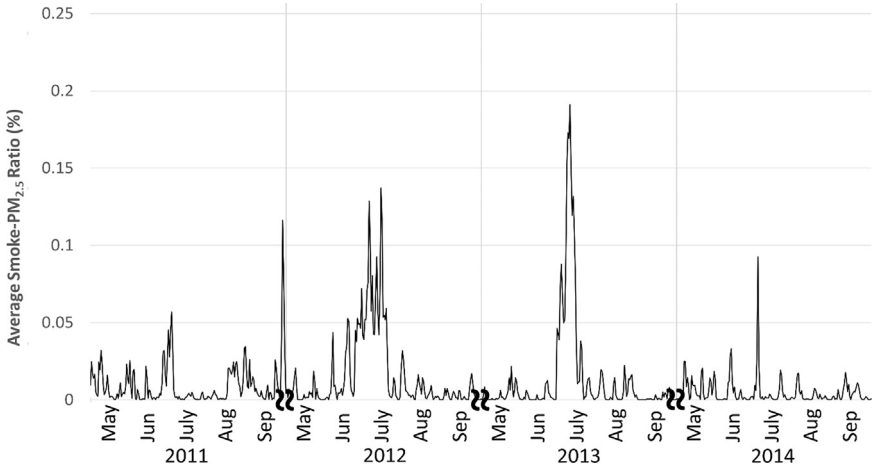

Fig. 2. Daily mean ratio of $PM_{2.5}$ attributed to wildfire. State-averaged time series data for fire seasons (May–August) 2011–2014 depicting ratio of modeled smoke $PM_{2.5}$ to total modeled $PM_{2.5}$.

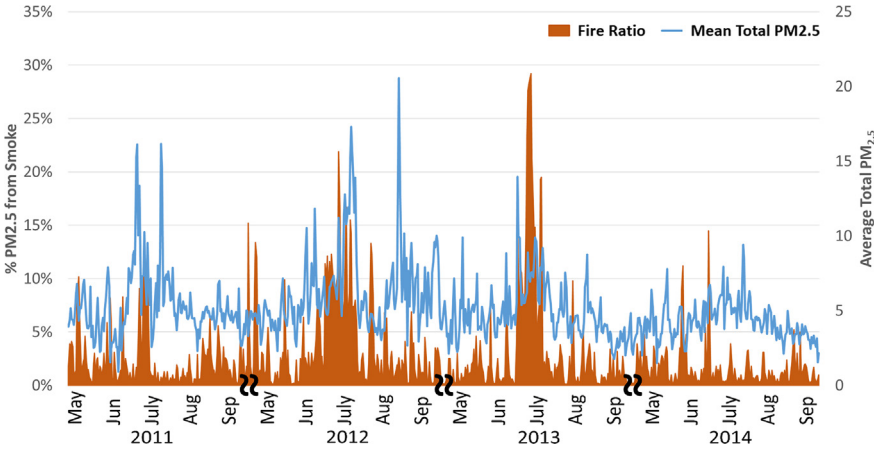

Fig. 3. Domain-wide daily mean total $PM_{2.5}$ and mean ratio of $PM_{2.5}$ on fire days (fire PM > 1%). Time series depicting both total and ratio of modeled smoke $PM_{2.5}$ to total modeled $PM_{2.5}$.

results were seen with smoke $PM_{2.5}$ exposure, with notable differences in overall upper respiratory infection (OR 1.015, 95% CI (1.005, 1.026)) and upper respiratory infection in children (OR 1.018, 95% CI (1.004, 1.003), see Supplemental Figs. 3 and 4). Using lag 0–1 for all respiratory outcomes, the results were again similar to the initial analysis with changes for overall and child-only upper respiratory infections; see Supplemental Fig. 5. Using a 7-day averaging window for respiratory outcomes, asthma was the only outcome to have a

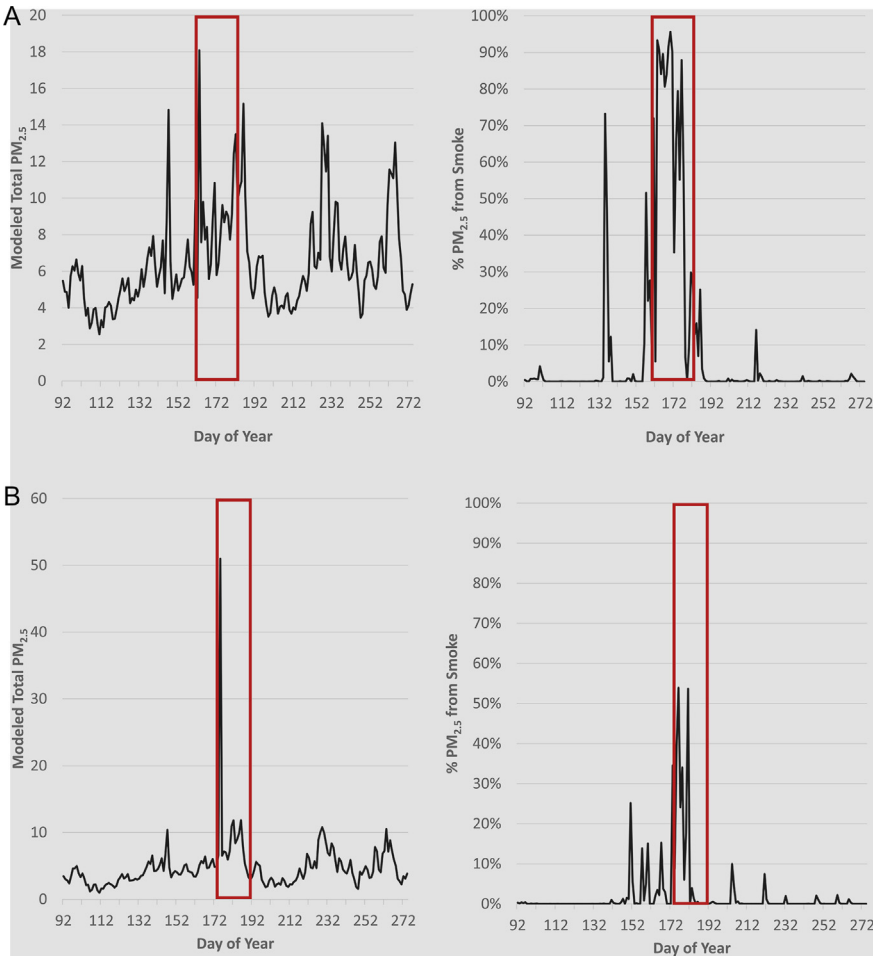

Fig. 4. Daily mean total $PM_{2.5}$ and mean ratio of $PM_{2.5}$ attributed to wildfire at two locations. Time series depicting both total and ratio of modeled smoke $PM_{2.5}$ to total modeled $PM_{2.5}$. A) Location near the High Park Fire (June 9–30, 2012) and B) Location near Waldo Canyon Fire (June 23–July 10, 2012). Red boxes indicate active fire days. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

significant positive association with smoke $PM_{2.5}$ exposure (OR 1.081, 95% CI (1.051, 1.112), see Supplemental Table 5). The associations for asthma, upper respiratory infection, bronchitis, and combined respiratory disease trended positive but not significant for 7-day averaged total $PM_{2.5}$ exposure (see Supplemental Table 6). A 3-day averaging window used for cardiovascular outcomes also yielded either null or negative results (Supplemental Tables 7 and 8).

3.3. Stratified analysis

To investigate potential effect modification of the relationship between exposures and respiratory outcomes, we conducted stratified analyses based on sex and age. While most sex-stratified total $PM_{2.5}$ results were null, an association was seen in females for bronchitis (OR 1.007, 95% CI (1.001, 1.013), see Supplemental Table 9), however, no significant results were observed for cardiovascular outcomes and both 2-day total and smoke $PM_{2.5}$ (Supplemental Tables 10 and 11). Associations for both female and male asthma cases and 3-day average smoke $PM_{2.5}$ were significant, with higher odds shown in female cases (OR 1.096, 95% CI (1.064, 1.128)) than in male cases (OR 1.063, 95% CI (1.029, 1.098)). Female bronchitis cases (OR 1.054, 95% CI (1.010, 1.101)) and female total respiratory cases (OR 1.027, 95% CI (1.015, 1.040)) were also positively associated with smoke $PM_{2.5}$. Additional sex-stratified, 3-day average smoke $PM_{2.5}$ results can be found in Supplemental Table 12.

Additionally, some outcomes exhibited differences when stratified on age. After age-stratification, there were no patterns found linking respiratory outcomes and total $PM_{2.5}$ with any specific age group (Supplemental Table 13). Regarding smoke $PM_{2.5}$, Fig. 6 also depicts the ORs and associated confidence intervals for each of the respiratory outcomes by age group. In children ages 0 to 18 years, significant positive associations were seen for asthma (OR 1.075, 95% CI (1.035, 1.116)). Adults aged 19 to 64 years of age exhibited positive associations for asthma (OR 1.091, 95% CI (1.060, 1.122)), bronchitis (OR 1.044, 95% CI (1.005, 1.085)), COPD (OR 1.056, 95% CI (1.015, 1.100)), and combined respiratory disease (OR 1.030, 95% CI (1.017, 1.044)) (see also Supplemental Table 14). For individuals 65 and older, there were no significant positive associations seen for respiratory outcomes. We found no positive associations for age-stratified total or smoke $PM_{2.5}$ and any of the cardiovascular outcomes (See Fig. 7 and Supplemental Tables 15 and 16). Additional results for stratification analyses using a 7-day averaging window for respiratory outcomes and a 3-day averaging window for cardiovascular outcomes can be found in Supplemental Tables 17–24. Of note, associations for both childhood and adult asthma, adult COPD, and adult combined respiratory disease events were positively associated with 7-day average smoke $PM_{2.5}$ (see Supplemental Table 17).

4. Discussion

In this study, we estimated associations between various health outcomes and acute exposure to non-smoke $PM_{2.5}$ and smoke $PM_{2.5}$ in the state of Colorado over a four-year period (2011–2014). The design of this study is centered on smoke $PM_{2.5}$ contributions to health

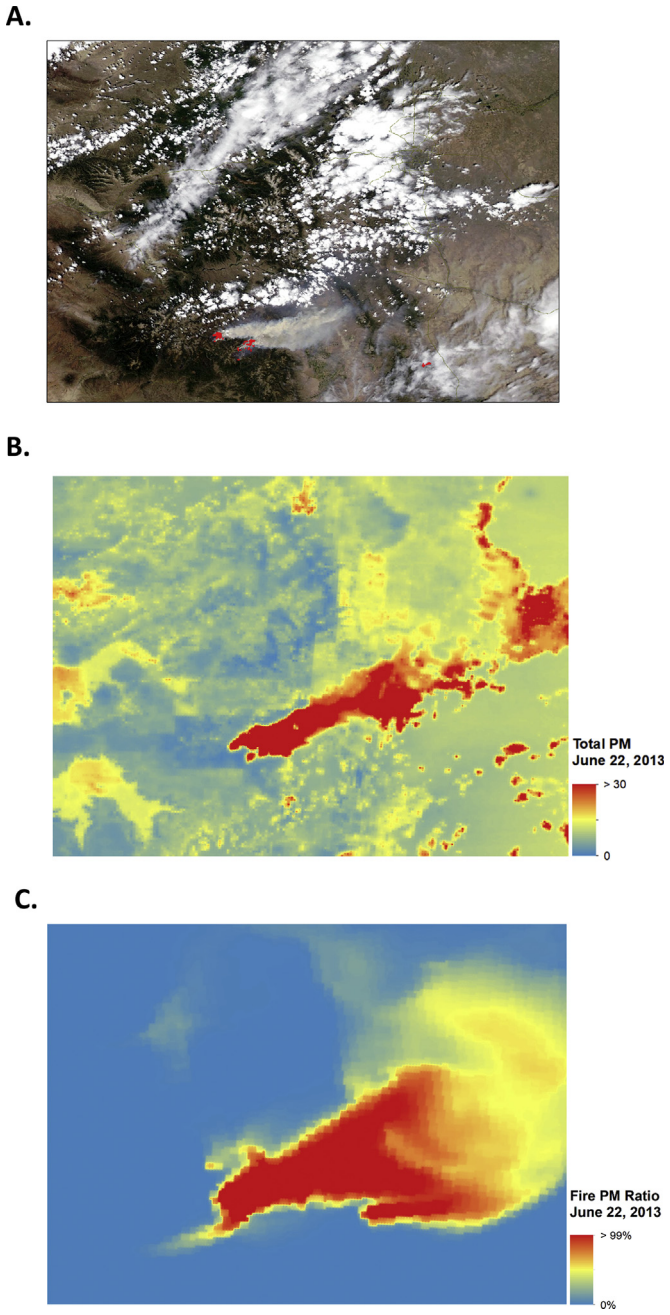

Fig. 5. Satellite smoke plume, modeled total $PM_{2.5}$ and smoke $PM_{2.5}$ for west fork fire complex, June 22, 2013. Modeled data corresponds to visible smoke plume as shown in A-C. A) Satellite image from June 22, 2013 with active West Fork Complex Fire (NASA, 2013). B) Total $PM_{2.5}$ for Colorado on June 22, 2013. C) Amount of $PM_{2.5}$ attributed to fire on June 22, 2013.

outcomes. This work builds on our previous work by improving exposure data metrics and expanding from a 1-month pilot study (Alman et al., 2016). The exposure data considers both spatial and temporal variability by including the use of satellite data to enhance the exposure estimates on an improved spatial scale of 4 km × 4 km. Another unique aspect of our exposure assessment is that we were able to separate smoke $PM_{2.5}$ from non-smoke sources and estimate risks attributable to wildfire smoke distinct from those due to $PM_{2.5}$ exposures from other sources.

As we hypothesized, many of the respiratory disease outcomes increased during periods of wildfire activity. For respiratory outcomes, we estimated an increase (OR = 1.036 (95% CI: 1.022, 1.050%)) in ED/hospitalizations per 1 μg/m^3 increase in fire smoke $PM_{2.5}$ exposure. The magnitude of the association was largest for asthma (OR = 1.081 (95% CI: 1.058, 1.105)). Additionally, we observed heterogeneity in the association estimates when stratifying by age group. Positive associations were observed for asthma events, where ED/hospitalizations increased significantly in children (OR = 1.075 (95% CI: 1.035, 1.116)) and in adults (OR = 1.091 (95% CI: 1.060, 1.122)) whereas the association estimate was lower in magnitude and was less precise for older adults (OR = 1.009 (95% CI: 0.920, 1.106)). Similarly, an increase was seen for combined respiratory diseases with increases in ED/hospitalizations and adults (OR = 1.030 (95% CI: 1.017, 1.044)). Specifically, in the adult group, increases were also shown for both bronchitis (OR = 1.044 (95% CI: 1.005, 1.085)) and COPD (OR = 1.056 (95% CI: 1.015, 1.100)). As opposed to other studies, there was no association shown for respiratory diseases when stratified for the older adult age group.

Table 1
Epidemiologic data descriptive statistics.

	Case count
Total records	490,368
Geocoded addresses	446,106
Non-geocoded addresses	44,262
Year of event	
2011	102,318
2012	102,574
2013	111,737
2014	129,477
Age ranges	
0–18 y	94,022
19–64 y	202,665
65+ y	149,419
Sex	
Female	241,282
Male	204,823

Unlike respiratory outcomes, we did not see a strong link between smoke $PM_{2.5}$ and cardiovascular outcomes. Results for combined cardiovascular disease yielded null results (OR = 0.998 (95% CI: 0.984, 1.011)). Similar results were shown for both the adult and older adult age groups. This is not wholly surprising given differing results in current literature regarding the links between cardiovascular outcomes and wildfire events. There are fewer examples of cardiovascular associations with wildfire smoke exposure compared to respiratory outcomes. Additionally, associations with cardiovascular outcomes tended to be substantially lower in magnitude than for the respiratory outcomes. These differences are consistent with published studies on both types of outcomes (Cascio, 2018; Deflorio-Barker et al., 2019; Dennekamp et al., 2011; Dennekamp et al., 2015; Johnston et al., 2014; Liu et al., 2015; Reid, Brauer et al., 2016; Wettstein et al., 2018). For example, in Deflorio-Barker et al. (2019), most cardiovascular outcomes were not significant with fire day PM2.5 using lag0–2. They also found similar results for smoke day all-cause cardiovascular outcomes were very similar to non-smoke days (OR 1.06 for smoke days vs OR 1.07 for non-smoke days (Deflorio-Barker et al., 2019)).

Our high-resolution epidemiological study furthers the current knowledge in the field by incorporating random forest modeling methods combining information from MAIAC AOD, CMAQ simulations, and ground measurements to elucidate the portion of PM2.5 present in the air due to wildfire smoke. Previous work has been done to enhance the spatial coverage and resolution of total PM2.5 estimates during wildfire events (Reid et al., 2019). While most work compared smoke and non-smoke days using various fire indicators, our study particularly focuses on the separation of smoke PM2.5 from other sources. In most work, researchers compared smoke and non-smoke days using a variety of methods different from our study (Reid et al., 2019; Reid, Jerrett et al., 2016). For example, satellite measurements are increasingly used to augment the spatially sparse ground monitoring for PM. However,

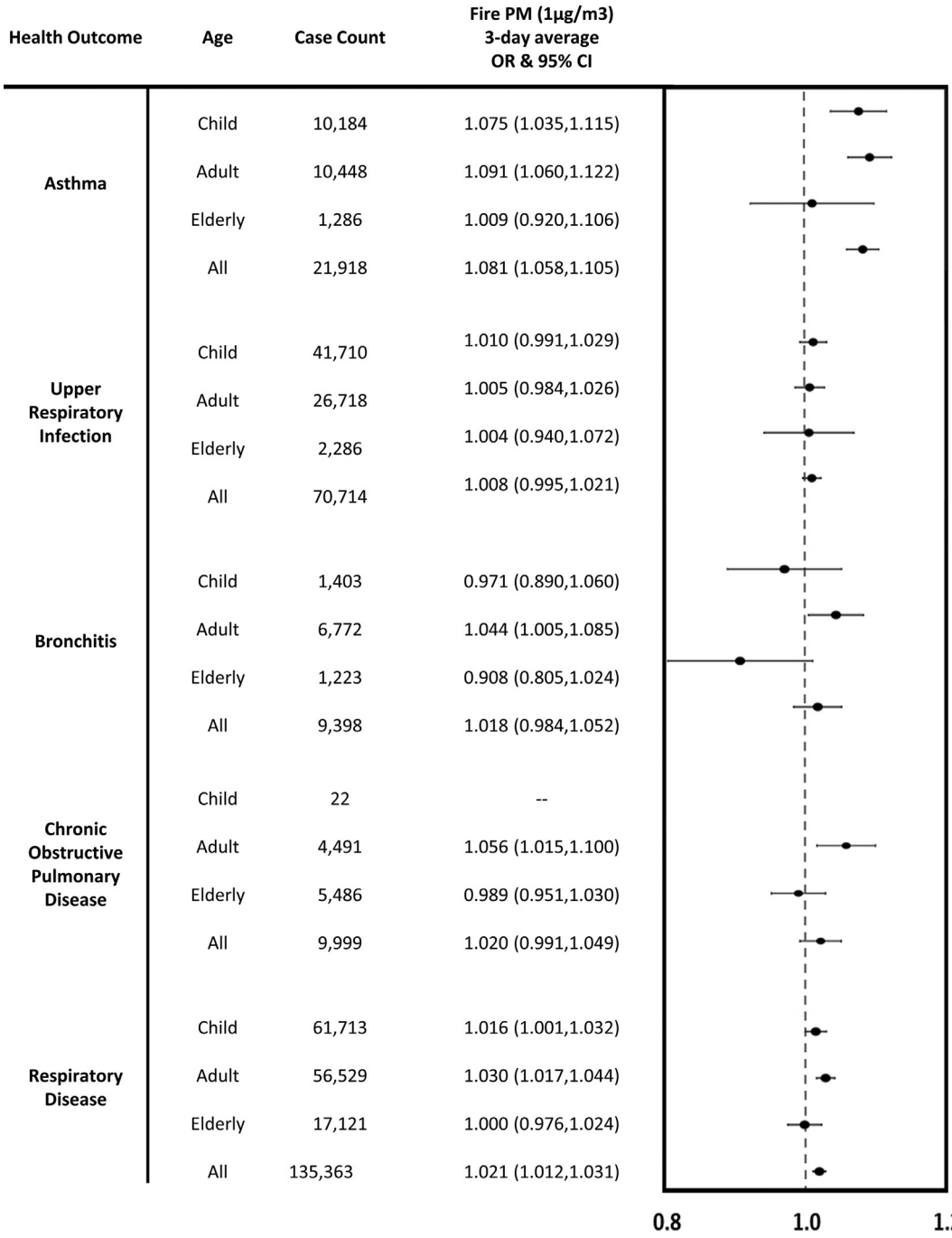

Fig. 6. Wildfire smoke $PM_{2.5}$ exposure and respiratory outcomes. Odds ratios for both total and age-stratified respiratory outcomes per 1 µg/m^3 increase in wildfire smoke $PM_{2.5}$ exposure, arranged by outcome and age group.

this remains a relatively new approach to capturing the smoke PM concentrations. A study by Liu et al. looked at the entire Western US at the county-level using combined satellite and ground data (J.C. Liu, 2017). They defined a fire indicator variable, or "smoke wave," which includes periods of at least two days of high pollution from wildfire smoke. Using this method, Liu et al. found associations between wildfire smoke exposure and various respiratory illnesses, but no associations with cardiovascular outcomes. Reid et al. (2015) used a machine learning approach to integrate multiple data sources including smoke indicators such as the distance to the nearest fire cluster and a smoke intensity calculation. The use of more advanced methods for predicting $PM_{2.5}$ exposure enhanced the exposure estimations, however, the $PM_{2.5}$ concentrations were not separated into smoke and non-smoke concentrations (Reid et al., 2015).

Other work has utilized methods combining wildfire emissions and smoke plume modeling. For example, Hutchinson et al. examined similar epidemiological questions using exposure data derived from a model that combined the Wildland Fire Emissions Information System and the Hybrid Single-Particle Lagrangian Integrated Trajectories (Hutchinson et al., 2018). Their study found increases in respiratory events with null cardiovascular results. However, the methods denoted fire-specific emissions due to fire location and progression from modeled progression maps and may not capture exposures as well as the use of chemical transport models. Ultimately, while our results carry similar interpretations to both studies, subtle dissimilarities may be seen as we utilize different air quality evaluation products and higher-

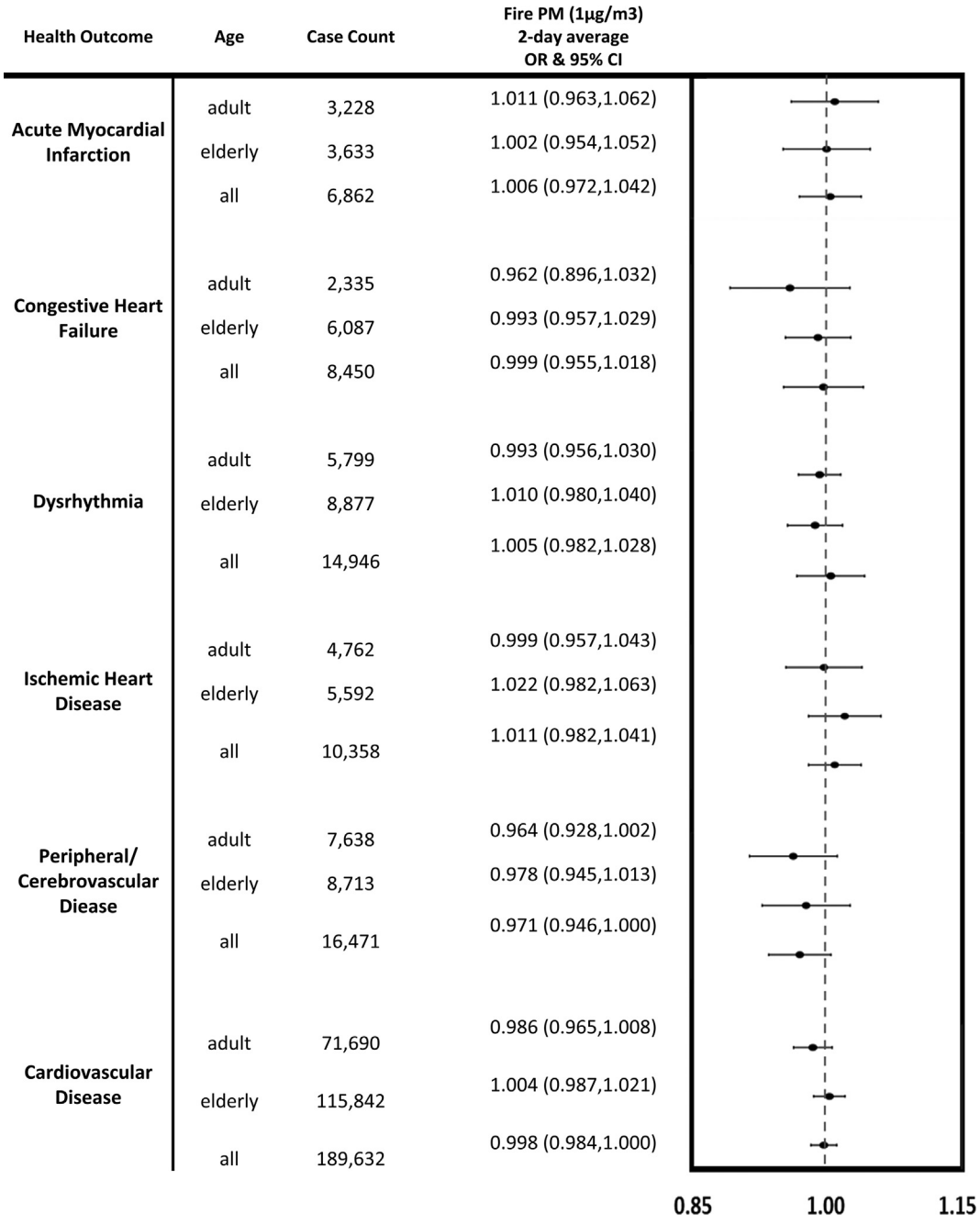

Fig. 7. Wildfire smoke $PM_{2.5}$ exposure and cardiovascular outcomes. Odds ratios for both total and age-stratified cardiovascular outcomes per 1 µg/m^3 increase in wildfire smoke $PM_{2.5}$ exposure, arranged by outcome and age group.

resolution meteorological and epidemiological data to better-define the local exposures for each event.

The asthma association found in our study is substantially larger than those shown in previous publications. In addition to Reid, Brauer et al. (2016), other studies found significant associations between smoke PM and health outcomes. Delfino et al. reported significant associations of OR = 1.043 between asthma and 2-day moving average smoke exposure for 10 µg/m3 increase in total PM2.5 concentration (Delfino et al., 2009). In a more recent study, Reid et al. also found a significant association for asthma and previous 2-day moving average smoke exposure, with an OR of 1.050 during fire events for a 10 µg/m3 increase in PM2.5 (Reid et al., 2019). Factoring in the domain-wide average smoke PM2.5 ratio for the study period (~28% for days with > 1% smoke PM), our result per 1 µg/m3 roughly translates to 1.08 per 4 µg/m3 of total PM2.5. This converted result is more aligned with previously reported values, and the larger effect estimate is likely due to improved exposure assessment. It is also important to remember that our methods are unlike the majority of previous literature. Namely, the general approach in previous studies is to model smoke exposure using smoke day indicators. Our approach differed in that we sought to isolate the actual concentration of PM2.5 directly from smoke. We originally hypothesized that there may be a difference in toxicity of smoke PM2.5 compared to non-smoke PM2.5. When compared with other literature, our findings suggest that smoke PM2.5 may actually be more damaging to human health. Aside from asthma outcomes, the majority of the health associations in this study fall in line with those found in previous literature. For example, Deflorio-Barker et al. (2019) also demonstrated stronger associations with respiratory outcomes than those with cardiovascular disease; with asthma exhibiting the largest OR of 1.06 (Deflorio-Barker et al., 2019).

While we did not investigate physiological mechanisms, these results may be explained by the toxicity of smoke $PM_{2.5}$. Since different chemical compositions of $PM_{2.5}$ may affect the body differently, it has been suggested that toxicological differences may play a role in how wildfire smoke PM affects the human anatomy and physiology. Multiple toxicological studies have shown differences in the composition and effects of wildfire smoke compared to ambient air (Franzi et al., 2011; Kim et al., 2018; Wegesser et al., 2010; Wegesser et al., 2009; Wong et al., 2011). It has been shown that the small particles found in wildfire smoke may be responsible for stimulation of mechanisms that lead to increased oxidative stress at the cellular level. Wegesser et al. (2009) observed significant changes in macrophage and neutrophil counts in mouse lung samples exposed to wildfire smoke PM compared to ambient air. An additional study by the same group, expanded on these findings to show that substances such as polycyclic aromatic hydrocarbons (PAH) can be present in much higher concentrations in smoke versus levels detected in ambient air (Wegesser et al., 2010). Franzi et al. (2011) looked specifically at the inflammatory responses due to wildfire smoke PM exposure. PM from wildfire smoke exhibited approximately five times more toxicity to lung macrophages than non-smoke exposure. This study also showed significant changes in reactive oxygen species and subsequent oxidative stress, leading to higher cell degeneration and potential apoptosis. Similarly, Kim et al. (2018) found significant increases in mouse lung neutrophils after exposure and that levels of lung toxicity were significantly associated with fuel type (Kim et al., 2018).

Despite the strengths of our study, some limitations remain. While we sought to enhance the exposure estimates for individual cases, some exposure misclassification is still possible given the assumption that the location of a person's address is a good representation of their short-term exposures to smoke PM. An additional limitation exists due to the use of modeled exposure data. However, as stated previously and despite this uncertainty, the model accurately captures the temporal and spatial trends of $PM_{2.5}$ measured by ground monitors and, thus give an accurate representation of overall trends. Additionally, several health events were left out of the analysis due to issues with address geocoding or non-Colorado residency. However, the exclusions were relatively small with only 9% of cases not used in the final analyses. Additionally, our analyses lacked the ability to differentiate chemical compositions of $PM_{2.5}$. Thus, we cannot link toxicological effects to our exposure metrics. Finally, the selection of averaging window size, though based on current literature, may also introduce error into the analysis.

Notwithstanding these limitations, our methods lend insight into important challenges that remain in the wildfire smoke exposure and health effects literature. The use of higher resolution enhanced exposure data provides a new approach to assigning exposure to individual events. Using multiple data products, our method aids in distinguishing wildfire smoke $PM_{2.5}$ from background $PM_{2.5}$. Unlike ground monitors that provide spatially sparse measurements, the exposure model used here provides daily concentrations for each 4 km × 4 km grid cell in our epidemiological study.

5. Conclusions

Supported by high-resolution $PM_{2.5}$ exposure estimates, we found significant associations between wildfire smoke and acute respiratory outcomes in Colorado, despite an absence of association with total $PM_{2.5}$ concentrations. Our findings point to potential toxic differences between smoke and non-smoke $PM_{2.5}$ exposure; suggesting that $PM_{2.5}$ from wildfire smoke could pose a significant threat to public health. This is especially true given the expected climate change-related impacts on wildfire incidence. It is, therefore, important to derive more accurate concentration-response relationships specific to wildfire smoke in order to develop a better understanding of future potential health risks based on increased wildfire activity. Taken together, the current analysis can inform public health agencies and healthcare systems regarding the potential future burden of wildfire smoke $PM_{2.5}$ exposure within the context of climate change. This information may be a key element in evaluating and enhancing current preparations aimed at wildfire-event response readiness.

Declaration of competing interest

The authors declare they have no actual or potential competing financial interests.

Acknowledgments

The authors would like to thank Kirk Bol and the Colorado Department of Public Health and Environment for providing the health data used in this analysis.

Funding sources

The work of J. Stowell, G. Geng, Y. Liu, and H. Chang was partially supported by the NASA Applied Sciences Program (grant NNX16AQ28G, PI: Y. Liu). This publication was developed under assistance agreement no. 83586901 awarded by the U.S. Environmental Protection Agency (PI: Y. Liu). It has not been formally reviewed by EPA. The views expressed in this document are solely those of the authors and do not necessarily reflect those of the Agency. EPA does not endorse any products or commercial services mentioned in this publication.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2019.105151.

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Seeking natural capital projects: Forest fires, haze, and early-life exposure in Indonesia

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Edited by S. Polasky, University of Minnesota, St. Paul, MN, and approved January 15, 2019 (received for review March 26, 2018)

Natural capital will be depleted rapidly and excessively if the long-term, offsite impacts of depletion are ignored. By examining the case of tropical forest burning, we illustrate such myopia: Pursuit of short-term economic gains results in air pollution that causes long-term, irreversible health impacts. We integrate longitudinal data on prenatal exposure to the 1997 Indonesian forest fires with child nutritional outcomes and find that mean exposure to air pollution during the prenatal stage is associated with a half-SD decrease in height-for-age *z* score at age 17, which is robust to several statistical checks. Because adult height is associated with income, this implies a loss of 4% of average monthly wages for approximately one million Indonesian workers born during this period. To put these human capital losses in the context of policy making, we conduct social cost–benefit analyses of oil palm plantations under different scenarios for clearing land and controlling fires. We find that clearing for oil palm plantations using mechanical methods generates higher social net benefits compared with clearing using fires. Oil palm producers, however, would be unwilling to bear the higher private costs of mechanical clearing. Therefore, we need more effective fire bans, fire suppression, and moratoriums on oil palm in Indonesia to protect natural and human capital, and increase social welfare.

sustainable development | environmental health | oil palm | cost–benefit analysis | health irreversibility

Economists will argue that natural capital has been depleted rapidly and excessively because the offsite lagged impacts of depletion are either ignored or remain unmeasured (1–4). We use the case of Indonesia to illustrate the extent of such oversight. Despite its vast tropical forests, forest loss is rapid because forests are burned to clear land cheaply and plant lucrative job-friendly export crops such as oil palm (5, 6). Unfortunately, such economic development is unsustainable because we ignore externalities of forest fires—air pollution and biodiversity loss, chief among others (4, 7). For example, forest fires in Indonesia, started to establish estate crops, burned out of control due to the El Niño-induced abnormally dry weather in 1997. These fires destroyed habitat (around 11 million hectares of forests), compromised hydrological services, and generated health-damaging air pollution (around 25% of global carbon emissions from fossil fuels came from this single event) (8–11). Despite the severity of this event, we do not know the full extent of health damages, especially the irreversible, offsite lagged human capital impacts. In addition, we do not know whether the costs (in terms of jobs and profits) of policies to avoid the haze will outweigh the social benefits (12).

In the post-2015 sustainable development goals (SDGs) era, debates rage in the global community about how best to protect natural capital, promote health, mitigate climate change, and reduce poverty (13, 14). The main question posed in our paper—do the economic benefits of avoiding health damages of haze from forest fires outweigh the economic costs of alternative policies—is relevant to these debates. Specifically, our research illustrates why SDGs should focus on reducing negative externalities, promoting intergenerational equity, and improving capabilities (15). We focus on the 1997 forest fires in Indonesia and the associated haze. The 1997 fires were one of the largest in recent history, but unfortunately, such forest fires have become even more frequent lately, including a round of devastating forest fires in 2015. Despite their magnitude and frequency, we know surprisingly little about the full social costs of these fires. While there is evidence on short-run health damages of air pollution, little is known about the long-term and intergenerational costs of early-life exposure to air pollution.

Most studies of early-life exposure to air pollution are conducted in high- or middle-income countries and focus on immediate birth outcomes (16, 17). From past analyses, there is strong evidence showing that early-life exposure to air pollutants is associated with low birthweight and preterm birth (16, 17). The suspected pathways from air pollutants to birth outcomes are inflammation and direct toxic effects to the placenta and fetus, oxygen supply to the fetus, and DNA expression (17). With respect to longer-term outcomes, the literature on the "fetal origins" hypothesis suggests that intrauterine health insults can cause lasting and irreversible damage to cardiovascular and respiratory health and that low birthweight is associated with shorter height in adulthood (18–21). Still, there are very few studies that specifically make the connection between environmental exposure to air pollutants at early-life and long-term outcomes (22).

Demonstrating that a short-term episode of extremely high air pollution has long-term health impacts is salient to many low- and middle-income countries, especially as many parts of Asia face frequent "airpocalypses" in recent years. In our study setting, the 24-h total suspended particulate (TSP) concentration reached as high as 4,000 $\mu g/m^3$ during multiple days in October 1997 in parts of Sumatra—the World Health Organization (WHO) recommended 24-h maximum for TSPs is 120 $\mu g/m^3$ (23). More recently, New Delhi experienced 24-h $PM_{2.5}$ levels of 700–1,200 $\mu g/m^3$ in November 2016—the WHO daily guideline is 25 $\mu g/m^3$. Also in late 2016, multiple cities in northern China attained their highest level of air pollution warning. Because earlier studies were mainly conducted in rich countries that experience very different exposure profiles, economic development, and environmental institutions, there is little evidence to guide policy makers and practitioners in low- and middle-income contexts.

This paper results from the Arthur M. Sackler Colloquium of the National Academy of Sciences, "Economics, Environment, and Sustainable Development," held January 17–18, 2018, at the Arnold and Mabel Beckman Center of the National Academies of Sciences and Engineering in Irvine, CA. The complete program and video recordings of most presentations are available on the NAS website at www.nasonline.org/economics-environment-and.

Author contributions: J.-S.T.-S. and S.K.P. designed research; J.-S.T.-S. and S.K.P. performed research; J.-S.T.-S. analyzed data; and J.-S.T.-S. and S.K.P. wrote the paper.

The authors declare no conflict of interest.

This article is a PNAS Direct Submission.

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This article contains supporting information online at www.pnas.org/lookup/suppl/doi:10.1073/pnas.1802876116/-/DCSupplemental.

Published online February 19, 2019.

It is not enough to stop at estimating environmental health damages, however, without considering how they stack up against the costs (and other benefits) of policies to control fires. The political economy of policies to control forest fires in Indonesia is complex and covers the entire gamut from government regulation to market incentives to voluntary control by firms and landowners (24–27). Currently, key policies include blanket bans on fires for clearing land and moratoriums on expanding oil palm (28, 29). Because oil palm is extremely lucrative in terms of foreign exchange revenue (e.g., it accounted for US$18.6 billion, or about 10% of Indonesia's total exports in 2015) and jobs (e.g., sector employs around 4.5 million workers), such moratoriums may be politically impractical and unsustainable. Moreover, firms will not comply with bans and moratoriums if enforcement is not strict (30, 31). Hence, we must consider costs and benefits of policies to detect and suppress forest fires before they spread, and to enforce bans and moratoriums (32). Such cost–benefit analyses (CBAs) can identify the parameters driving the maximizations of social welfare, given the health benefits of reduced fires and haze (7). All of these options are complicated by the fact that fire severity depends on the location, and the costs and benefits vary by stakeholders (33).

Study Rationale and Design

First, it is difficult to know the extent of irreversible and offsite lagged human capital impacts because high-quality longitudinal data that faithfully depict long-term impacts have been difficult to find in environmentally vulnerable and economically poor locations (7, 34). Thus, we carefully integrate longitudinal data on prenatal exposure to the pollution from the 1997 Indonesian forest fires with data on nutritional outcomes, genetic inheritance, climatic factors, household inputs, and various sociodemographic factors. Second, methodologically flexible approaches are needed because of the interdisciplinary nature of issues in sustainable development and planetary health (4). We show how rigorous statistical analyses and numerous robustness checks can be applied to a multisectoral panel dataset to control for potential sources of confounding the correlation of prenatal haze exposure with adult height. While previous research has drawn attention to the deaths caused by the forest fires, we show that survivors also suffer large and irreversible losses of human capital because of haze exposure (35, 36). Third, scientists are often unwilling (because of norms or incentives) to provide approximate, if imperfect, practical advice that policy makers seek, preferring to provide precise answers to questions, even if these questions are irrelevant or untimely for policy (37). To avoid such "type III" errors and to put such human capital losses in context, we conduct a CBA of various policies to avoid fires in oil palm plantations (38–40). Specifically, we combine our haze-height results with other costs and benefits, and also account for the heterogeneity and uncertainty inherent in the model parameters.

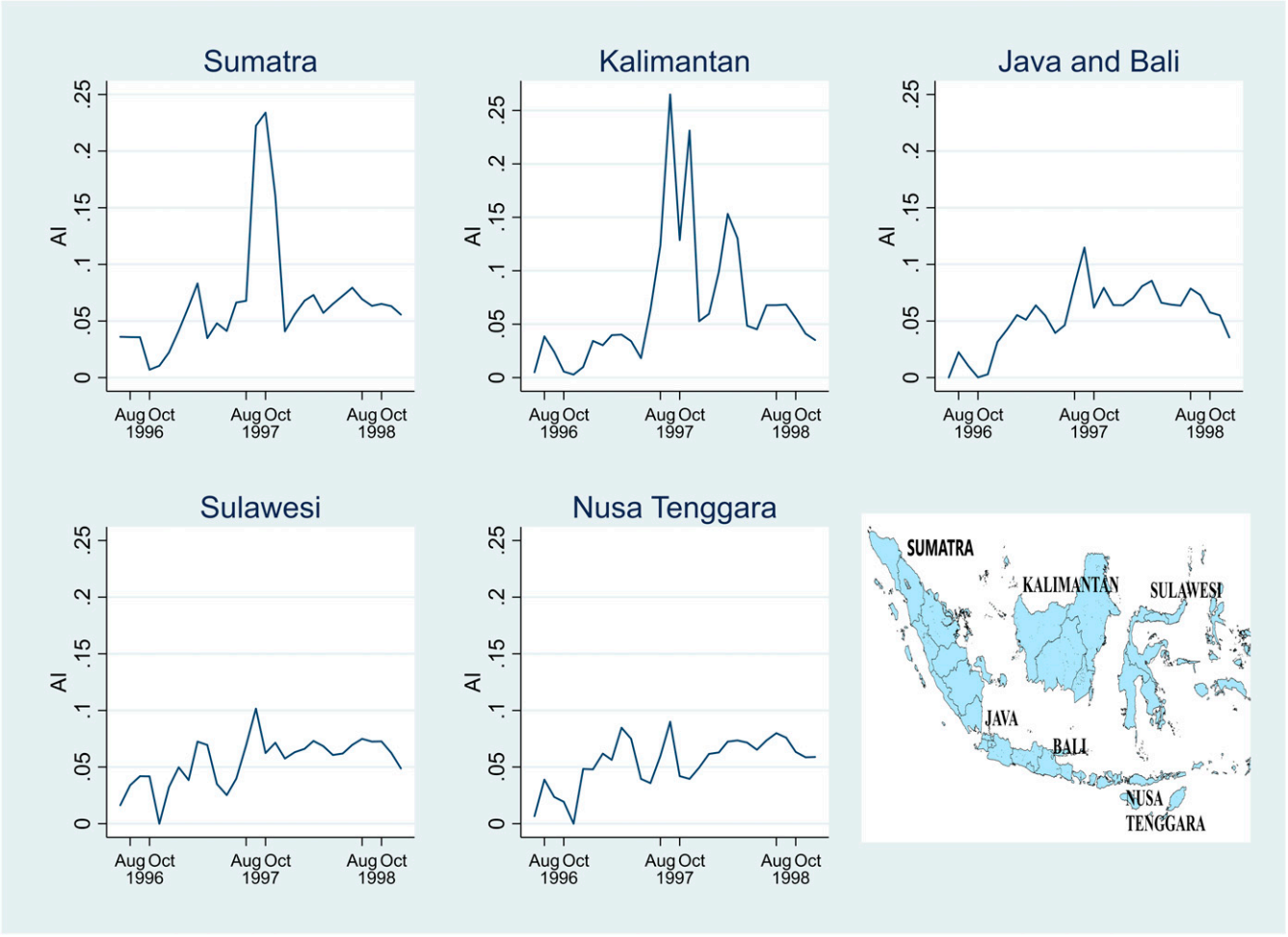

Fig. 1. Spatial and temporal distribution of air quality across Indonesia.

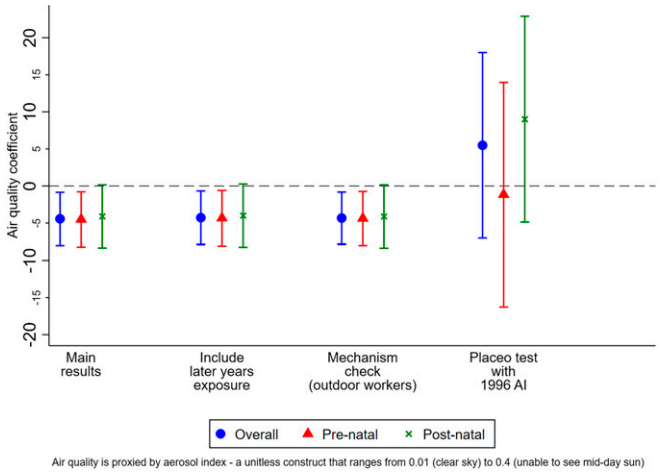

Fig. 2. Impact of early-life air pollution on HAZ for different regression specifications.

Results

Our data show that the average 1997 air pollution exposure [aerosol index (AI)] is 0.1 (where 0.01 represents crystal-clear sky with maximum visibility and 0.4 represents trouble seeing the midday sun). As shown in Fig. 1, however, AI varies significantly in space and time, with exposure ranging from 0 (18% of sample, from Sulawesi and Nusa Tenggara) to 0.3 (18% of sample, from Kalimantan and Sumatra). Average height-for-age *z* score (HAZ) is negative, suggesting that Indonesian children are shorter than the reference group from the United Kingdom. The other environmental, household, and parental variables are summarized in *SI Appendix*, Table S1.

Fig. 2 shows effects of AI, including the 95% confidence interval, on child's HAZ 17 y after exposure [i.e., fourth round of the Indonesian Family and Life Survey (IFLS)]. The first, second, and third coefficient in each set of results represent the overall, prenatal, and postnatal impact, respectively, of early-life exposure (*Materials and Methods*). Results from the main specification show that the mean level of exposure (i.e., AI = 0.1) translates to a 0.41 decrease in HAZ (or about 3.4 cm, equivalently) by 2014. The full results are also presented in *SI Appendix*, Table S2. These results show that the decrease in HAZ is statistically consistent across earlier waves of the IFLS. In other words, children in our analysis experienced a decrease in height from 3 y of age, and this impact persisted through the age of 17.

We further examined whether this early-life effect was attributable to prenatal or postnatal exposure. When exposed during the prenatal stage, the impact on HAZ is essentially unchanged—the average effect is 0.43. In contrast, while the coefficient for postnatal exposure is negative, it is statistically insignificant. Thus, the relationship we detect appears to be completely driven by prenatal exposure.

To ensure our results are not driven by confounders and spurious correlations, we undertake a series of robustness checks (*Materials and Methods*). We confirm that our findings are not driven by (*i*) high levels of pollution in later years, (*ii*) an indirect effect of severe air pollution on a family's ability to work and to earn income, (*iii*) something about the location, rather than the exposure per se, and (*iv*) overall reductions in food consumption during the forest fire months.

To put these estimates of irreversible health impacts in context, we conduct a CBA of oil palm plantations by including social externalities (*Materials and Methods*). Briefly, the analysis of haze-reducing policies considers benefits to health (both avoided losses in income and in mortality), tourism, and transportation, and costs to firms (land preparation) and to agencies for program implementation. To acknowledge and model the heterogeneity and uncertainty inherent in the parameters used in the CBA, we conduct Monte Carlo simulations by allowing each parameter to take on a range of values. This process yields 10,000 net present values (NPVs) for each scenario, summarized in a cumulative distribution function (CDF) for each scenario.

Starting with the baseline scenario of fire-based clearing, we see that 30% of the CDF is negative (Fig. 3). This finding is particularly sensitive to emissions attributed to oil palm fires, exposure to air pollution, income growth, and mortality and human capital losses (*SI Appendix*, Fig. S1). First, we consider the main alternative of clearing mechanically (and avoiding fires) to establish plantations and find that the CDF of social NPV is

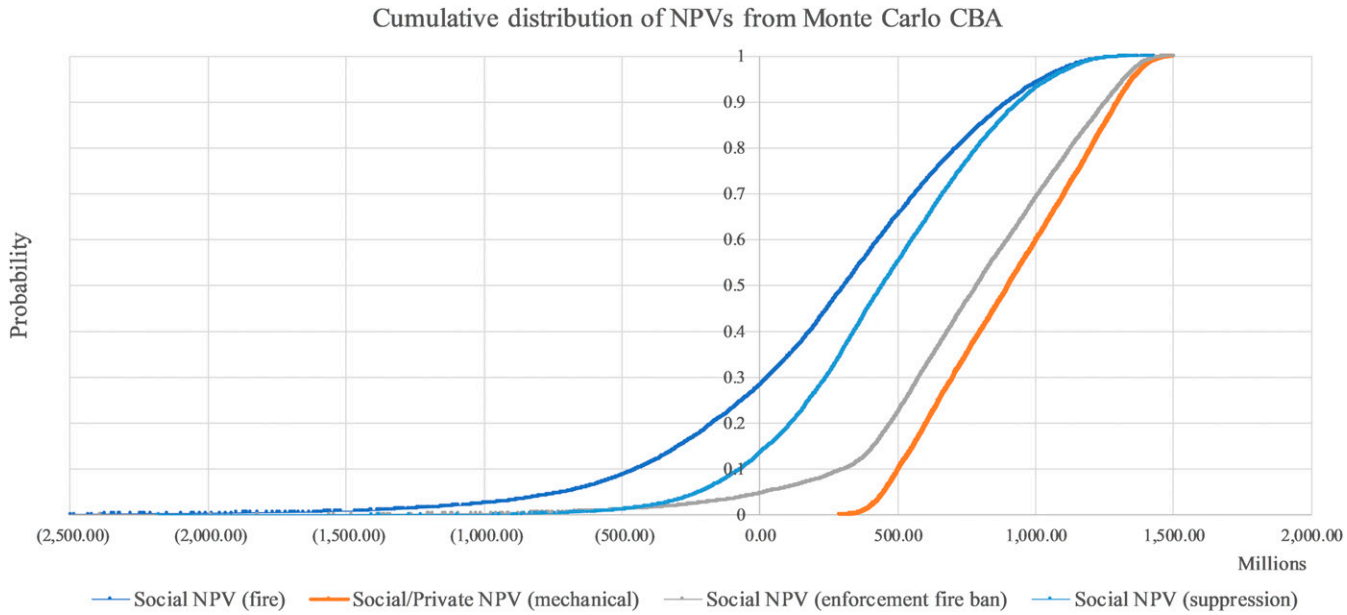

Fig. 3. Comparing social welfare of land clearing for oil palm using fire, mechanical options, and public policies (e.g., ban and suppression).

wholly positive (Fig. 3). This is because the averted air pollution-related health losses are much larger than the increased mechanical clearing costs. Second, we consider stronger enforcement of the ban. The social NPV under this policy is now closer to the social welfare of mechanical clearing (Fig. 3) and is sensitive to the emissions attributed to oil palm, probability of being caught using fires, size of penalty, air pollution attributed to forest fires, and income growth (*SI Appendix*, Fig. S3). Finally, we also consider a fire detection and suppression policy. The distribution of social NPV is mostly positive and better than the baseline by 15 percentage points (Fig. 3). As expected, this finding is sensitive to program efficiency, program longevity, emissions attributed to oil palm, income growth, and program costs (*SI Appendix*, Fig. S4). Collectively, these analyses show that social net benefits from clearing for oil palm using fire is lower compared with net social benefits of (*i*) clearing using mechanical means, (*ii*) stronger enforcement of fire bans, and (*iii*) better fire suppression efforts.

Discussion

Recent studies have used simulations, derived from exposure-response models, to suggest that air pollution from fires is potentially causing deaths in Southeast Asia (36, 41). In our study, we use actual data on (*i*) smoke emissions from a major forest fire event, and (*ii*) children's demographics, to test hypotheses that complement such assumptions-based simulations. Also, unlike the earlier studies that focused on human lives potentially lost, we draw attention to the millions others who survive but with decreased functioning and capability (42, 43). We find a statistically significant negative effect of in utero exposure to air pollution on adults' height—a 0.41 decrease in HAZ at age 17 (or 3.4 cm) due to mean level of in utero exposure to air pollution during the 1997 Indonesian forest fires. Furthermore, these results are robust to a series of checks for confounding factors.

When we feed these health impacts into a broader CBA, and consider the various costs and benefits of policies and practices to control fires and avoid haze, we find that mechanical clearing delivers higher net social benefits compared with fire-based clearing. However, the additional costs of mechanical clearing will reduce firms' profits by 7% on average and as much as 25% (*SI Appendix*, Fig. S2), implying that most firms will not voluntarily comply with the ban. Indeed, previous studies documented the pervasive use of fires even though fire bans were implemented in the 1990s (30, 31).

This implies the need for complementary policies to detect and suppress fires or more rigorously enforce a fire ban. We find that such policies will increase social welfare, even without including the full list of ecological costs of fire-based land clearing such as carbon and habitat. More generally, our CBA illustrates a framework that could be used to evaluate other policies currently being considered by governments and nongovernmental consortiums (e.g., the roundtable for sustainable palm oil), such as green bonds to compensate oil palm firms for profit losses from mechanical clearing that would reduce emissions (26, 27, 44, 45).

Because Monte Carlo simulations reveal that the social NPV of these policies depend on factors that vary by location (e.g., policy effectiveness, emissions attributed to oil palm, local income growth), targeting will be efficient (45, 46). These findings provide strong justification for ongoing Indonesian government policies, including those that focus on restoring peatlands both inside and outside concessions so that forest fires will not spread to the peats (10, 47–49).

In sum, our study contributes to the literature on natural capital loss, forest fires, haze, health, and economic development in three ways. That is, following calls from implementation science research, we attempt to provide approximate, if imperfect, practical advice that policy makers seek, instead of stopping at precise (and sometimes irrelevant or untimely) estimates (7, 37, 50). First, we are one of the first studies of the lagged impacts of early-life exposures to air pollution, using data from Indonesia, a middle-income country critical to global conservation. Second, analyses of planetary health policies—which are multisectoral and interdisciplinary in nature—require methodologically flexible approaches (4). To this end, we first estimate the haze-height effect by applying rigorous quasi-experimental methods on a multisectoral dataset of health, socioeconomic, demographic, and environmental variables. Next, we use these impact estimates in a CBA of various policy solutions to the haze problem. Third, we use Monte Carlo simulations to account for the heterogeneity and uncertainty associated with the many costs and benefits (51, 52). More broadly, this combination of estimation and simulation illustrates an applied research framework (such as the Natural Capital Project) that can be used to mainstream conservation science into the decision making by communities, companies, governments, and donors (53, 54).

Materials and Methods

Data for Statistical Analysis. Data for our regression analysis of health outcomes on air pollution are drawn from three publicly available sources. First, health outcomes and household characteristics are from the 1997, 2000, 2007, and 2014 rounds of the IFLS (55). Early life is defined as prenatal or in utero to the first 6 mo following birth; this period represents the maximum growth velocity for humans (56). Using records of birth dates and mothers' location of residence (defined at the district level), we identify the air pollution exposure for each fetus from August to October 1997, when the fires and air pollution were most intense (23). The final panel used in analysis contains 560 children that were in their early life during August to October 1997 and appeared in each of four waves of the longitudinal survey. The birth months of these children ranged from March 1997 to August 1998 and are mostly uniformly distributed among all months (*SI Appendix*, Table S3). We consider HAZs for each child as our outcome because it is derived from well-established worldwide protocols for measurement and is strongly associated with adulthood socioeconomic outcomes (56, 57). The IFLS also provides data on child and parent demographics and other household variables.

Second, because ground monitoring of air quality is scarce in Indonesia (as in much of the developing world), we use satellite-derived data, that is, AI by the Total Ozone Mapping Spectrometer of the National Aeronautics and Space Administration (NASA) from August to October 1997, to proxy for air quality. AI is the monthly amount of atmospheric aerosols, such as dust and smoke, on a 1° × 1.25° grid that has been shown to reliably represent ambient air quality (58). Third, we obtain data on rainfall and temperature from the National Center for Atmospheric Research Global Precipitation Climatology Centre and NASA's Goddard Institute for Space Studies surface temperature analysis, respectively. Climatic factors are included as controls as they may confound the association between health and AI (59).

We attribute AI exposure to each individual in the following manner. First, from the 1997 IFLS, we know birth dates and district locations of affected individuals ("affected" is defined as being in utero or first 6 mo of life from August to October 1997). While one might be concerned that households may have moved in anticipation of the impending haze, earlier studies using the IFLS data have shown that this is not the case (9). Second, using the satellite-derived data, we assign monthly AI to each district in Indonesia. Third, we assign an average AI exposure to each individual depending on how many months of their early-life stage fall within August to October 1997. For example, an individual born in March 1997 would be defined as being exposed to 1 mo of the haze in August 1997 for his early-life (postnatal) period. On the other hand, an individual born in December 1997 would be defined as being exposed to 3 mo of haze from August to October 1997 for his early-life period (prenatal) and his exposure would be defined as the average AI for the 3 mo. AI has also been used in earlier studies to analyze the health impacts from exposure to air pollutants caused by the 1997 Indonesia forest fires (9, 35, 60, 61). This is partly because AI has been ground truthed, as in, it tracks closely with ground-measured pollution from biomass fires (58). Any measurement error that stems from using AI to proxy for air quality would be classical in that it would result in attenuation bias (i.e., more conservative "smaller" estimates), not systematic bias (i.e., wrong inference).

Statistical Approach. The impact of AI on height, β_1, is obtained by regressing child's health, y_{ijt}, on AI, AI_{ijt}, and a host of controls using least-squares regression method:

$$y_{ijkt} = \beta_0 + \beta_1 AI_{ijt} + \beta_2 AI_{ijt} \cdot I(y_{2000}) + \beta_3 AI_{ijt} \cdot I(y_{2007}) + I(y_{2000}) + I(y_{2007}) + X_i\gamma + \delta_j + \alpha_t + \varepsilon_{ijkt}, \quad [1]$$

where i, j, and t represent child i from location j and born in period t. The subscript k denotes the particular survey in which height was measured (i.e., IFLS 2000, IFLS 2007, or IFLS 2014). X is a vector of parent and household characteristics that could impact child's HAZ, for example, parents' education, parents' heights (a genetic contribution to the child outcomes), and household inputs at birth such as sanitation and clean cooking fuels. $I(y_{2000})$ is an indicator variable equal to 1 if the height was obtained from IFLS 2000. δ and α are district—the same scale at which AI is measured—and birth month fixed effects, respectively. The birth month-by-birth year fixed effects are included to control for unobserved factors that are constant across all individuals born in the same year and month, such as macroeconomic conditions (e.g., currency devaluation related to the Asian financial crisis) or seasonal weather patterns, which might otherwise confound the relationship between AI and HAZ. Similarly, district fixed effects are included to control for any unobserved factors constant across individuals born in the same location, such as access to local nutrition programs. Thus, following an established empirical method in applied statistics, the relationship between air quality and height is identified by removing any confounding differences attributable to location and season. Last, ε is an idiosyncratic error term. To guard against potential biases from the "Moulton" effect due to coarseness of the AI data, we cluster SEs at both the district and birth month-by-birth year levels (62). Three rounds of data from the same individuals are pooled. Therefore, when AI is interacted with the year in which the survey was administered, β_2 and β_3 represent changes in impact of early-life AI on height in the 2000 and 2007 surveys. In other words, the impact of early-life exposure of AI on height at 17 y is β_1, whereas the impact of early-life exposure of AI on height at 3 y is $\beta_1 + \beta_2$, and $\beta_1 + \beta_2 + \beta_3$ at 10 y. By way of summary, note that the AI coefficients are estimated using variation in air quality at the spatial-temporal level. This means that any potential confounders (e.g., the Asian financial crisis) would need to covary at the same temporal (by month) and spatial (by districts) scale with air pollution to bias the estimates of the AI coefficients.

Eq. **1** can be modified to separate the effects of prenatal versus postnatal exposure AI on height:

$$y_{ijkt} = \beta_0 + \beta_1 preAI_{ijt} + \beta_2 postAI_{ijt} + \beta_3 preAI_{ijt} \cdot I(y_{2000}) + \beta_4 preAI_{ijt} \cdot I(y_{2007}) + \beta_5 postAI_{ijt} \cdot I(y_{2000}) + \beta_6 postAI_{ijt} \cdot I(y_{2007}) + I(y_{2000}) + I(y_{2007}) + X_i\gamma + \delta_j + \alpha_t + \varepsilon_{ijkt}. \quad [2]$$

The main difference between Eqs. **1** and **2** is that β_1 and β_2 in Eq. **2** report the effects of prenatal and postnatal exposure to AI, respectively, when the respondents are at 17 y, whereas β_1 in Eq. **1** reports the overall effect of any exposure to AI. Similarly, the coefficients for the other interaction terms in Eq. **2** report any remaining effects of AI from prenatal and postnatal AI exposure. Statistical analyses are conducted using Stata 14.

Robustness Checks for Statistical Analysis. To confirm that our results are not driven by spurious correlations and confounding factors, we conduct four robustness checks.

First, it is possible that the height impacts are driven by high levels of pollution in later years. *SI Appendix*, Table S1 demonstrates that the 1997 pollution was unprecedented, and AI levels were much higher in 1997 compared with later years. Moreover, the district fixed effects control for later years' exposure for all individuals within the same district. The district fixed-effects strategy would not work, however, if those exposed at birth were systematically more likely to migrate to heavily polluted, dirtier locations. Thus, we gathered further information on households' migration history and computed the AI exposure for 1998 and 1999 for their updated locations. In regression analysis that includes 1998 and 1999 AI as additional explanatory variables, only the 1997 exposure is statistically significant (Fig. 2; full results in *SI Appendix*, Table S2). We are not claiming that exposure to air pollution later in life does not impact height, rather that the initial (1997) early-life exposure is the dominant channel.

Second, we consider other nonpollution mechanisms by which height could be affected by the fires. For example, all else being equal, severe air pollution may have reduced a family's ability to work, and, in turn, this would decrease household income and, consequently, caloric intake. This is especially true for those engaged in outdoor work. This loss-of-income mechanism can be tested by interacting the AI variable with proportion of adult household members engaged in outdoor work. Indeed, the impact on HAZ is stronger in this subsample of households with outdoor workers; that is, while the AI coefficient mostly retains similar magnitude and statistical significance from the main results, there seem to be additional impacts on HAZ for households with higher proportion of outdoor workers (Fig. 2 and *SI Appendix*, Table S2). This means that this channel of outdoor work partially explains HAZ differences. However, the key AI coefficients remain significant for all three waves, signaling that the loss-of-income mechanism still leaves room for a large, direct impact of exposure on growth.

Third, to test whether there is something about the location, rather than the exposure itself, that is driving the association, we used the true exposure group but assigned a placebo AI exposure (1996 AI, true exposed cohort). That is, we assigned the 1996 AI exposure to our existing sample of exposed children. These children could not have been exposed to the pollution in 1996, as this is the year before their conception. The placebo test demonstrates this effect: The total impact of 1996 AI on HAZ is insignificant for each of the three rounds (Fig. 2 and *SI Appendix*, Table S2).

Last, we consider another source of potential confounding: if the forest fires influenced reduced food consumption. We can test this channel because of the timing of the surveys, as some household were surveyed during and others after the forest fires. Regression analysis of food consumption shows no statistical difference in consumption during and after the forest fires (*SI Appendix*, Table S3).

CBA. CBAs essentially compare the discounted stream of costs and benefits arising from a project or policy. In this setting, the CBA puts our estimates of health irreversibilities in context but is also essential for understanding the implementation of conservation policies in general (12). First, we conduct a social CBA of oil palm plantations by including social externalities. Second, because the private optimum will diverge from what is best for society, we also conduct the CBA from firms' perspectives to learn, for example, if credible enforcement can incentivize firms to behave in a way so that higher social welfare is achieved. Briefly, the analysis of haze-reducing policies considers benefits to health (both reductions in mortality and morbidity), tourism, and transportation, and costs to firms (land preparation) and to agencies for program implementation. While we included the ecological costs of mechanical clearing, we are not able to include ecosystem-related costs of fire clearing related to carbon and habitat because we could not find conclusive quantitative data on these benefits. Including these other costs would make the net social benefits of oil palm cleared using fire even more negative.

Third, to acknowledge and model the heterogeneity and uncertainty inherent in the parameters used in the CBA, we conduct Monte Carlo simulations (63). That is, we allow each parameter to take on a range of values (obtained from the literature) and specify a statistical distribution for these values (either uniform or normal distribution). For example, the AI–HAZ relationship is estimated in this study with a SD. To fully utilize the range behind each parameter, we ran 10,000 trials in the Monte Carlo simulation whereby, in each trial, a value for each parameter is randomly from the specified statistical distribution. Eventually, this process yields 10,000 different NPVs, which constitute a CDF.

The 26 parameters that underlie these computations and the eight primary equations that combine these parameters to estimate benefits and costs are described in *SI Appendix*, Tables S3 and S4. Without describing each and every parameter and equation (*SI Appendix*), we briefly summarize some of the key computations here.

Haze attributed to oil palm. Neither are forest fires the only source of air pollution in Indonesia, nor are all forest fires caused by establishing oil palm plantations. While there is a large literature examining the role of the causes of the 1997 forest fires and its effects, there is no specific study that directly quantifies the pollution attributable to oil palm or any concessions (38, 39). However, we can draw on this literature and estimate the attributable fraction as follows. First, we compare the AI in the August to October 1997 period to both August to October 1996 and to January to June 1997 periods, when there were fewer forest fires. Thus, we estimate that 57–77% of air pollution is from forest fires during the August to October 1997 period. Second, we rely on a recent study to approximate the forest fire emissions attributable to oil palm plantations, which suggests the range 10–60% of all pollution emissions is because of oil palm plantations (48). Third, we multiply these two fractions to compute the proportion of air pollution attributed to oil palm-related forest fires. Critically, recognizing that this attributable fraction can vary (as with other causes) from the low to high range of this product, we build this variability into the 10,000 Monte Carlo simulations, introduced previously.

Avoided mortality damages. Epidemiological models estimate the total mortality burden for the 1997 fires in Indonesia to be around 15,000 deaths (64). In monetary terms, we assign a value-of-statistical life (VSL) of US$108,900 (in 2008 US dollars) to these deaths. This VSL estimate is obtained by starting first with OECD baseline VSL of US$3.3 million (in 2008 US dollars) and then applying "benefits transfer" logic.

Avoided income loss. For loss of income, we start with a well-established literature that shows that relative height is correlated with adult mortality, morbidity, neurodevelopmental, and economic capability (56). Specifically, height has been shown to be correlated with earnings; for example, 1 SD in HAZ at adulthood is associated with 8% increase in adult income (57). Thus, our estimate of a 0.41 SD HAZ decrease (i.e., from the 2014 wave when the cohort is about 17 or 18 y old) translates into ∼3.3% decrease in income through human capital channels. Consider the fact that 1.13 million individuals were in their prenatal stage during August through October 1997 in the impacted provinces of Sumatra or Kalimantan, where the air pollution and fires were most intense (that is, what we report next is a conservative estimate because others in other provinces were partially exposed but we do not count them). Assuming, (*i*) their working age spans 21–58 (the official retirement age), (*ii*) the average annual wage for blue-collar work at US$860 (from Indonesian Statistical Department), (*iii*) a social discount rate of 8%, and (*iv*) an annual real wage increment of 2% for the first 15 working years (from Indonesian Statistical Department), we estimate that the lifetime productivity loss for this exposed population of 1.13 million is about US$392 for each individual.

Profits of oil palm plantations. The operating costs and revenue of oil palm plantations are obtained from Butler et al. (65). The plantations are evaluated on a 25-y basis and the range of operating costs and revenue is based off the high- and low-yield scenarios in the original analysis. The size of the oil palm planation is assumed to be 100,000 ha, based off new plantation area in 1998 (66). Clearing by fire costs between $82/ha to US$320/ha (47, 67). In contrast, clearing using mechanical means is much higher and ranges from low of $200/ha to $990/ha (47, 67).

Fire detection and suppression. Our analysis of the early detection and suppression policy rests primarily on the related parameters of program costs and effectiveness, and program life. The effectiveness of this policy ranges from 0 to 0.6. That is, emissions from oil palm plantations is reduced by 60% if fully effective. Additionally, to allow for spatial targeting of this policy, we model positive correlation between program effectiveness and both the levels of emissions from oil palm and the income growth. The Indonesian government approximates that the costs of early-detection and suppression program would range between US$450 million to US$2.3 billion. Last, we assume this program will last between 2 and 10 y.

Enforcing the fire ban. We model an enforcement policy for fire bans and the firm's reaction on three parameters—probability of detecting which plantations are using fires, the resulting penalties, and firms' risk aversion. We conservatively estimate the probability of identifying fires to range from 0 to 20%. The penalties are derived from recent court judgements on plantation owners that were found guilty of starting fires and are estimated to range from US$300/ha to US $40,000/ha. A risk-adverse firm would reduce fires more than proportional to the ratio of expected penalty vs. additional costs of mechanical clearing. We consider the risk aversion to range between 0.1 (risk averse) and 3 (risk taking).

ACKNOWLEDGMENTS. For excellent comments, we are grateful to the seminar participants at Duke University, Indian Statistical Institute Delhi (Centre for Research on the Economics of Climate, Food, Energy and Environment's inaugural meeting), National University of Singapore, Zhejiang University, and the National Academy of Sciences (Sackler Colloquium), and to Marc Jeuland, Emily Pakhtigian, Steve Polasky, Alberto Salvo, Chen Shuai, E. Somanathan, Anant Sudarshan, Faraz Usmani, and two anonymous reviewers. Ruoming Fang provided excellent research assistance.

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Air Quality Impacts from Prescribed Forest Fires under Different Management Practices

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Received May 13, 2007. Revised manuscript received November 16, 2007. Accepted January 21, 2008.

Large amounts of air pollutants are emitted during prescribed forest fires. Such emissions and corresponding air quality impacts can be modulated by different forest management practices. The impacts of changing burning seasons and frequencies and of controlling emissions during smoldering on regional air quality in Georgia are quantified using source-oriented air quality modeling, with modified emissions from prescribed fires reflecting effects of each practice. Equivalent fires in the spring and winter are found to have a greater impact on $PM_{2.5}$ than those in summer, though ozone impacts are larger from spring and summer fires. If prescribed fires are less frequent, more biofuel is burnt in each fire, leading to larger emissions and air quality impacts per fire. For example, emissions from a fire with a 5-year fire return interval (FRI) are 72% larger than those from a fire of the same acreage with a 2-year FRI. However, corresponding long-term regional impacts are reduced with the longer FRI since the annual burned area is reduced. Total emissions for fires in Georgia with a 5-year FRI are 32% less than those with a 2-year FRI. Smoldering emissions can lead to approximately 1.0 or 1.9 $\mu g/m^3$ of $PM_{2.5}$ in the Atlanta $PM_{2.5}$ nonattainment area during March 2002.

Introduction

Air pollutants from a prescribed fire about 80 km southeast of metro Atlanta on February 28, 2007 led to parts of the city being exposed to unhealthy levels of $PM_{2.5}$ for several hours. Observed 1-h $PM_{2.5}$ concentrations at several monitors in the city reached higher than 145 $\mu g/m^3$ (U.S. National Ambient Air Quality Standard (NAAQS) for 24-h $PM_{2.5}$ is 35 $\mu g/m^3$), increasing by over 100 $\mu g/m^3$ in two hours (*1*). In addition, as the plume hit, 1-h average ozone concentrations increased markedly from 63 to 95 ppb at one monitor.

Unlike wildfires, prescribed fires are intentionally ignited in order to maintain ecosystem health and minimize adverse impacts of long-term fire suppression while protecting property (*2–5*). About 2 million acres per year of federal forests were burned by prescribed fires from 1998 to 2006, in comparison to around 6 million acres of wildfires (*6*). Prescribed fires and wildfires together contributed about 20% of the fine particulate matter ($PM_{2.5}$) emissions in the United States (*7*). Results from both field measurements and numerical modeling have shown significant air quality degradation due to forest fire emissions (*8–10*).

Prescribed fires are usually planned for conditions that are not likely to lead to their becoming uncontrolled, and when feasible they are often planned to reduce impacts on populated areas. They are typically limited in extent, spatially and temporally. Therefore, emissions and corresponding air quality impacts from prescribed fires can be reduced by adopting smoke reduction techniques and choosing better dispersion conditions for burning, as suggested by both U.S. Environmental Protection Agency (EPA) and U.S. Forest Service (*4, 11*). Smoke reduction is usually achieved by reducing burned area and fuel consumption, and increasing combustion efficiency of fires. Such techniques include, but are not limited to, reducing forest fuels using mechanical and chemical methods, igniting back fires or aerial ignition, burning before precipitation or at high frequencies, using air curtain incinerators, and rapid mop-up (*4, 11*). Different technologies and their combinations can be chosen for different management goals. Though significant air quality impacts from application of different technologies are expected, such impacts are rarely quantified.

Increased application of prescribed fires is expected, given their characteristics of being controlled and requirements from ecosystem and air quality management (*4, 5*). Furthermore, a recent study showed that climate change led to increased wildfire activities in the western United States (*12*). Appropriate management practices, including prescribed fires, are increasingly required to reduce wildfire hazards. Therefore, understanding how forest management practices can change air quality impacts from prescribed fires should be addressed. Here, a source-oriented air quality model, capable of predicting air quality under different emissions and meteorological conditions, is employed. Historical air quality conditions are first reproduced using the actual prescribed fire emission patterns together with emissions from other sources as inputs. Emissions from prescribed fires are then modified to reflect the effects of various management practices.

Methods

Georgia, where forests cover more than 66% of the total land and prescribed fires have been widely used, is chosen for this case study. More than 92% of Georgia forestland is owned by private parties. Between 1994 and 2005, an average of 0.86 million acres per year of private and public forests were burned by prescribed fires in Georgia (*13*), in comparison with an average of 2 million acres per year in the United States on Federal forests. These fires mainly burn in the southern pine forests (Supporting Information, Figure 1), and consume understory fuels, such as grass, live shrubs, and needles, without significantly damaging trees (*2, 13, 14*).

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10.1021/es0711213 CCC: $40.75
Published on Web 03/05/2008

Air quality impacts from forest fires with different burning seasons and frequencies are evaluated in this paper, as well as air quality impacts from emissions during the smoldering combustion stage.

Historical air quality conditions during 2002 are simulated using the Community Multiscale Air Quality (CMAQ) model v. 4.3 (*15*), a three-dimensional, detailed photochemical atmospheric model. Meteorological conditions are simulated with the Pennsylvania State University (PSU)/National Center for Atmospheric Research (NCAR) Mesoscale Modeling System Generation 5 (MM5) (*16, 17*) and emissions are processed with the Sparse Matrix Operator Kernel Emissions (SMOKE) Modeling System v. 2.1 (*18*). The 2002 Visibility Improvement State and Tribal Association of the Southeast (VISTAS) emission inventories (*19*) are used with updated biomass burning emissions (*20*). Modeling performance is evaluated by comparing simulations with ozone observations from EPA's Aerometric Information Retrieval System (AIRS, http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm), and with total and speciated $PM_{2.5}$ observations collected as part of the Interagency Monitoring of Protected Visual Environments (IMPROVE, http://vista.cira.colostate.edu/improve/), the SouthEastern Aerosol Research and Characterization (SEARCH) (*21*), the Assessment of Spatial Aerosol Composition in Atlanta (ASACA) (*22*) and the Speciation Trends' Network (STN, http://www.epa.gov/ttn/airs/airsaqs) networks. Mean normalized bias for simulated ozone is within ±15%, and mean normalized error is less than 35% (*23*). Overall performance of simulated $PM_{2.5}$ is well within recent performance suggestions (*24*). Detailed information on the modeling system and performance can be found elsewhere (*20*).

Emissions from forest fires are calculated as the product of the burned area (A), fuel consumed per area (F_a) and an emission factor (E_f) (*25*):

$$E = A \times F_a \times E_f \quad (1)$$

Here, A is determined from current forest fire records (*13*), F_a is the amount of biomass consumed during a forest fire per area, and E_f is the ratio of the mass of pollutant emitted per unit mass of fuel consumed. F_a and E_f are functions of fuel condition (e.g., moisture content and availability) and meteorology. Forest managers choose to burn when fuel conditions are within specific limits, in order to sustain a burn, but minimize potential damage (e.g., to roots). Such fuel properties are chosen for simulation here.

Burning Season. Forest management issues involve choice of periods for prescribed fires, mainly depending upon the purpose of burning and ecosystem requirements. In Georgia, burning during winter and spring is most common (Supporting Information, Figure 2), as forests burned during summer and fall are more likely to die, and burning is harder to control due to commonly unstable atmospheric conditions in these periods (*26*). More than 86% of prescribed fires were scheduled between December and April according to records between 1994 and 2005, with 37% of the annual total occurring in March alone (*13*).

Four months in 2002, including January, March, May, and July, are selected to represent different burning seasons. March is chosen since it is the month with the most prescribed fires in Georgia. Burning in January is also frequent, with forest area burned about one-third of that in March. Natural wildfires are mainly ignited by lightning and occur in Georgia during May and June when lightning frequency is high and summer thunderstorms have not provided much moisture (*2, 4, 27*). Therefore, burning in May is also studied. Finally, burning in July is investigated with particular interest in corresponding air quality impacts during summer ozone seasons. Fall is not considered, because it is neither a naturally preferred season nor practical for prescribed fires. Simulations with and without prescribed fire emissions during respective months of 2002 are first conducted to investigate air quality impacts from existing fires. Emissions from March 2002 prescribed fires are also individually input into CMAQ for the other three months, together with the applicable emissions from other sources pertaining to the specific month, which vary according to time of year and meteorology. Such simulations are used to evaluate air quality impacts of the same fires during different burning seasons.

Burning Frequency. Burning frequencies (characterized by fire-return intervals, FRIs) influence fuel consumption. For a fixed burned area A, forest fire emissions change proportionally with F_a (1), which increases with longer FRIs. Prescribed fires in Georgia are currently applied to specific areas periodically in intervals of 2−5 years (*2, 14*), and would burn too severely if FRIs were longer than 5 years (*2*). The characteristic F_a (F_{aC}) for the prescribed fires in Georgia is 4 tons/acre for a 3.5-year FRI (the mean interval when considering 2 to 5 years), which has been used to develop the most recent emission inventory (*28*). Here, F_{aC} is used to calculate F_a values for 2-, 3-, 4-, and 5-year FRIs by multiplying with a relative ratio calculated for each FRI. These ratios are estimated using F_a values calculated by a fire behavior model, the First Order Fire Effects Model (FOFEM, http://fire.org) v 5.21. Default preburn fuel characteristics (such as relative abundance of particular fuelbed components and the condition of the fuel) for loblolly and slash pines (major forest types burned by prescribed fires in Georgia) at various ages are used as inputs. Since the default inputs in FOFEM do not represent fuel conditions in Georgia, the F_as calculated by FOFEM are used to scale the F_{aC} calculated for Georgia to each FRI.

The above estimates for a specific burned area are referred to as an "individual" fire impact, assuming FRIs for other prescribed fires in Georgia do not change. When FRI changes are applied to all forests in Georgia, the corresponding estimates are referred to as an "aggregate" fire impact. In this case, since FRIs influence not only F_a, but also yearly burned acreage (A), corresponding emissions do not simply increase with FRIs as does a single fire. For example, in Georgia, about 0.86 million acres of forests were burned per year by prescribed fires (average for 1994 to 2005 (*13*)). With the assumed 3.5-year FRI, the total forest area under management using fires is approximately 3 million acres (0.86 multiplied by 3.5). If a 2-year FRI were used, 1.5 million acres would be burned each year, and if a 5-year FRI were employed, 0.6 million acres would be burned. Here, annual emissions from prescribed fires with different FRIs ranging from 2 to 5 years in Georgia are calculated with respective A and F_a values.

Flaming and Smoldering. There are two combustion stages of forest fires: flaming and smoldering. Of the two, smoldering combustion is relatively incomplete with larger emissions per mass of fuel burned and lower heat release (*5, 29*). Due to the different heat release rate and timing, emissions during these two stages also have different dispersion behaviors in the atmosphere. Since flaming and smoldering emissions sometimes occur simultaneously, the flaming stage is defined, here, as emissions which are influenced by the strong convection associated with a flame front (*29, 30*). Thus, the portion of smoldering emissions which occurs during flaming is defined as a part of the flaming stage.

Air quality impacts from emissions during each combustion stage are simulated using CMAQ during March 2002, when prescribed fires are the largest in Georgia. Since prescribed fire emissions in the original emission inventory are total emissions from both stages, such emissions are split into each stage based on corresponding emission fractions.

Such fractions are estimated using two different methods. One method uses specific F_a values in combination with applicable emission factors for each combustion stage (*25, 31*). F_as are estimated using two fire behavior models, the Fire Emission Production Simulator (FEPS, http://www.fs.fed.us/pnw/fera/feps/) v1.0, and the Fire Characteristic Classification System (FCCS, http://www.fs.fed.us/pnw/fera/fccs). They can calculate separate F_a values during flaming, short-term smoldering, and residual smoldering combustion (RSC). According to the above definition of combustion stages, F_a values during both flaming and short-term smoldering in the two models are treated as flaming, and RSC is treated as smoldering. The other method is based on the diurnal temporal profile (showing hourly emission fractions) (*7*) and typical operation times for prescribed fires (*11, 13, 26*). The period between 10:00 and 17:00 is treated as flaming stage, and the rest as smoldering. Hourly emission fractions in the profile during the period designated for each stage are added to calculate emission fractions for each stage. In addition, the diurnal profile for total emission during both stages is split into two different profiles for flaming and smoldering. The hourly fractions during each stage defined above are renormalized to calculate the new diurnal profiles. The difference between simulations with and without specific emissions shows respective air quality impacts.

Results and Discussion

Burning Season. Different burning seasons feature varying meteorological conditions (ventilation, sunlight, and humidity) and levels of biogenic emissions. Monthly averages (and peaks for ozone) are calculated for $PM_{2.5}$ and ozone to compare air quality impacts during different burning seasons. $PM_{2.5}$ contributions from historical prescribed fires in 2002 averaged over the state of Georgia peak in March, being 4.8 $\mu g/m^3$ in March and 1.5 $\mu g/m^3$ in January, though local short-term contributions can be much higher. Corresponding contributions averaged for the Atlanta $PM_{2.5}$ nonattainment area are smaller, being 1.9 $\mu g/m^3$ in March and 0.7 $\mu g/m^3$ in January due to a longer distance from the prescribed fires. Source contributions of historical prescribed fires are negligible during May and July.

When emissions originally calculated for prescribed fires in March 2002 are applied to January, May, and July 2002, significant differences in their $PM_{2.5}$ contributions are simulated. The impacted regions and magnitudes diminish from January to July (Supporting Information, Figure 3). Such emissions lead to 7.3 $\mu g/m^3$ in January, 3.4 $\mu g/m^3$ in May, and 3.0 $\mu g/m^3$ in July of $PM_{2.5}$ averaged for the state of Georgia (Table 1). Impacts on $PM_{2.5}$ in the Atlanta nonattainment area are 2.0 $\mu g/m^3$ in January, 1.3 $\mu g/m^3$ in May, and 0.9 $\mu g/m^3$ in July. Decreased burning impacts during summer seasons can be explained by stronger vertical mixing and increased thunderstorm activity. Thunderstorms both increase ventilation and lead to pollutant rainout, evidenced, in part, by the increased rain in July versus May (11.8 cm versus 8 cm, http://www.sercc.com/climateinfo/monthly/state_avg_data/Georgia_prcp.html).

It is interesting to note a local discrepancy in the seasonal variation of $PM_{2.5}$ impacts from fires in the Okefenokee swamp, a Class I area located in the southeast of Georgia. When applying the same emissions from prescribed fires as in March 2002, the model shows fire contributions of 3.7 $\mu g/m^3$ of $PM_{2.5}$ in January, 1.4 $\mu g/m^3$ in March, 0.6 $\mu g/m^3$ in May, and 1.1 $\mu g/m^3$ in July (Table 1). This local difference (higher contribution during July than May) is partially explained by change of prevailing wind direction, and should be addressed in control strategy developments for protecting air quality in specific areas. In order to reduce $PM_{2.5}$ impacts, burning during summer seasons might be preferable for Georgia considering air quality impacts, alone, as tested for 2002.

TABLE 1. Source Contributions from Prescribed Fires in Georgia during January, March, May, and July 2002 Simulated with Two Sets of Emissions: (A) Simulations with Historical Prescribed Fires Emissions in the Respective Months and (B) Simulations with Historical March 2002 Emissions Applied to January, May, and July 2002 [a]

		January	March	May	July
A. Source contributions from historical prescribed fires in respective months of 2002					
$PM_{2.5}$	Georgia average	1.5	4.8	0.1	0.1
	Atlanta average	0.7	1.9	0.1	<0.1
	Okefenokee	2.7	1.4	<0.1	<0.1
Ozone	8-h average Atlanta	−0.01	0.30	0.02	<0.01
	8-h peak Atlanta	0.06	1.0	0.08	<0.01
	1-h maximum	2.2	16	0.73	0.98
B. Source contributions from the same prescribed fires emissions as in March 2002					
$PM_{2.5}$	Georgia average	7.3	4.8	3.4	3.0
	Atlanta average	2.0	1.9	1.3	0.9
	Okefenokee	3.7	1.4	0.6	1.1
Ozone	8-h average Atlanta	<0.01	0.30	0.40	0.27
	8-h peak Atlanta	0.18	1.0	2.4	0.48
	1-h maximum	12	16	21	23

[a] Monthly average $PM_{2.5}$ source contributions ($\mu g/m^3$) for Georgia and Atlanta refer to spatial averages of simulations for all grids within the state of Georgia and the Atlanta $PM_{2.5}$ nonattainment area, respectively. The Atlanta $PM_{2.5}$ nonattainment area includes 22 counties according to U.S. EPA designation on December 17, 2004. Values for Okefenokee refer to the simulations for the grid where the IMPROVE Okefenokee site (in Class I area) is located. Ozone source contributions are first calculated as monthly average and peak of daily maximum 8-h ozone (ppbv). Monthly average and peak ozone contributions are calculated for each grid cell, and then such contributions are averaged for all grids inside the Atlanta Metropolitan area (including 32 counties). They are referred to as "8-hr average Atlanta" and "8-hr peak Atlanta". Maximum 1-h ozone contributions in the whole modeling domain are also provided.

Prescribed fires have also been viewed as a source of ozone pollution during summer due to their NO_x, VOC, and CO emissions, and are addressed by different policies (e.g., burning bans in the Atlanta area during the summer O_3 season). In 2002, prescribed fires led to an increase of 1.0 ppbv during March in the monthly peak ozone concentrations averaged over the Atlanta metropolitan area (including 32 counties, a region with historical O_3 problems), with negligible contributions in May and July due to few fire activities (Table 1). Their ozone contributions in January are relatively small due to slow photochemical processes, though there were significant prescribed fires in that month. Slightly negative O_3 source contributions in January are observed when excess NO_x emitted from fires titrate O_3 and radicals.

When the same level of prescribed fires as in March 2002 is applied to other months, additional emissions lead to an increase of 0.18 ppbv monthly peak ozone in January averaged over Atlanta, 2.4 ppbv in May, and 0.48 ppbv in July (Table 1 and Supporting Information, Figure 3). Though O_3 formation potentials in July are the highest, less O_3 is formed by the additional prescribed fire emissions in July than in May and March. This is due to more rapid dispersion and reduced ozone sensitivities at high ozone levels in July. Since exceedance of the O_3 NAAQS is not observed during January and March in the Atlanta area, impacts of prescribed fires in these periods on O_3 are of less concern from a regulatory point of view, but may still have health implications.

TABLE 2. Typical Annual Burned Area (A), Fuel Consumption (F_a), and Emissions from Prescribed Fires with Different FRI in Georgia

FRI (year)	A (million acres)	F_a (tons/acre)	emissions (10^3 tons) CO	VOC	NO_x	NH_3	SO_2	PM_{10}	$PM_{2.5}$
2−5 (3.5)	0.86	4.0	519	24	11	2.3	3.1	51	43
2	1.51	2.9	668	31	14	3.0	4.0	65	56
3	1.00	3.5	535	25	12	2.4	3.2	52	45
4	0.75	4.4	498	23	11	2.2	3.0	48	42
5	0.60	5.0	455	21	10	2.0	2.7	44	38

The above ozone impacts averaged for the Atlanta area are relatively small, since ozone impacts of fires peak in their vicinity and most fires in Georgia are far from Atlanta. In nearby areas, historical fires in 2002 led to a maximum increase of 16 ppbv in 1-h ozone concentrations during March. When applying the same emissions from prescribed fires as in March 2002, the model shows maximum fire contributions of 12 ppbv during January, 21 ppbv during May, and 23 ppbv during July in 1-h ozone concentrations. The increasing trend from January to July agrees with corresponding photochemical potentials.

The same daily emissions from forest fires have been assumed in the simulations due to lack of data, even though not all meteorological conditions are equally preferred for burning. Impacts of prescribed fires on $PM_{2.5}$ concentrations vary significantly from day to day (Supporting Information, Figure 4). Violations of the 24-h $PM_{2.5}$ standard (35 $\mu g/m^3$) are simulated when source contributions from prescribed fires are large. Explicitly, probability of daily $PM_{2.5}$ source contributions from prescribed fires larger than 35 $\mu g/m^3$ is about 4% for grids in Georgia and days during January (Supporting Information, Figure 5). The probability is around 2% in March and very small in May and July. However, the probability of locations near a prescribed fire having such an exceedance is quite high. For the Atlanta $PM_{2.5}$ nonattainment area, March is the month with the highest daily $PM_{2.5}$ impacts. Exceedance days generally have poor dispersion characteristics or a wind direction toward the Atlanta area, and should be avoided in burning practice.

Relationships between air quality and forest fires during different seasons change with pollutants concerned, distance of fires and concerned regions, and wind directions. In order to meet requirements from varying air quality and ecosystem management goals, air quality impacts of both O_3 and $PM_{2.5}$ should be considered, along with other associated impacts on human health, visibility, climate, and ecosystem health. Different seasons are also associated with different fuel conditions, and thus corresponding emissions and air quality impacts vary. For example, fuel moisture contents are high during summer (the growing season). Higher fuel moisture contents are usually associated with less fuel consumption and more incomplete combustion (so higher emission factors). According to eq 1, emissions could either increase or decrease, as well as corresponding air quality impacts. Detailed information such as fuel moisture content by component is required to more fully understand air quality impacts of prescribed fires under different fuel conditions during different seasons. However, such information is rarely available. The results above are based on typical fuel conditions for prescribed fires, and mainly reflect impacts from different meteorological conditions during different seasons.

Burning Frequency. Ratios of fuel consumption (F_a) at different forest ages calculated by FOFEM are similar among different forest types and are further averaged to estimate the ratios of F_a with different FRIs. While the F_a with the current average of 3.5-year FRI is 4 tons/acre, the F_a values with 2-, 3-, 4-, and 5-year FRIs are, respectively, 2.9, 3.5, 4.4, and 5.0 tons/acre. For an "individual" fire, emissions are proportional to F_a. Emissions from an individual forest fire with a 5-year FRI are approximately 72% larger than those from a fire of the same acreage if a 2-year FRI was employed. Their corresponding air quality impacts on local $PM_{2.5}$ have similar trends, since fire emissions mainly impact primary $PM_{2.5}$ species and are approximately linear to their impacts on $PM_{2.5}$ concentrations in current modeling. For an "aggregate" impact (Table 2), annual emissions from prescribed fires in Georgia with a 5-year FRI (38 thousand tons $PM_{2.5}$) are 32% less than those with a 2-year FRI (56 thousand tons $PM_{2.5}$), as less forest area is burned each year when a less frequent FRI is used. Less burned area offsets the increase of F_a per fire.

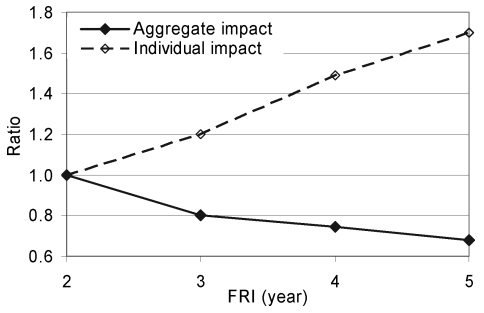

FIGURE 1. Trends of "individual" and "aggregate" prescribed fire emissions with different fire-return intervals (FRIs). "Individual impact" refers to the case when FRIs only changes for an individual fire and "aggregate impact" refers to the case when FRIs change for all forest area.

The opposing trends between "individual" and "aggregate" forest fire emissions and corresponding air quality impacts on $PM_{2.5}$ (Figure 1) pose a critical problem in forest and air quality managements in choosing an optimized FRI. Generally, a longer FRI is preferred to reduce long-term and regional air quality impacts, while a shorter FRI helps avoid intense short-term and local impacts. Specifically, a longer FRI can lower forest fire impacts on annual average $PM_{2.5}$ levels, however, increase chances of higher daily $PM_{2.5}$ levels. Thus, protecting acute exposure and responding to the new more stringent 24-h NAAQS, would suggest using more frequent burning (a smaller FRI), while attaining the annual standard would be more likely under less frequent burning strategies. In addition, the locations of forest fires are important for policy decisions. If forest fires are close to a sensitive area, short FRIs might be adopted to avoid acute deterioration of air quality though sacrificing longer term air quality. Longer FRIs might be employed to minimize long-term air quality impacts in relatively remote regions, where there is less concern about local episodic air quality impacts. Moreover, the increased risk of fire escaping with a longer FRI should also be considered in forest management.

Flaming and Smoldering. Prescribed fires emitted 560 tons/day $PM_{2.5}$ in Georgia during March 2002, using the VISTAS fire emission estimation method (*28*). Thirty percent (170 tons/day) of such emissions are released during smoldering, according to the diurnal temporal profile for prescribed fires and the designated periods for both combustion stages (Table 3). Corresponding $PM_{2.5}$ source contributions during both stages are mainly caused by primary $PM_{2.5}$ emissions. While some impacts on ozone from forest fires are simulated, such impacts are small in March and are not discussed here. Simulations with respective emissions

TABLE 3. $PM_{2.5}$ Emissions from Prescribed Fires in Georgia during Flaming and Smoldering and Corresponding Monthly Average $PM_{2.5}$ Source Contributions for March 2002 Using Two Different Methods: "Diurnal Profile" and "Specific F_a and E_f"[a]

	diurnal profile			specific F_a and E_f		
	total	flaming	smoldering	total	flaming	smoldering
emissions (tons/day)	560	390	170	560	250	310
Georgia	4.8	1.9	2.9	6.6	1.2	5.4
Atlanta	1.9	0.90	1.0	2.5	0.60	1.9

[a] The values for Georgia and Atlanta refer to spatial averages of simulations for all grids within the state of Georgia and the Atlanta $PM_{2.5}$ nonattainment area, respectively.

and diurnal temporal profiles (Supporting Information, Figure 6) during flaming and smoldering indicate that the total prescribed fires lead to 4.8 $\mu g/m^3$ of monthly average $PM_{2.5}$ for Georgia, 60% of which is caused by emissions during smoldering. In the Atlanta $PM_{2.5}$ nonattainment area, these fires lead to a monthly average $PM_{2.5}$ of 1.9 $\mu g/m^3$, with 53% from smoldering (Table 3).

When using specific F_a values and emissions factors for each combustion stage, we estimate that 60% of CO, 55% of VOC, 20% of NO_x, 70% of NH_3, 70% of SO_2, and 55% of $PM_{2.5}$ and PM_{10} emissions are from the smoldering stage. Explicitly, F_a values estimated by fire behavior models (FEPS and FCCS) for typical forest types in Georgia (e.g., loblolly pine and slash pine) indicate that approximately 38% of fuels are consumed during the smoldering stage. In comparison, fuel consumption during the smoldering stage was reported to be 38–44% in the Brazilian Amazon (*32*) and over 50% in temperate and boreal fires (*33*). Prescribed forest fires in Georgia mainly consume surface fuels; large woody and below-ground fuels are usually not consumed during smoldering. Therefore, less fuel is consumed during smoldering in Georgia, supporting the estimates by FEPS and FCCS. Even though estimated fractions of fuel consumption during the flaming stage are larger than those during smoldering, respective emission factors are much higher during smoldering for all pollutants except NO_x. As such, higher emission fractions during smoldering than flaming (except NO_x) are estimated. Such larger $PM_{2.5}$ emissions during smoldering (310 tons/day) increase estimated $PM_{2.5}$ source contributions from prescribed fires, by an additional 1.8 $\mu g/m^3$ averaged over Georgia and 0.6 $\mu g/m^3$ for the Atlanta area. The larger emissions during smoldering also lead to increased $PM_{2.5}$ contributions from smoldering: 81% for Georgia and 76% for Atlanta.

Large differences in estimated air quality impacts from forest fires during different combustion stages suggest the need to improve our understanding of emissions during the different stages. In addition, using the same diurnal profile for all fires is an approximation, recognizing that different fires will have different temporal characteristics. We have chosen a single one based upon the average found for prescribed fires. Information on fire-specific diurnal profiles is desired for further study. In spite of these uncertainties, air quality impacts per unit emissions during smoldering are larger than those during flaming, as dispersion is reduced during night when smoldering dominates. If techniques mentioned above (e.g., preprocessing fuels with a large potential to smolder using mechanical methods, keeping high moisture in large woody fuels, burning before precipitation, and rapid mop-up) are applied to reduce emissions during the smoldering stage, air quality impacts from forest fires can be significantly reduced. Due to an almost linear relationship between forest fire emissions and corresponding impacts on $PM_{2.5}$, a 50% reduction in smoldering emissions would lead to an approximately 1.5 or 2.7 $\mu g/m^3$ reduction in monthly $PM_{2.5}$ source contributions simulated for March 2002 in Georgia, using the two different methods. Similarly, such reduction can reduce approximately 0.5 or 1.0 $\mu g/m^3$ $PM_{2.5}$ in the Atlanta $PM_{2.5}$ nonattainment area.

Though impacts from other management practices or smoke reduction techniques are not discussed here, such impacts can be readily quantified using similar approaches. Different types of management practices can be applied at the same time and impact each other. For example, less frequent burning can lead to more fuels in larger sizes, which usually can not be consumed completely during flaming and contribute significantly to smoldering emissions. Impacts of controlling smoldering emissions are thus related to burning frequencies, and can be quantified using the approach developed in this study as long as there is information regarding fuel distributions by burning frequencies.

The quantified air quality impacts of prescribed fires in this study are for fires under typical fuel conditions and are based on meteorological conditions during 2002. Ignoring variability in fuel conditions and year-to-year variability in meteorological conditions can lead to uncertainties, however, the conclusion that air quality impacts of prescribed fires vary significantly with forest management practices, will not change. This conclusion is important for air quality management decisions. Due to the important role of fires in natural system and their significant impacts on air quality, cooperation between air quality and forest management specialists is crucial. This study provides information to bridge the two different areas, and highlights information that often is not available but would greatly enhance our understanding of air quality impacts from prescribed fires. Quantification of such impacts under different forest management practices is becoming critical to nonattainment designation, control strategy development, and effective air quality and ecosystem management. With the increased application of prescribed fires in forest management to reduce the risk of wildfires and improve ecosystem health, the methods and information provided can help avoid episodes leading to significant deterioration of air quality.

Supporting Information Available

Map of burned areas for prescribed forest fires in Georgia, daily $PM_{2.5}$ concentrations, and source contributions from prescribed fires and temporal diurnal profiles. This information is available free of charge via the Internet at http://pubs.acs.org.

Acknowledgments

The work reported in this manuscript is funded by the U.S. Environmental Protection Agency through grants (RD83096001, RD82897602, and RD83107601). We acknowledge Alan Dozier, Dan Chan, Neal Edmonson, and Dr. Jim Paul of the Georgia Forestry Commission for their valuable information on prescribed fires in Georgia.

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ES0711213

Health effects of the 2012 Valencia (Spain) wildfires on children in a cohort study

Environmental Geochemistry and Health

June 2016, Volume 38, Issue 3, pp 703–712 | Cite as

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Original Paper
First Online: 29 July 2015

Abstract

In July 2012, two simultaneous wildfires burnt a big area in Valencia (Spain), where a birth cohort study (INMA) is being developed. The heavy smoke covered the whole INMA study area for several days. We aimed at evaluating the 2012 Valencia wildfire effects on the health of children enrolled in the INMA-Valencia cohort. Two weeks after the extinction of the wildfires, a phone survey was conducted and finally 460 individuals were enrolled. We considered a wildfire period (12-day interval when they were active) and a control period (12-day interval just before wildfires). Parents were asked about respiratory symptoms experienced during both periods, and during wildfires only about the preventive measures adopted and the perception of exposure, along with individual data collected through the different follow-up surveys of the cohort. Conditional logistic regression models were applied, and we

included interaction terms for asthma/rhinitis and level of perception of exposure; 82.4 % perceived smoke smell outdoors, 40 % indoors and more than 90 % of the families observed the presence of ash. An adjusted odds ratio of 3.11 [95 % confidence interval 1.62–5.97] for itchy/watery eyes and 3.02 [1.41–6.44] for sore throat was obtained. Significant interaction terms for rhinitis and asthma in itchy/watery eyes and sneezing, and only asthma for sore throat were obtained. Exposure to wildfire smoke was associated with increased respiratory symptoms in this child population, particularly affecting susceptible individuals with asthma or rhinitis.

Keywords

Wildfire Children Allergy Air pollution Cohort Asthma Rhinitis

Electronic supplementary material

The online version of this article (doi: 10.1007/s10653-015-9753-5 (https://doi.org/10.1007/s10653-015-9753-5)) contains supplementary material, which is available to authorized users.

Notes

Acknowledgments

Fondo de Investigación Sanitaria (FIS)-FEDER (Fondo Europeo de Desarrollo Regional) 06/1213, 07/0314, PS09/02647 FIS PI11/02591, FIS PI11/02038, and FIS PI13/02032.

Compliance with ethical standards

Conflict of interest

The authors declare that they have no actual or potential competing financial interests.

Supplementary material

10653_2015_9753_MOESM1_ESM.doc (52 kb)
Supplementary material 1 (DOC 52 kb)

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Cite this article as:
Vicedo-Cabrera, A.M., Esplugues, A., Iñíguez, C. et al. Environ Geochem Health (2016) 38: 703. https://doi.org/10.1007/s10653-015-9753-5

- Received 23 October 2014
- Accepted 22 July 2015
- First Online 29 July 2015
- DOI https://doi.org/10.1007/s10653-015-9753-5
- Publisher Name Springer Netherlands
- Print ISSN 0269-4042
- Online ISSN 1573-2983

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- Published in cooperation with

  Official Journal of theSociety for Environmental Geochemistry and Health

Personalised recommendations

Environment International, Vol. 17, pp. 117-134, 1991
Printed in the U.S.A.

0160-4120/91 $3.00 + .00

SMOKE EMISSIONS FROM WILDLAND FIRES

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EI 9007-172M (Received 18 July 1990; accepted 2 August 1990)

Biomass burning is a major source of emissions to the atmosphere. Some of these emissions may change global climate. This paper uses combustion efficiency as an independent variable for predicting emission factors for, among others, carbon monoxide, carbon dioxide, methane, and particulate matter. Other gases are correlated with the release of carbon monoxide. The release of nitrogen and sulfur-based compounds occurs in relation to their content in the biomass. The Sundance Fire is used to model the emissions from major fires that have occurred in the United States. Approximately 1 Tg of biomass was consumed by this fire, which released 0.019, 0.151, 1.545, and 0.007 Tg of particulate matter, carbon monoxide, carbon dioxide, and oxides of nitrogen, respectively. Other fires have released over 50 times this amount. Global emissions of various products of combustion are dependent on the combustion efficiency of the fires.

INTRODUCTION

Biomass burning is a major contributor of "greenhouse" gases and particulate matter to the atmosphere. The net effect on global climate is not well quantified, and there is a need for better source information regarding the total biomass consumed globally and the quantity and time of release of the important emissions. Radke (1989) estimates that, on a global scale, 10 Pg/y (Pg = Petagram, 10^{15} g) of biomass are consumed. This includes all forms of biomass consumption. Seiler and Crutzen (1980) estimated global biomass burning to contribute 2-3.3 Pg of carbon in the form of carbon dioxide to the atmosphere each year. Crutzen et al. (1985) estimated carbon monoxide emissions of 0.8 Pg/y from biomass burning. Hegg et al. (1989) estimated the production of several trace gases based on the ratio of trace gas to CO emission ratios. Estimates of biomass consumption based on chemical mass balance considerations may be lower than previously estimated because of new information on the efficiency of the combustion processes that produce CO_2 emissions. In this paper, we discuss factors that lead to the production of incompletely oxidized combustion products and the significance of these on the global production of emissions. A large wildfire, the Sundance Fire of 1967, is modeled and used to estimate emissions from other historically important, large wildfires. Models describing the rate of emission per unit mass of fuel consumed are presented as a function of combustion efficiency. Combustion efficiency is defined as the percentage of carbon released during

combustion of biomass fuels in the chemical form of carbon dioxide.

In the United States 1988 was one of the most extreme "fire years" in recent history. Wildfires burned 2 Mha of land, including 300 000 ha in Yellowstone National Park. If we consider an average fuel consumption of 45 Mg/ha, the total fuel consumed by wildfires in the United States in 1988 was 90 Tg (Tg = Teragram, 10^{12} g). In addition, Chi et al. (1979) estimated that prescribed fires burn an average of 36.6 Tg of biomass per year. The total wildland biomass fuel consumed by planned fires and wildfires in the United States represents less than 2% of the estimated global biomass consumption.

Historically, smoke from biomass fires has been a major societal problem. Only since about 1970 has information been developed describing the content of smoke. Interpreting the effects of smoke on health and radiation transfer through the atmosphere is continuing. Much of the research has focused on smoke production for prescribed fires, basically using three techniques:

1. Ground level measurements with instruments on towers or suspended in smoke from near full-scale prescribed fires (Ward and Hardy 1984; Ward et al. 1989a).

2. Airborne measurements by flying instrumented aircraft through the plumes of prescribed fires of different fuel loadings (Radke et al. 1990).

3. Modeled fuels burned in controlled environment combustion laboratory facilities (Ward 1989).

The few direct measurements of smoke emissions from wildfires were made in conjunction with an examination of the assumptions on which the nuclear winter hypothesis was based (Turco et al. 1984 1990; Crutzen and Birks 1982; Radke et al. 1988; Hegg et al. 1989). This paper summarizes existing data and applies the information to a well-documented wildfire. The model for the wildfire is used to estimate emissions for other historical fires and to provide new concepts regarding global emissions from biomass fires.

Large biomass fires, historically, have been a major source of smoke emissions to the atmosphere. Plummer (1912) describes in detail smoke phenomena in emphasizing that forest fires are tremendously damaging economically. We quote here from the observations of Plummer (1912):

"A thrifty forest purifies the air we breathe, and it is an irony of nature that when it goes up in smoke it causes a pollution of the atmosphere. The mischief thus caused is by no means trivial, since a heavy pall of smoke interrupts business, interferes with navigation, and, turning [day] into [night], compels the use of artificial light. Such conditions have [occurred] over an expanse of many thousands of miles, and the actual loss must be very great. In the vicinity of a great fire the atmosphere sometimes carries ashes and burning brands to a distance of several miles....Forest fires are the most frequent cause of widespread pollution of the atmosphere, and the volume of the pollution is exceeded only in the case of violent volcanic eruptions....A large forest fire has an appreciable effect upon the surrounding atmosphere, causing a movement of the air toward the fire. This effect is quite local, and is overbalanced if there is a strong wind blowing, which will drive the fire before it....During the great forest and city fire at Fernie, British Columbia, August 1-8, 1908, which was accompanied by a high wind, flaming trees, timbers, lumber, and sections of buildings were carried. This fire burned a strip 3 miles wide for a distance of about 20 miles. During the great Idaho fire of August 20-22, 1910, the same phenomena were observed....The tendency is for smoke to spread out and to be dissipated, but if the volume is great it may be identified for hundreds of miles, even when the cause of it is unknown....These phenomena, observed from time immemorial, have been known by various names—in this country as dark days, dry fogs, Indian summers, and colored rains."

During the severe fire year of 1987, valleys in northern California and southern Oregon were "smoked in" for weeks, causing anomalous temperature depressions of up to 20°C (Robok 1988). He reported more than 400 people per day were treated for respiratory problems. Tomato plants in Happy Camp died. In 1988, fires burned for four months in Yellowstone National Park. Severe local air pollution problems existed. Wildland firefighters made about 12 000 medical visits because of respiratory problems during the four months (Ward et al. 1989b). Fire-related phenomena are being investigated. For example, one large-scale research fire in the Province of Ontario, Canada, produced a smoke cloud that had numerous discharges of lightning, over 50 mm of precipitation, and washout of a significant fraction of the suspended smoke. Smoke-related phenomena of historical time are now beginning to be understood scientifically.

FIRE PROCESSES

Within the perimeter of an advancing fire, different combustion processes of flaming, smoldering, and glowing compete for available fuel and are markedly different phenomena that contribute, in part, to the diversity of combustion products. The fuel characteristics (including arrangement, size distribution, moisture, and chemical composition) affect the dura-

tion of the flaming, smoldering, and glowing combustion phases.

Flaming and smoldering are distinct combustion processes that not only appear different, but involve different chemical reactions. Flaming combustion dominates during start-up, with the fine fuels and surface materials supplying the volatile fuel required for the rapid oxidation reactions to be sustained in flaming. The heat from the flame structure and the diffusion and turbulent mixing of oxygen at the surface of the solid fuel contribute to the heat required to sustain the pyrolysis processes. Early in the flaming phase, the volatile hydrocarbons are vaporized from the fuels. Later the cellulosic and lignin-containing cellular materials decompose through pyrolysis. These processes produce the fuel gases that sustain the visible flaming processes.

Once carbon begins to build up on the solid fuel surfaces, the pyrolytic reactions no longer produce sufficient fuel gases to maintain the flame envelope. For combustion to continue, oxygen must diffuse to the surface of the fuel. Diffusion of oxygen and the availability of oxygen at the fuel surface is enhanced through turbulence in the combustion zone and through premixing by introducing the oxygen at ground level. This allows oxidation to take place at the solid fuel surface and provides for heat evolution and heat feedback to accelerate the pyrolytic reactions and volatilization of the fuel gases from the solid fuel. The process ultimately leads to the production of charcoal, where the only combustion occurring is of the glowing type—a surface reaction of oxygen with carbon.

The Sundance Fire was a typical high-intensity wildfire with all of the combustion processes described above taking place simultaneously and on a large scale (Anderson 1968). Flame lengths undoubtedly exceeded 50 m. Fire-induced winds would have exceeded 40 m/s coupled with strong updrafts estimated to be as high as 40 m/s. The resultant winds would have produced phenomenal turbulence within the combustion zone and within the lower troposphere. Large pieces of partially consumed fuel particles were carried long distances and deposited ahead of the main fire front. Major pockets of unburned hydrocarbon gases undoubtedly exploded periodically above the main fire front. No research has yet examined emissions from such a violent fire. However, inferences of emissions can be made from data now available.

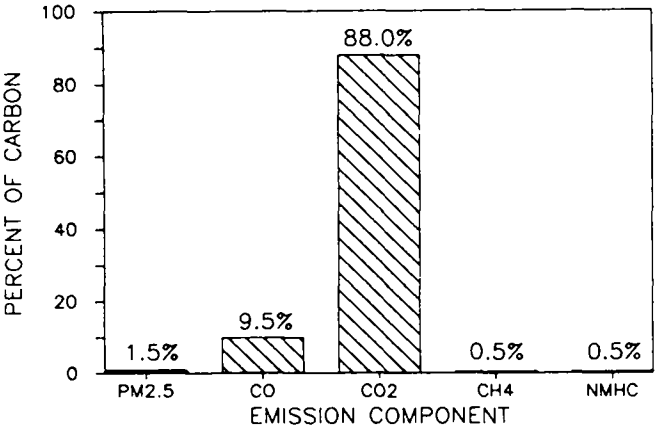

Fig. 1. The average percentage of total carbon released by biomass burning in the United States in the form of CO, CO_2, and hydrocarbons. PM2.5 is particulate matter less than 2.5 μm diameter.

SMOKE PRODUCTION

The smoldering combustion phase produces high emissions of particulate matter and CO. Fires of low intensity (those in which the flaming combustion phase is barely sustained) produce high emissions of particulate matter. The formation of particulate matter results primarily from two processes: (1) the agglomeration of condensed hydrocarbon and tar materials, and (2) mechanical processes that entrain fragments of vegetation and ash.

Release of carbon

When biomass fuels are burned, carbon is released in the form of CO_2, CO, CH_4, hydrocarbons, particulate matter, and other substances in decreasing abun-

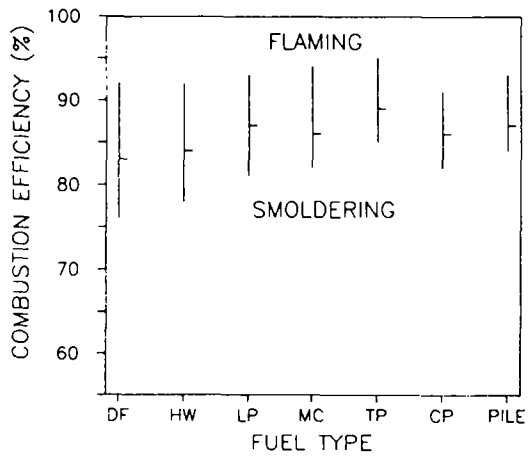

Fig. 2. Combustion efficiency for seven fuel types for flaming, smoldering, and weighted average. (DF-Douglas-fir, HW-Hardwood, LP-Ponderosa pine and lodgepole pine [long-needled conifers], MC-Mixed conifer, TP-Tractor piled, CP-Crane piles, and Pile-Combined TP and CP.).

dance (Fig. 1). A carbon mass-balance procedure is frequently used to characterize the fuel consumed in producing the emissions measured (Radke et al. 1990; Ward and Hardy 1984; Ward et al. 1979). The sum of CO_2 and CO accounts for more than 95% of the carbon released during the combustion of biomass. Combustion efficiency is calculated from the measured concentrations of carbon-containing gases and particles above background released from the combustion of biomass fuels. The combustion efficiency is never 100% for biomass burned in the open environment and generally ranges from 50 to 95%. Generally, the combustion efficiency is lowest for the smoldering combustion phase and highest for those fires with good ventilation and vigorous flame action. Combustion efficiency is illustrated in Fig. 2 for seven fuel types tested in the Western United States. In this paper, combustion efficiency is used as the independent variable for modeling the rate of release of emissions.

Formation of particles

Forest fires are a complex form of the diffusion flame process where pyrolysis of solid fuels produces fuel gases that interdiffuse with oxygen from the atmosphere. As the interdiffusion of fuel and oxygen develops and intensifies, the flame characteristics and the chemical processes occurring in the flame zone change. It is highly probable that, for the Sundance Fire, a level of fire intensity was above that associated with optimal combustion efficiency. Under these extreme fire intensity conditions, fuel may no longer pass through an active oxidation zone. At times, even in lower intensity fires, pockets of unburned, partially oxidized gaseous fuels escape the combustion zone or undergo delayed ignition. The influence of flame turbulence on combustion efficiency is not fully understood. However, as the intensity of the fire increases and the zone of complete mixing of gaseous fuel and oxygen moves farther from the solid fuel, combustion efficiency is believed to decrease and the abundance of the products of incomplete combustion to increase.

Because of the increased depth and height of the flame zone, heading fires and area fires create an extended reducing environment in which continued pyrolysis and synthesis of hydrocarbon gases and fragmented particles can occur under conditions of reduced oxygen content. (A "heading fire" moves with the wind. An "area fire" may result from the ignition of many point fires within a sizable area, and these point fires joining together forming a large region totally involved in flaming combustion.) In addition, heat is reradiated from the particles to the atmosphere, which can slow down the reactions as the unburned gases and particles are convected away from the active combustion zone. If the temperature in the interior of the flame zone is appropriate (<800°C), rapid formation of particles and accretion of carbonaceous organic particles will occur. Consumption of the particles requires prolonged exposure at high temperatures (>800°C) in a zone with near ambient (21%) concentration of oxygen (Glassman 1977).

Mass-fire experiments performed in Canada during 1988 and 1989 demonstrated the important effect of oxygen deficiency on flame structure and on emissions production (Susott et al. in press). The pulsation phenomenon often observed for large fires is thought to be closely coupled to oxygen deficiency. Oxidation of the particles depends partly on the degree of premixing of pyrolyzed fuel and oxygen that takes place in the zone of active solid fuel pyrolysis. Greater premixing results in production of less particulate matter.

Particle number and volume distribution

The size and content of smoke particles have significant health implications. Small diameter particles (fine particles less than 2.5 μm in diameter) may be drawn deep into the human lung and are defined as the respirable fraction. The respirable fraction contains particles of a diameter that also have a maximum effect on visibility and radiation transfer in the atmosphere. The concentration of smoke particles by diameter classes has been measured using sophisticated instruments aboard aircraft to cover the broad distribution of particle sizes from 0.01 μm to 43 μm (Radke et al. 1990). The results suggest a pronounced number concentration peak at a diameter of 0.15 μm. The volume distribution that, for a first approximation, represents the mass distribution was found to be bimodal with peaks at 0.5 μm and greater than 43 μm (Fig. 3).

Ward and Hardy (1984) measured a large difference in emission factors for particles of the respirable size range (PM2.5) as compared to particles without regard to size (PM). This difference increased proportionally to an increase in the rate of heat release on an area basis (Fig. 4). They noted a slight decrease in emission factors for PM2.5 with an increase in PM emission factors over the range of rates of heat release tested. Radke et al. (1988) noted a similar increase in PM emission factors and concurred that this increase probably results from an increased level of turbulence in the combustion zone.

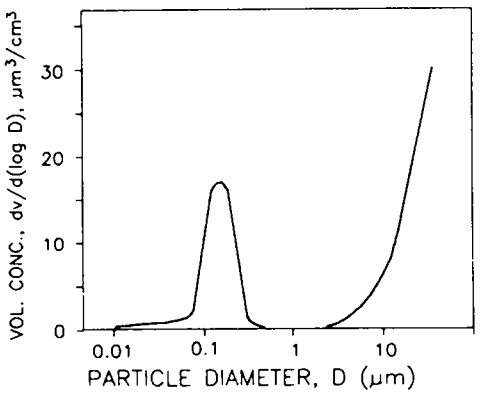

Fig. 3. Volume by particle size fractions measured for prescribed fires of logging slash in the Western United States from an airborne sampling platform (Radke et al. 1990).

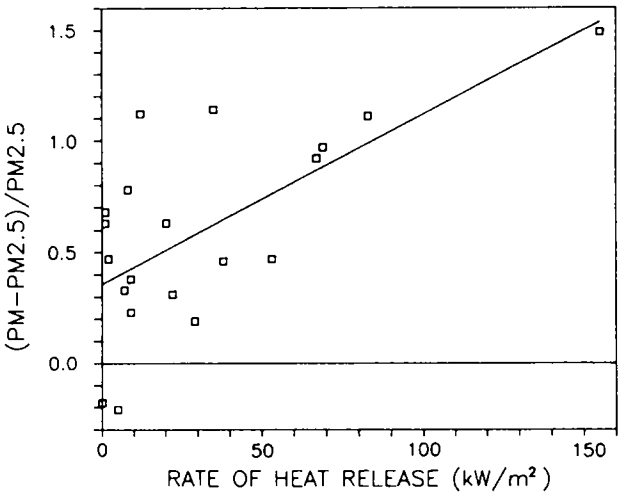

Fig. 4. The ratio of differences between emission factors for total particulate matter and particulate matter less than 2.5 μm as a function of the rate of heat release.

Emission factors for particulate matter

Ward et al. (1988) summarized those emission factor data available for different fuel types by region within the United States. For chaparral fuels in the Southwest, palmetto gallberry of the Southeast, and possibly sagebrush of the Intermountain West, similar PM emission factors of about 15 g/kg can be used. The emission factors for PM for long-needled conifer litter fuels burned with backing and heading fires range from 20-50 g/kg of fuel consumed, respectively. Fires in cured grass generally have lower emission factors of 10 g/kg of fuel consumed. The emission factors are quite similar for broadcast burns of logging slash, regardless of species, ranging from 11-13 g/kg, 12-14 g/kg, and 18-20 g/kg of fuel consumed for PM2.5, PM10, and PM, respectively. Emission factors for piled logging slash with no soil incorporated in the pile are 4, 4, and 6 g/kg of fuel consumed for PM2.5, PM10, and PM, respectively. For piled slash, when the pile contains 35% organic soil mixed with the woody fuel, the emission factors range up to 35 g/kg of fuel consumed.

Although many measurements of the concentration of emissions have been made, ancillary data regarding fuel and fire conditions associated with the production of the measured emissions are often lacking. Airborne sampling of emissions from fires is often poorly supported by ground truth observations or measurements. Generally, investigators recognize the combustion efficiency differences between flaming and smoldering combustion phases. But the composite samples taken using airborne systems have seldom been effective differentiating either combustion phase. For many fuel types, emissions from the smoldering phase overwhelm emissions produced through flaming combustion processes—typical of measurements of smoke from wildfires and during the later stages of prescribed fires.

The emissions sampling system of Ward and Hardy (1984) was used to measure PM and PM2.5 emission factors for several different fuel types in Washington, Oregon (Ward et al. 1989a), and California (Ward and Hardy 1989). The data for PM and PM2.5 are plotted as a function of combustion efficiency in Fig. 5. Regression models indicate a 35% increase in the PM emission factors over the emission factors for PM2.5 for the same levels of % combustion efficiency (CE). For PM and PM2.5, the regression equations are:

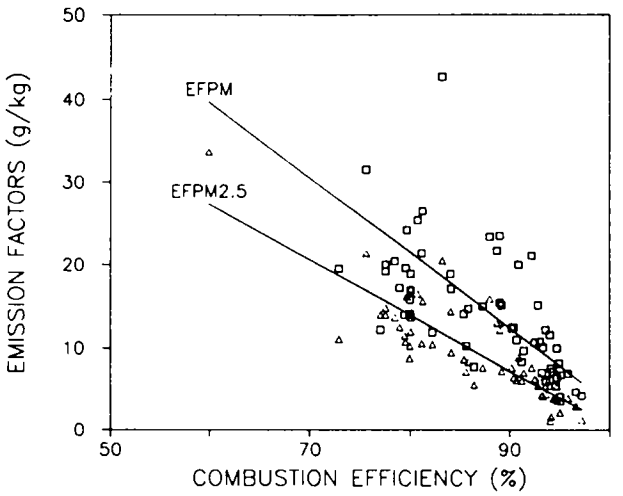

Fig. 5. Emission factors for total particulate matter (PM) and for particles less than 2.5 μm diameter (PM2.5) as functions of combustion efficiency.

$$EFPM = 93.3 - 90.5*CE, \quad R^2 = 0.54; \quad (1)$$

and

$$EFPM2.5 = 67.4 - 66.8*CE, \quad R^2 = 0.74. \quad (2)$$

Standard error of the estimate values for the regression lines are ±3.0 g/kg and ±6.3 g/kg for Equations 1 and 2, respectively. The standard error of the estimate values illustrate the variance that has not been explained due to combustion efficiency. Combustion efficiency is defined in the previous section, "Release of Carbon." As combustion efficiency decreases, the emission factors for PM and PM2.5 increase. The PM and PM2.5 models are used in a later section to compute the source strength for the Sundance Fire.

Hegg et al. (1989) found nearly identical results for a single fire sampled in Canada. Their plot of CO and CO_2 ratios are converted to combustion efficiency and used as the independent variable. The slope of the regression line (EFPM = 108.6-108.0*CE, R^2 = 0.71) is similar to Equation 1. Data were collected during a pilot study in Brazil of one savanna-like (cerrado area) and two tropical deforestation fires (Ward et al. 1990). These fires show high values of combustion efficiency ranging from 92-97% and the emission factors for PM less than 5.0 µm diameter ranged from 4-7 g/kg. Both sets of data superimpose nicely on Fig. 5.

Other factors such as rate of heat release have a pronounced effect on the size and mass of particles produced. Generalized models are needed, based on factors affecting fire spread, fuel consumption, and combustion efficiency for predicting the production of smoke.

The results of Ward and Hardy (1984) demonstrated for a number of fuel types that (1) emissions of particulate matter range over a factor of 10 depending on fire and fuel conditions that affect combustion efficiency; (2) brushy areas produce the most smoke per ton of fuel consumed and have higher rates of production of benzo[a]pyrene than non-brushy areas; (3) fires of higher intensity (long flame lengths) produce proportionately larger particles than are found in low-intensity and smoldering combustion fires; (4) CO is abundantly produced from open fires and, generally, on a mass basis exceeds the production of particles by a factor of 10; (5) hydrocarbon gases are a small part of the total amount of carbon released from the combustion of forest fuels; and (6) emission factors for particles released from fires tend to increase inversely to combustion efficiency (Ward et al. 1989a).

Emissions of trace elements

The trace elements for samples of PM2.5 are shown in Fig. 6 as a percentage of the PM2.5 by combustion phase and weighted for the entire fire. All the samples of the trace elements are from broadcast burns of logging slash from coniferous species. The sodium component is especially high for the flaming phase; nearly 2.75% of the PM2.5 is sodium.

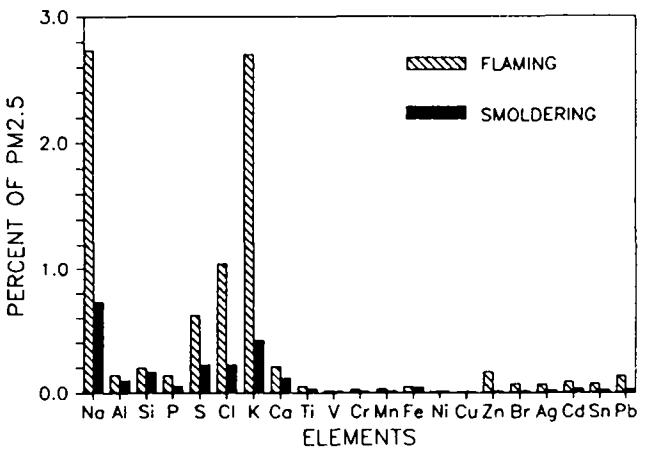

Fig. 6. Percentage composition of particulate matter less than 2.5 µm diameter in smoke from logging slash fires in the Western United States.

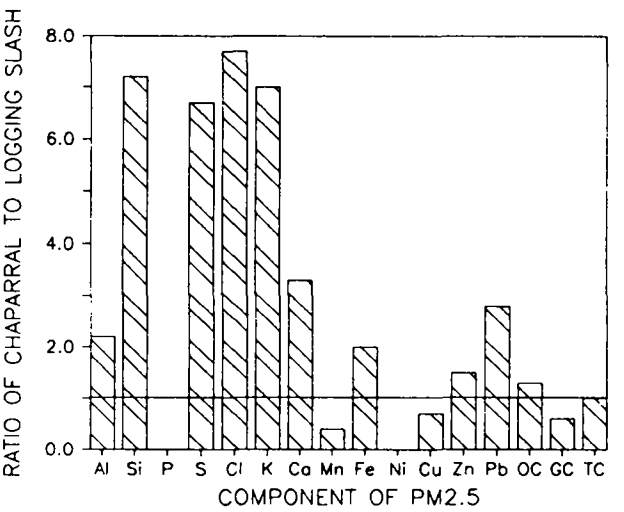

Fig. 7. Ratio of trace element content for smoke from logging slash and chaparral fires in the Western United States.

The sodium, sulfur, chlorine, and potassium contents of PM2.5 are high during the higher temperature flaming phase of the fire. Generally, as the combustion efficiency increases, more of the carbon is consumed, thus increasing the percentage of mass reported as trace elements. Ward and Hardy (1984) found that the sum of sulfur, chlorine, and potassium (S + Cl + K) is correlated with the rate of heat release (r = 0.92). Iron released with the PM2.5 is slightly greater during the latter periods of the smoldering phase than during the flaming phase or first part of the smoldering phase. The large difference in potassium content in the combustion phase leads to accentuating the potassium-to-carbon ratio differences by combustion phase. Potassium was released proportional to the rate of heat release.

Differences in emissions of trace elements were noted as a function of fuel type by Ward and Hardy (1988; 1989). The average values for trace materials produced with the PM2.5 during the flaming phase were generally higher for the chaparral fires of California than for the logging slash broadcast fires of the Washington and Oregon areas (Fig. 7). The production of sulfur, chlorine, and potassium for the chaparral fires was an order of magnitude larger than for the slash fires. Fire intensity was much higher for some of the chaparral test fires than for the logging slash fires. The fire intensity ranged up to a maximum rate of heat release on a square meter basis of nearly 3 MW—or nearly an order of magnitude larger than for the logging slash fires. Emissions of lead from fires in southern California were high relative to fires in the Pacific Northwest. We currently do not have adequate information to separate the effects of the rate of heat release and fuel chemistry in the prediction of the content of particles. It is generally accepted that the trace elements are released in the highest proportion to the carbon contained with the particles for the highest intensity fires. However, the lead content may be higher for the California fires because of a higher deposition rate in the California area from sources outside the forest environment.

Emissions of graphitic and organic carbon

Emissions of graphitic carbon are especially important because of the contribution to the absorption of light. Because the absorption by the smoke emissions is due primarily to graphitic carbon, the specific absorption coefficient correlates well with the graphitic carbon content of the aerosol (Patterson et al. 1986). Emission factors for graphitic carbon were found to range from 0.46-1.18 g/kg of fuel consumed for logging slash of the Pacific Northwest. In tests of pine needle (slash pine) fires in a controlled environment combustion laboratory, emission factors were measured as high as 5.40 g/kg of fuel consumed. The results suggest an inverse correlation between specific absorption and emission factors. This is in agreement with the inverse correlation of the rate of heat release with the percentage graphitic carbon content reported by Ward and Hardy (1984). Generally, emission factors for PM2.5 have been found to be lower for higher intensity fires.

Organic carbon content of particulate matter is especially important because of the types of organic compounds associated with the particles. The polynuclear organic material is contained as a fraction of the organic carbon content of the particles, and contains the important class of compounds known as polynuclear aromatic hydrocarbons—some of which are known to have carcinogenic properties. The carbon fraction of the organic content of particulate matter ranges between 30 and 60%. Benzo[a]pyrene is the most studied of the compounds contained in this fraction. Emission ratios were found to range from 2-274 μg/g of particulate matter for heading and backing fires, respectively (McMahon and Tsoukalas 1978). Measured ratios of benzo[a]pyrene to particulate matter were reported in the range of 0.4-222 μg/g of particulate matter for fires in coniferous species logging slash in the Western United States (Ward 1989). The highest values occur for the smoldering combustion phase and lowest for the flaming combustion phase for the highest intensity fires.

Emissions of CO and other trace gases

CO is the second most abundant carbon-containing gas produced during the combustion of biomass (Fig. 1). Combustion efficiency is highly correlated with the ratio of the production of CO relative to CO_2 (Fig. 8). Ward (1989) found particulate matter concentration to be correlated with CO concentration (r = 0.89). Reinhardt (1989) found the concentration of formaldehyde to be correlated with the concentration of CO (r = 0.93). Generally, emission factors for CO on a mass basis are 10 times greater than for the fine particle fraction. Emission factors for CO range from 60 g/kg to over 300 g/kg of fuel consumed.

During several days of sampling haze layers in the Amazon region of Brazil using airborne and real time sampling techniques, Andreae et al. (1988) found a molar ratio of elevated CO to elevated CO_2 of 0.085. This ratio was used for calculating emissions of other materials. Their CO to CO_2 ratio gives a calculated

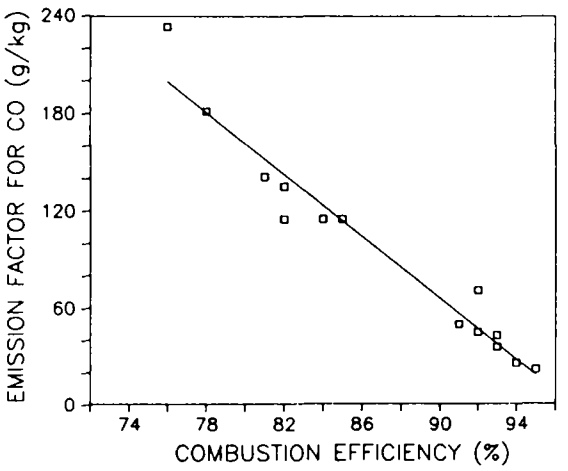

Fig. 8. Functional relation for CO and combustion efficiency.

emission factor for CO of 87 g/kg of fuel consumed. Also working in Brazil, Crutzen et al. (1985) measured concentrations of CO, CO_2, and selected hydrocarbons using mostly grab-sampling, ground-based sampling techniques. The calculated range of emission factors for CO for these measurements ranged from 167-209 g/kg. This is generally higher than the fire-weighted average emission factors for logging slash fires in the Western United States of 171 ±42 g/kg. Hegg et al. (1989) reported emission factors for CO of 91 ±21 g/kg of fuel consumed. Their measurements were for several fuel and fire types on the West Coast of the United States and in the Province of Ontario, Canada. These differences may be a result of either vegetation or moisture content differences, or both.

The large differences between Hegg et al. (1989), Ward et al. (1989a), Andreae et al. (1988), and Crutzen et al. (1985) cannot be resolved through combustion efficiency arguments and differences in fuel and fire complexes. Airborne sampling of emissions from biomass fires often involves measurements of emissions that are a few µL/L above the background concentration. The background concentration of CO_2 can vary dependent on time of day, solar insolation, mixing in the lower boundary layer, and the location of the experiment relative to urban sources. In addition, airborne samples may include a disproportionate quantity of emissions from the flaming combustion phase because the plume is generally much more buoyant during the times of maximum rates of heat release. On the other hand, measurements of emissions from fires taken a few meters above the flames may not allow enough time for adequate quenching.

Table 1. Mean values of emission factors for compound x divided by the associated emission factor for CO as presented by Hegg et al. (1989).

Compound x	EFx/EFCO
O_3	0.060 +/- 0.05
NH_3	0.014 +/- 0.008
CH_4	0.031 +/- 0.003
C_3H_6	0.006 +/- 0.001
C_2H_6	0.005 +/- 0.002
C_3H_8	0.003 +/- 0.001
C_2H_2	0.003 +/- 0.001
N_2H_4	0.002 +/- 0.002
N_2O	0.004 +/- 0.001
NO_x	0.070 +/- 0.040

Despite questions concerning the representativeness of the measurements of CO emission factors, the airborne measurements are the most extensive set of data available today, and the supporting measurements of trace gases expressed as a ratio to CO are equally valuable. The emission factor ratios of Hegg et al. (1989) are listed in Table 1 and will be used in the projections for the Sundance Fire, United States' contribution to the global budget of trace gases, and revised projections for the global emissions.

Of critical importance are the correlations of the concentration of other combustion products with the concentration of CO. Ward et al. (1989a) cross-correlated emissions data from near full-scale experimental fires for six fuel types and found highly significant positive correlation coefficients between CO and the following emissions: PM (0.80), PM2.5 (0.84), CO_2 (0.63), CH_4 (0.88), and nonmethane hydrocarbons (0.79). This suggests that the production of CH_4 and possibly other low-molecular weight hydrocarbons can be scaled to the production of CO as listed in Table 1. In addition, Ward et al. (1989a) found combustion efficiency cross correlation coefficients to be highly significant with emission factors for the compounds: EFPM (-0.73), EFPM2.5 (-0.83), EFCO (-0.97), $EFCH_4$ (-0.75), EFNMHC (-0.62), and $EFCO_2$. The regression models as a function of combustion efficiency (CE) are:

$$EFCH_4 = 42.7 - 43.2*(CE), \quad R^2 = 0.77; \tag{3}$$

$$EFCO = 961 - 984*(CE), \quad R^2 = 0.95; \tag{4}$$

and

$$EFCO_2 = 1833*(CE). \tag{5}$$

The algorithm for computing $EFCO_2$ values is derived from the definition of combustion efficiency and the chemical composition of biomass ($C_6H_9O_4$). The ratio $EFCH_4/EFCO$, calculated for the regression equations 3 and 4, ranges from 0.046 at 75% combustion efficiency to 0.065 at 95% combustion efficiency. This compares with the ratio of Hegg et al. (1989) in Table 1 of 0.031 and the range of values presented by Cofer et al. (1989) of 0.040-0.068 for combustion efficiencies ranging from 87-91%.

The same argument is much less convincing for the nitrogen-based species listed in Table 1. In general, NO_x varies proportionally to the nitrogen content of the fuel (Clements and McMahon 1980). The ozone concentration may only be remotely related to either NO_x or the concentration of reactive hydrocarbons and may be more closely coupled with the level of insolation receipt (Evans et al. 1977). Andreae et al. (1988) extensively studied haze layers over the Amazon region of Brazil and found ozone production contributed significantly to the regional ozone budget during the dry season when most of the burning occurs.

THE SUNDANCE FIRE: A CASE EXAMPLE

The Sundance Fire is used as an example to calculate source strength functions for the various emissions, not because it was an extremely large fire (reaching a maximum size of 22 626 ha), but because the fire growth and fuels contributing to the main fire front and the subsequent smoldering zone were well-quantified. In addition, the Sundance Fire is typical of fires that have a high rate of growth during one diurnal period. As with most wildfires, there is little opportunity to quantify the fuels prior to the fire. Therefore, the fuel consumption must be reconstructed by knowing the forest type; sampling fuels from adjacent unburned areas of similar vegetation, composition, and disturbance history; and from interviews with fire management personnel with a familiarity of the fire and the site. The Sundance Fire was well-documented by a team of scientists from the Intermountain Fire Sciences Laboratory in Missoula, Montana (Anderson 1968).

The fire burned during a time of extreme drought coupled with low humidity, high temperature, and high wind speeds. The net result was a fire that moved rapidly, covering 25.7 km between 1400 and 2300 h on September 1, 1967, with a convection column that reached 10.3 km into the atmosphere. The fire burned through mixed conifer forests interspersed with logged areas. It crowned through the young and overmature stands equally and with little regard for stand density. The crown fuel consumption ranged from 0.46-1.17 kg/m^2 with the brush consumption averaging 0.61 kg/m^2. The ground litter and duff consumption ranged from 0.22-4.48 kg/m^2.

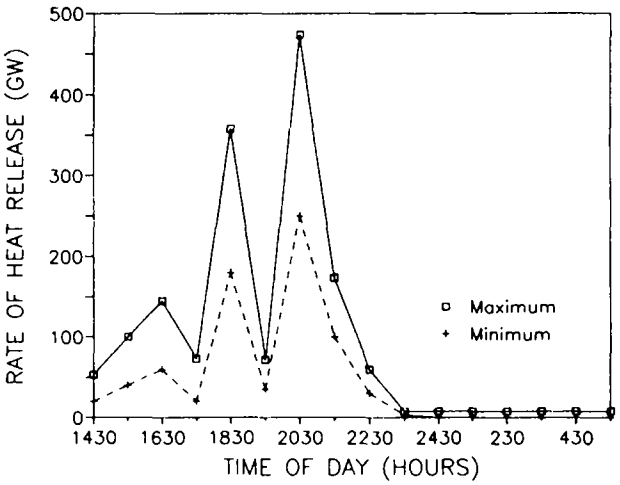

Fig. 9. Maximum and minimum rates of heat release for the main fire front of the Sundance Fire, September 1-2, 1967 (Anderson 1968).

Total maximum and minimum rates of heat release for the advancing fire front were estimated by Anderson (1968) for each hourly increment of time (Fig. 9) dependent on the available fuel as well as intelligence reports of the location of the fire perimeter. The maximum rate of heat release of 500 GW occurred at about 2000 h with estimated wind speeds of 20 m/s from the southwest. This period coincides with the time of maximum expected rate of release of emissions.

This paper uses the estimates of maximum rates of heat release for the main fire front to calculate the fuel consumption for three fire environments: (1) for the main fire front, (2) for the area of secondary flaming (not linked with the main fire front), and (3) for the area immediately following the flaming combustion where fuel was consumed by the smoldering combustion process. The available data are used in the following way to compute the rate of fuel consumption:

(1) The assumption was made (Anderson 1968) that all of the crown, brush, and 20% of the ground fuel (litter and decomposed plant parts) were consumed through the flaming combustion process (except for a period from 1700 to 1800 h discussed below). Only a portion of the heat release from this fuel contributed to the fire front. These estimates and the proportional breakdown between the fuel con-

sumption that occurred in the active fire front and the secondary flaming zones are presented in Table 2. Anderson (1968) provided maximum and minimum fuel loadings for the ground and crown fuel components. Average values were used in the calculations: 0.82, 0.61, and 2.35 kg/m^2 for the crown, brush, and ground fuel consumption, respectively.

(2) Values of percentage consumption for each of the three classes of fuels were used in developing a weighted heat release for each hour of the fire (Table 2). This hourly value was divided into the total maximum heat release for the flame front (Fig. 9) to calculate the fuel consumption for the active flame front.

(3) The balance of the fuels consumed in the zones of secondary flaming combustion was used to calculate a ratio of fuels consumed between the secondary and active fire fronts (Table 3). This ratio was multiplied by the fuel consumption per m^2 for the active fire front to compute the total fuel consumption on a per-m^2 basis in the zone of secondary flaming combustion.

(4) The smoldering combustion process was assumed to have consumed 80% of the ground fuel except for one period from 1700 to 1800 h, when 30% of the ground fuel contributed to the heat entering the convection column from the main fire front. Table 3 shows the ratio of smoldering to flaming by hourly increments. These ratios were multiplied by the total flaming fuel consumption to calculate the total mass of fuel consumed (on a per m^2 basis) through smoldering combustion.

(5) Smoldering combustion was assumed to reach a maximum rate of consumption immediately following the flame front passage and to die exponentially over a 12-h period (Ward and Hardy 1984). The smoldering fuel consumption was computed using the following equation:

$$W_s = (1 - FP)\,(2.35\ \text{kg/m}^2)\,(1 - \text{EXP}\,(-T/t)) \qquad (6)$$

where

FP = percentage consumption during flaming phase,

T = time since ignition, hours,

t = decay time to consume 63% of total, 1 h.

As a result, 99.8% of the smoldering combustion fuel consumption occurred in the first 8 h following ignition. Equation 6 uses a longer decay constant than that measured by Ward and Hardy (1984) for the

Table 2. Fuel consumption data for Sundance Fire (Anderson 1968) and the calculation of the weighted heat release based on the fuel consumption by type.

Time	Main fire front fuel consumption			Main fire front fuel consumption			Total primary flaming	Weighted heat release
	Ground	Brush	Crown	Ground	Brush	Crown		
	-(dimensionless)-			-------(kg/m^2)----------				(kJ/g)
1430	0.20	0.75	0.10	0.471	0.454	0.082	1.007	17.73
1530	0.20	0.90	0.40	0.471	0.545	0.327	1.343	17.38
1630	0.10	0.95	0.80	0.235	0.575	0.654	1.464	16.95
1730	0.30	0.85	0.20	0.706	0.515	0.163	1.384	17.64
1830	0.10	0.90	0.95	0.235	0.545	0.776	1.557	16.84
1930	0.06	0.95	0.60	0.141	0.575	0.490	1.207	17.05
2030	0.06	0.60	0.95	0.141	0.363	0.776	1.281	16.61
2130	0.04	0.70	0.80	0.094	0.424	0.654	1.172	16.72
2230	0.06	0.80	0.70	0.141	0.484	0.572	1.198	16.89
2330	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
2430	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
0130	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
0230	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
0330	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
0430	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
0530	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
0630	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
0730	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
0830	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
0930	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
1030	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
1130	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
1230	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95
1330	0.06	0.60	0.50	0.141	0.363	0.409	0.913	16.95

Table 3. Fuel consumption by type for the Sundance Fire (Anderson 1968) and the multiplier for calculating fuel consumption for the secondary flaming and smoldering combustion zones.

Time	Secondary flaming fuel consumption				Factor for secondary flaming	Factor for smoldering
	Ground	Brush	Crown	Total		
	-------(kg/m^2)----------				(Dimensionless	
1430	0.000	0.151	0.734	0.886	0.880	0.995
1530	0.000	0.061	0.490	0.550	0.410	0.995
1630	0.235	0.030	0.163	0.429	0.293	0.995
1730	0.000	0.091	0.653	0.744	0.537	0.774
1830	0.235	0.061	0.041	0.337	0.216	0.995
1930	0.330	0.030	0.326	0.686	0.569	0.995
2030	0.330	0.242	0.041	0.613	0.478	0.995
2130	0.377	0.182	0.163	0.721	0.616	0.995
2230	0.330	0.121	0.245	0.695	0.581	0.995
2330	0.330	0.242	0.408	0.980	1.073	0.995
2430	0.330	0.242	0.408	0.980	1.073	0.995
0130	0.330	0.242	0.408	0.980	1.073	0.995
0230	0.330	0.242	0.408	0.980	1.073	0.995
0330	0.330	0.242	0.408	0.980	1.073	0.995
0430	0.330	0.242	0.408	0.980	1.073	0.995
0530	0.330	0.242	0.408	0.980	1.073	0.995
0630	0.330	0.242	0.408	0.980	1.073	0.995
0730	0.330	0.242	0.408	0.980	1.073	0.995
0830	0.330	0.242	0.408	0.980	1.073	0.995
0930	0.330	0.242	0.408	0.980	1.073	0.995
1030	0.330	0.242	0.408	0.980	1.073	0.995
1130	0.330	0.242	0.408	0.980	1.073	0.995
1230	0.330	0.242	0.408	0.980	1.073	0.995
1330	0.330	0.242	0.408	0.980	1.073	0.995

diedown phase of broadcast burns of logging slash. The reasoning is that the fire severity was much greater than normally encountered when using prescribed fire. Generally, as the fuel moisture content declines below about 35%, on an oven-dry weight basis, the ground fuels are consumed independently of the woody fuels and litter (Sandberg and Ottmar 1983). In this case, the forest floor fuel moisture content ranged from 8-20%.

(6) The ratio of smoldering to flaming fuel consumption as distributed using the exponential decay function (Equation 6) is used to generate Table 4.

(7) Hourly incremental fuel consumption for the flame front, secondary flame area, and smoldering, along with the cumulative fuel consumption for the three fuel consumption categories are presented in Table 5. In particular, the maximum rate of fuel consumption occurred between 2000 and 2100 h and reached a rate of 0.28 Tg/h. The total fuel consumption for the Sundance Fire is estimated to be 1.02 Tg. This is about 1% of the total biomass consumed annually within the United States by all wildland biomass fires.

Source strength calculations for the sundance fire

To calculate source strength for a fire, the rate of fuel consumption must be known (Table 5) along with the appropriate emission factors (Fig. 10) for the gases and particles.

For the Sundance Fire, about 50% of the mass of the fuel was consumed through smoldering combustion. This affected the types and quantity of emissions produced as was discussed previously in the section on "Smoke Production." The rate of consumption of fuels involved in smoldering combustion peaks almost immediately following the flaming combustion period, and then dies out at a rate approximating an exponential decay function, with the time constant being dependent on the dryness of the compact fuel layers and the depth of the duff layer (Ward and Hardy 1984).

The combustion efficiency for the Sundance Fire is thought to be similar to that for many broadcast prescribed burns of logging slash. For prescribed burns of logging slash from harvesting of Douglas-fir/western hemlock forests, the combustion efficiency ranges from an average of 77% for smoldering com-

Table 4. Distribution of fuel consumption for the smoldering combustion component. Note that the exponential decay function (text, Equation 6) is used to distribute the consumption for the smoldering combustion phase based on the total fuel consumed through the flaming combustion process.

Time	Smoldering combustion fuel consumption by hourly areas (elapsed time, hours)												Total for hour
	1	2	3	4	5	6	7	8	9	10	<'''>	16	
	(kg/hour)												
1430	13421												13421
1530	4937	19366											24303
1630	1816	7124	26401										35341
1730	668	2621	9712	15188									28189
1830	246	964	3573	5587	61737								72107
1930	90.4	355	1314	2055	22712	15820							42347
2030	33.3	130	484	756	8355	5820	100728						116307
2130	12.2	48.0	178	278	3074	2141	37056	40153					82940
2230	4.5	17.7	65.4	102	1131	788	13632	14772	13407				43919
2330	1.7	6.5	24.1	37.6	416	290	5015	5434	4932	2277			18434
2430	0.6	2.4	8.9	13.8	153	107	1845	1999	1814	838	2277		9059
0130	0.2	0.9	3.3	5.1	56.3	39.2	679	735	668	308	838...>		5610
0230		0.3	1.2	1.9	20.7	14.4	250	271	246	113	308...>		4341
0330			0.4	0.7	7.6	5.3	91.9	100	90.3	41.7	113...>		3874
0430				0.3	2.8	2.0	33.8	36.6	33.2	15.3	41.7		3702
0530					1.0	0.7	12.4	13.5	12.2	5.6	...>	2277	3639
0630						0.3	4.6	5.0	4.5	2.1	...>	838	1338
0730							1.7	1.8	1.7	0.8	...>	308	492
0830								0.7	0.6	0.3	...>	113	180
0930									0.2	0.1	...>	41.7	66.1
1030										0.0	...>	15.3	24.3
1130											...>	5.6	8.9
1230											...>	2.1	3.3
1330											...>	0.8	1.2
1430											...>	0.3	0.4
1530											...>	0.1	0.1
1630												0.0	0.0

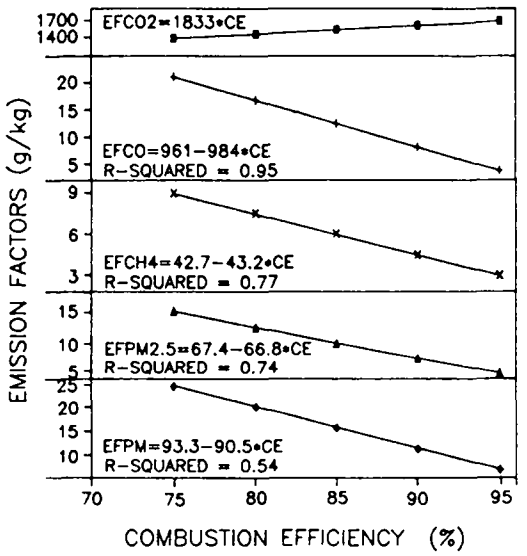

Fig. 10. Emission factors for PM, PM2.5, CH_4, CO, and CO_2 are functions of combustion efficiency. For the Sundance Fire, combustion efficiencies of 90 and 75% were used for the flaming and smoldering combustion phases, respectively.

bustion to about 92% for the flaming phase (Ward et al. 1989a). For airborne samples of smoke from wildfires in Oregon during 1987, Hegg et al. (1989) quantified emission factors for CO_2, CO, and hydrocarbons. Here, combustion efficiencies are calculated from their measurements for the smoke of the Silver Fire in southern Oregon of 89% and for the Myrtle Creek Fire, 88.7%.

Combustion efficiency values of 75% for the smoldering phase and 90% for the flaming phase are used for the Sundance Fire. From Fig. 10 and Equations 1 to 5, the corresponding emission factors for PM, PM2.5, CH_4, CO, and CO_2 for the flaming phase are 11.9, 7.3, 3.8, 75.0, and 1650 g/kg, respectively, and for the smoldering phase are 25.4, 17.3, 10.3, 222.6, and 1375 g/kg, respectively.

Hourly emissions of PM, PM2.5, CH_4, CO, and CO_2 are presented in Table 6. The rate of fuel consumption was cyclic with three major peaks occurring that are correlated with the rate of heat release

Table 5. Hourly fuel consumption for the main fire front, secondary flaming area, and smoldering combustion for the Sundance Fire.

Hour	Primary flame	Secondary flame	Total flaming	Smoldering	Cumulative
	(Gg/h)				(Tg)
1430	11.35	9.99	21.33	13:42	0.035
1530	21.84	8.95	30.78	24.30	0.090
1630	32.46	9.51	41.97	35.34	0.167
1730	15.71	8.44	24.14	28.19	0.219
1830	80.68	17.46	98.14	72.11	0.390
1930	16.03	9.12	25.15	42.35	0.457
2030	108.32	51.80	160.12	116.31	0.734
2130	39.50	24.33	63.83	82.94	0.880
2230	13.48	7.83	21.31	43.92	0.946
2330	1.75	1.87	3.62	18.43	0.968
2430	1.75	1.87	3.62	9.06	0.980
0130	1.75	1.87	3.62	5.61	0.990
0230	1.75	1.87	3.62	4.34	0.998
0330	1.75	1.87	3.62	3.87	1.005
0430	1.75	1.87	3.62	3.70	1.012
0530	1.75	1.87	3.62	3.64	1.020
0630				1.34	1.021
0730				0.49	1.021
0830				0.18	1.022
0930				0.07	1.022
1030				0.02	1.022
1130				0.01	1.022
1230				0.0	1.022
1330					1.022

for the fire (Fig. 9). Flaming and smoldering rates of CO production are illustrated in Fig. 11. Diedown for the smoldering combustion emissions follows the exponential diedown model (Equation 6).

The rate of release over time and total mass of trace gas species can be calculated using the EF_x/EF_{CO} ratios in Table 1. A listing of the rate of release for those gases is provided on an hourly basis in Table 7 and the total trace gas emissions illustrated in Fig. 12. The emissions are scaled to the rate of release of CO which is based on the overall combustion efficiency and mix of smoldering and flaming emissions. The correlation between emission rates for CO and that for CH_4 and nonmethane hydrocarbons is fairly good. For other nitrogen-based compounds, the emissions are scaled more closely to the nitrogen content of the fuel complex (Clements and McMahon 1980). The relation of NO_x to combustion efficiency has not been established.

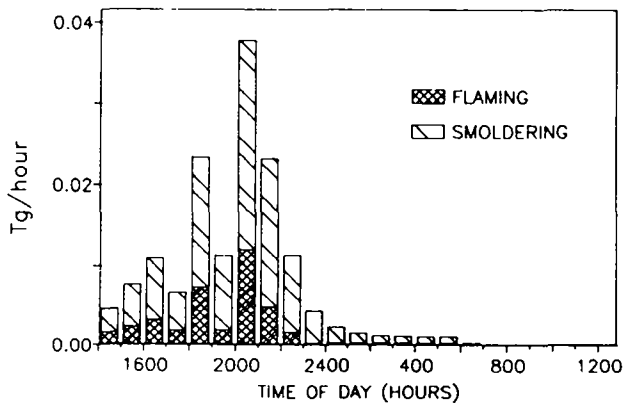

Fig. 11. Hourly rate of production of CO for the Sundance Fire by phase of combustion.

Total emissions

The Sundance Fire was a fast-moving, high-intensity fire that released a tremendous volume of smoke into the atmosphere over a short period. Rates of emission release are presented in Tables 6 and 7. These functions can be integrated to find the total emissions released during the course of the fire. Even though the fire exhibited high rates of heat release and fire growth, the overall magnitude of the emissions produced is less than 0.02% of the emissions

Table 6. Hourly emissions of PM, PM2.5, CH_4, CO, and CO_2 for the flaming phase and the total of flaming plus smoldering for the Sundance Fire, September 1-2, 1967.

Time	PM		PM2.5		CO		CO_2		NO_x	
	FLAM	TOT	FLAM	TOT	FLAM	TOTAL	FLAM	TOTAL	FLAM	TOTAL
1430	0.25	0.59	0.16	0.39	1.60	4.59	35.20	53.64	0.08	0.22
1530	0.37	0.98	0.22	0.65	2.31	7.72	50.78	84.20	0.12	0.37
1630	0.50	1.40	0.31	0.92	3.15	11.01	69.23	117.82	0.16	0.53
1730	0.29	1.00	0.18	0.66	1.81	8.09	39.83	78.58	0.09	0.38
1830	1.16	3.00	0.71	1.96	7.36	23.41	161.90	261.03	0.38	1.12
1930	0.30	1.38	0.18	0.92	1.89	11.31	41.48	99.70	0.10	0.53
2030	1.90	4.85	0.17	3.18	12.01	37.90	264.15	424.04	0.62	1.82
2130	0.76	2.87	0.46	1.90	4.79	23.25	105.30	219.32	0.24	1.10
2230	0.25	1.37	0.16	0.92	1.60	11.38	35.16	95.54	0.08	0.54
2330	0.04	0.51	0.03	0.35	0.27	4.38	5.97	31.32	0.01	0.20
2430	0.04	0.27	0.03	0.18	0.27	2.29	5.97	18.43	0.01	0.11
0130	0.04	0.19	0.03	0.12	0.27	1.52	5.97	13.68	0.01	0.07
0230	0.04	0.15	0.03	0.10	0.27	1.24	5.97	11.94	0.01	0.06
0330	0.04	0.14	0.03	0.09	0.27	1.13	5.97	11.30	0.01	0.05
0430	0.04	0.14	0.03	0.09	0.27	1.10	5.97	11.06	0.01	0.05
0530	0.04	0.14	0.03	0.09	0.27	1.08	5.97	10.98	0.01	0.05
0630	0.00	0.03	0.00	0.02	0.00	0.30	0.00	1.84	0.00	0.01
0730	0.00	0.01	0.00	0.01	0.00	0.11	0.00	0.68	0.00	0.05
0830	0.00	0.01	0.00	0.00	0.00	0.04	0.00	0.25	0.00	0.00
0930	0.00	0.00	0.00	0.00	0.00	0.02	0.00	0.09	0.00	0.00
1030	0.00	0.00	0.00	0.00	0.00	0.01	0.00	0.03	0.00	0.00
1130	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.00	0.00
1230	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
1330										
Totals (Tg)	0.006	0.019	0.004	0.012	0.038	0.151	0.844	1.545	0.002	0.007

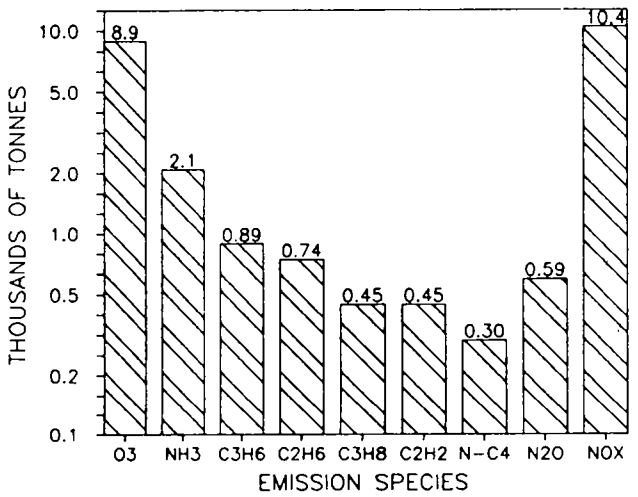

Fig. 12. Total trace gases released from the Sundance Fire.

released globally on an annual basis. Even in the United States, the relative emissions released by one large fire, as the Sundance Fire, are less than 1% of the total released from burning of wildland biomass fires annually.

The total emissions released from the Sundance Fire are thought to be a good model for conifer forest types where a significant ground fuel component exists. For the Sundance Fire, about 50% of the fuel consumption occurred through smoldering combustion. For savanna fires and possibly even tropical deforestation fires, the smoldering component is thought to be much lower. In these cases, higher levels of combustion efficiency would need to be used. This would tend to reduce the emissions of PM, PM2.5, and CO relative to the quantity of biomass consumed. Other sulfur and nitrogen compounds are not correlated through combustion efficiency mechanisms but are more closely coupled to the abundance of sulfur and nitrogen in the fuel complex.

APPLICATION OF EMISSIONS DATA

One application of the emission factors reviewed in this paper is for global estimates of emissions production. In this section, we compare the estimate of emissions for the Sundance Fire with those of

Table 7. Release of trace gases scaled to the release of CO using ratios of Hegg et al. (1989) presented in Table 1.

Time	O_3	NH_3	CH_4	C_3H_6	C_2H_6	C_3H_8	C_2H_2	N-C_4	N_2O	NO_x
	(Gg/h)									
1430	0.275	0.064	0.142	0.028	0.023	0.014	0.014	0.009	0.018	0.321
1530	0.463	0.108	0.239	0.046	0.039	0.023	0.023	0.015	0.031	0.540
1630	0.661	0.154	0.341	0.066	0.055	0.033	0.033	0.022	0.044	0.771
1730	0.485	0.113	0.251	0.049	0.040	0.024	0.024	0.016	0.032	0.566
1830	1.405	0.328	0.726	0.141	0.117	0.070	0.070	0.047	0.094	1.638
1930	0.679	0.158	0.351	0.068	0.057	0.034	0.034	0.023	0.045	0.792
2030	2.274	0.531	1.175	0.227	0.190	0.114	0.114	0.076	0.152	2.653
2130	1.395	0.326	0.721	0.140	0.116	0.070	0.070	0.047	0.093	1.628
2230	0.682	0.159	0.353	0.068	0.057	0.034	0.034	0.023	0.046	0.796
2330	0.262	0.061	0.136	0.026	0.022	0.013	0.013	0.009	0.018	0.306
2430	0.137	0.032	0.071	0.014	0.011	0.007	0.007	0.005	0.009	0.160
0130	0.091	0.021	0.047	0.009	0.008	0.005	0.005	0.003	0.006	0.106
0230	0.074	0.017	0.038	0.007	0.006	0.004	0.004	0.002	0.005	0.087
0330	0.068	0.016	0.035	0.007	0.006	0.003	0.003	0.002	0.004	0.080
0430	0.066	0.015	0.034	0.007	0.006	0.003	0.003	0.002	0.004	0.077
0530	0.065	0.015	0.034	0.007	0.005	0.003	0.003	0.002	0.003	0.076
0630	0.018	0.004	0.009	0.002	0.002	0.001	0.001	0.001	0.001	0.021
0730	0.007	0.002	0.003	0.001	0.001	0.000	0.000	0.000	0.000	0.008
0830	0.002	0.001	0.001	0.000	0.000	0.000	0.000	0.000	0.000	0.003
0930	0.001	0.000	0.001	0.000	0.000	0.000	0.000	0.000	0.000	0.001
1030	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
1130	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
1230	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
1330										
TOTALS	9.11	2.126	4.71	0.911	0.759	0.456	0.456	0.304	0.607	10.63

other historical wildfires in the United States. Major fire episodes have occurred in Siberia (Shostakovitch 1925), for example, that produced at least 10^3 times more smoke than the Sundance Fire. Brown and Davis (1973) provide the area burned, lives lost, mechanism of ignition, weather, and general fuels descriptions for several of the larger wildfire episodes in the United States over the past 200 y including the Great Idaho Fire, the Tillamook Fire, the Yacoult Fire, and the Air Force Bomb Range Fire in eastern North Carolina (Wade and Ward 1973). Most of these fires exhibited behavior associated with high intensity, "blowup" fires that probably exhibited major periods of heavy smoke production from smoldering combustion processes.

Assuming that the ratios of flaming to smoldering and the fuel consumption are similar to the Sundance Fire, other major fires are examined based on a comparison of the area burned (Table 8). The ratio of the historical fire area to the Sundance Fire is used as a multiplier for computing the total emissions from the fires listed in Table 8. The procedure used to compute the emissions from the Sundance Fire accounts for the combustion efficiency differences between flaming and smoldering combustion of the fuels.

The emission factors to be used for the analysis of these fires are developed based on a combustion efficiency relationship. In addition, results discussed for prescribed fires are considered in estimating emission factors for wildfires. The larger data base used for developing the combustion efficiency relation can be applied in developing particulate matter and CO emission factors for wildfires. As discussed, some of the emissions are directly correlated with emissions of CO, CH_4, or particulate matter. So, by estimating the source strength of one of the primary combustion products, other emissions source strengths can be estimated.

Now we discuss the application of the combustion efficiency relations presented in this paper as it can be applied to understanding the mixture of emissions released from fires on a global scale. Seiler and Crutzen (1980) estimated global biomass consumption from all major sources and the upper limit of their estimate of 3.3 Pg of biomass carbon consumption is used in Fig. 13 to base the release of other

Table 8. Listing of major fires over the past 200 y in the United States (Brown and Davis 1973) and the estimated emissions of PM, CO, and CH_4. Other emissions can be scaled from the emissions of CO according to the ratios provided by Hegg et al. (1989) listed in Table 2. The area of the Sundance Fire was 22 635 ha.

Fire	Location	Fire area/ Sundance (dimensionless)	Total emissions PM	CO	CH_4
			----(Tg)-----		
Miramichi and and Maine (1825)	New Brunswick and Maine	53.65	1.02	8.15	0.39
Peshtigo and Michigan (1871)	Wisconsin and Michigan	67.60	1.29	10.27	0.49
Far West, Yacoult (1902)	Washington and Oregon	17.88+	0.34	2.72	0.13
Adirondack (1903)	New York	11.39	0.22	1.73	0.08
Great Idaho (1910)	Idaho and Montana	53.65	1.02	8.15	0.39
Tillamook (1933)	Oregon	5.56	0.11	0.84	0.04
Alaska (1957)	Alaska	89.42	1.70	13.58	0.65
(1969)		75.12	1.43	11.41	0.54
Air Force Bomb Range (1971)	North Carolina	0.52-	0.01	0.08	0.01
United States (1988)	Total	89.42-	1.70	13.58	0.65

carbon containing combustion products. We have applied Equations 1 and 3 through 5 in distributing the carbon released among the major emissions released when the biomass fuels are burned in the open environment. If we assume a globally-weighted combustion efficiency of 90%, then 2.97, 0.213, 0.009, 0.023, and 0.008 Pg of carbon is calculated as being released in the form of CO_2, CO, CH_4, particulate matter, and nonmethane hydrocarbons (NMHC), respectively. From Fig. 13, changes in the average combustion efficiency affect the overall mixture of combustion products released to the atmosphere. For example, if the global combustion efficiency for burning of biomass is found to be 95%, then the carbon released as CO_2 would increase by 5%, and that released in the form of other products of incomplete combustion would be decreased by a corresponding amount.

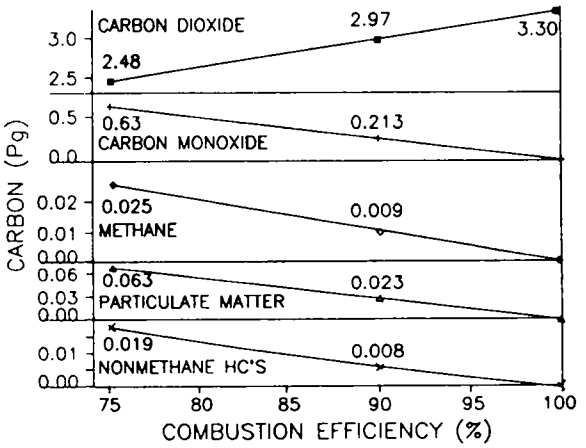

Fig. 13. Global emissions of carbon in the form of CO_2, CO, CH_4, particulate matter, and nonmethane hydrocarbons based on combustion efficiency and the upper limit estimate by Seiler and Crutzen (1980) of the release of 3.3 Pg/y of carbon from burning of biomass fuels.

Other products of combustion can be scaled to one of the primary products of combustion (Hegg et al. 1989, Table 1.). One additional example is provided in Fig. 13 where emission factors for NMHC (C_2-C_6 compounds) are well correlated with emission factors for CH_4 (EFNMHC = 0.760 + 0.616(EFCH$_4$), R^2 = 0.69).

We have demonstrated the application of algorithms for predicting the mix of combustion products from various combustion sources of different biomass fuels globally. The application of the algorithms for fuel types outside of the United States has not been extensively validated. The tests completed and literature values (Crutzen et al. 1985; Andreae et al. 1988) suggest that the mix of carbon-containing emissions released from different biomass fuel types in other regions (for example, Brazil) fits the algorithms developed from extensive field tests of emissions produced from prescribed fires in the Western United States (Ward et al. 1989). It is expected that weighted combustion efficiencies for broad classifications of biomass and fire types can be used with the algorithms presented here to improve the overall estimates of the release of emissions from biomass fires globally.

SUMMARY

Flaming and smoldering combustion processes affect the production of emissions. CO and CO_2 combined account for 90-95% of the carbon released during biomass burning. Combustion efficiency ranges from 50-80% for smoldering combustion and from 80-95% for flaming combustion. Many of the compounds released during biomass burning are correlated with combustion efficiency and can be scaled to the release of CO.

The size distribution for particles produced from biomass burning is bimodal, with particle-mass peaks occurring near 0.5 μm and greater than 43 μm. The abundance of the larger sized particles close to the source are released in relation to the intensity of the fire (rate of heat release per unit area). The mass of particulate matter between 1μm and 10 μm makes up less than 10% of the total mass.

A major wildfire in North America, the Sundance Fire, is used as a model to scale the emissions from several historical fires in North America. The Sundance Fire consumed approximately 1.02 Tg of fuel divided almost equally between flaming and smoldering. Approximately 0.0189 Tg of PM, 0.151 Tg of CO, and 1.54 Tg of CO_2 were released from the 22 635-ha wildfire. Ratios of CO to other trace gases were used to estimate the release of NO_x, NH_3, and N_2O, of 0.0106, 0.0021, and 0.0006 Tg, respectively. It is recognized, however, that the release of nitrogen and sulfur compounds may be more closely coupled to the nitrogen and sulfur content of the biomass.

Global-scale emissions released from the combustion of biomass fuels are difficult to estimate based on a few measurements in the United States. The representativeness of fires in the United States and areas of savanna and tropical deforestation are questioned because of potential differences in fuel chemistry and combustion efficiency. In addition, the combustion efficiency may be much higher for fires of the tropical areas than previously reported. Global biomass consumption inventories should include data on the characteristics of the biomass fuel consumed by the fires for different biomes, the ratio of fuel consumption by flaming and smoldering combustion processes, and the general chemistry of the strata of fuel consumed. These data will facilitate improved estimates of the release of emissions into the atmosphere from both wildland fires and other fires for agricultural purposes using models similar to those presented in this paper and that are presently being developed.

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California Wildfires of 2008: Coarse and Fine Particulate Matter Toxicity

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Background: During the last week of June 2008, central and northern California experienced thousands of forest and brush fires, giving rise to a week of severe fire-related particulate air pollution throughout the region. California experienced $PM_{10–2.5}$ (particulate matter with mass median aerodynamic diameter > 2.5 μm to < 10 μm; coarse) and $PM_{2.5}$ (particulate matter with mass median aerodynamic diameter < 2.5 μm; fine) concentrations greatly in excess of the air quality standards and among the highest values reported at these stations since data have been collected.

Objectives: These observations prompt a number of questions about the health impact of exposure to elevated levels of $PM_{10–2.5}$ and $PM_{2.5}$ and about the specific toxicity of PM arising from wildfires in this region.

Methods: Toxicity of $PM_{10–2.5}$ and $PM_{2.5}$ obtained during the time of peak concentrations of smoke in the air was determined with a mouse bioassay and compared with PM samples collected under normal conditions from the region during the month of June 2007.

Results: Concentrations of PM were not only higher during the wildfire episodes, but the PM was much more toxic to the lung on an equal weight basis than was PM collected from normal ambient air in the region. Toxicity was manifested as increased neutrophils and protein in lung lavage and by histologic indicators of increased cell influx and edema in the lung.

Conclusions: We conclude that the wildfire PM contains chemical components toxic to the lung, especially to alveolar macrophages, and they are more toxic to the lung than equal doses of PM collected from ambient air from the same region during a comparable season.

Key words: air pollution, alveolar macrophage, lung inflammation, mouse, $PM_{2.5}$, PM_{10}, source-specific particulate matter. *Environ Health Perspect* 117:893–897 (2009). doi:10.1289/ehp.0800166 available via *http://dx.doi.org/* [Online 2 February 2009]

During the last week of June 2008, central and northern California experienced a major outbreak of wildfires caused by a series of lightning strikes that was unprecedented in the past century in its extent and severity, with transport of smoke over large distances from the fires, especially in the Central Valley. A regional map with the location of the largest of these fires illustrated is available from the California Department of Forestry and Fire Protection (2008). Air quality in the region was severely affected by the smoke from these fires, and millions of people were exposed to quantities of wildfire-generated particulate matter (PM) greatly in excess of the current PM standards. Hourly levels of PM with mass median aerodynamic diameter < 2.5 μm ($PM_{2.5}$) at Tracy (near our sampling site) peaked at 160 μg/m^3 (San Joaquin Valley Air Pollution Control District 2008), whereas hourly concentrations of PM with mass median aerodynamic diameter < 10 μm (PM_{10}) peaked at 200 μg/m^3. Further to the north in the Sacramento River Valley, closer to the major fires, $PM_{2.5}$ values of 262 μg/m^3 were reported on the same days. Thus, PM with mass median aerodynamic diameter > 2.5 μm to < 10 μm ($PM_{10–2.5}$) and $PM_{2.5}$ concentrations were greatly in excess of the California 24-hr average ambient air quality standards ($PM_{10–2.5}$, 50 μg/m^3; $PM_{2.5}$, 35 μg/m^3) and among the highest values reported at these stations since data have been collected for PM pollution in these size classifications. These observations raise concerns about the potential health impact of exposure to high levels of wildfire PM, as the possible health effects associated with these acute exposures to PM from wildfires at these very high levels are not understood.

$PM_{10–2.5}$ and $PM_{2.5}$ samples were obtained during the last week of June 2008, when the fires were at their worst, from a U.S. Environmental Protection Agency (EPA) designated National Air Emissions Monitoring Study site that was heavily impacted. The monitoring included data on $PM_{10–2.5}$ concentrations logged every 2 min (Series FH 62C14 Beta Sampler; Thermo Electron Corp., Franklin, MA). Peak value observed during the 2 days studied was 381 μg/m^3, with values between 200 and 380 μg/m^3 logged routinely over a period of several hours in the late afternoon and early evening of 26 June. Thus, the values reported at Tracy, the nearest San Joaquin Valley Air Pollution Control District monitoring site, probably underestimate the actual concentrations at our sampling site. This manuscript describes a toxicologic analysis of both the coarse and fine particles ($PM_{10–2.5}$ and $PM_{2.5}$) collected during the 2-day period of peak air pollution during June 2008, and compares the toxicity of wildfire PM with PM collected from nearby ambient air under normal conditions during June 2007. This manuscript will demonstrate that the inherent toxicity on an equal-dose basis is greater for the wildfire PM than that of PM from normal ambient air in this region. This is a novel and unexpected observation.

Materials and Methods

Particulate matter used in this study was collected with a high-volume air sampler (model GS2310; Andersen Instruments Inc., Smyrna, GA) equipped with a four-stage cascade impactor (series 230, Andersen Instruments Inc.) in the summer months from a location in the northeast of the San Joaquin Valley in California. Slotted aluminum substrates (Tisch Environmental, Cleves, OH) were used for PM collection. The nominal flow rate used for collection was 20 ft^3/min, with particle size cutoffs of 10.2, 4.2, 2.1, and 1.3 μm. For the purposes of this manuscript, we will refer to coarse PM as particles with a mass median aerodynamic diameter range of 10.2–2.1 μm and fine PM as particles within 2.1–1.3 μm. After collection, substrates from each stage were weighed; particles were removed by scraping with a spatula and stored at –80°C in vials. Thirty minutes before use, particles were suspended in phosphate-buffered saline (PBS), pH 7.6 (Mediatech, Inc., Herndon, VA) at the desired concentration. The final pH of the resultant suspension was pH > 7.

Bioassay techniques in the mouse have been validated and optimized as described previously (Wegesser and Last 2008). Briefly, male BALB/c mice 8–10 weeks of age (25–30 g) were purchased from Charles River Breeding Laboratories (Wilmington, MA). Mice were housed, four animals per cage, in filtered Bio-Clean facilities in the Animal Resources Center (University of California Davis, CA). Animals received water and standard feed (Purina Rat Chow) *ad libitum* and were allowed to acclimate for 1 week before any experimental procedures. The animals were kept on a 12-hr light/dark cycle at room temperature (68–70°F)

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We thank F. Mitloehner, S. Cliff, N. Kado, and D. Bennett for their assistance with access to the sampling site and PM sampling.

This study was funded in part by ES-07059 from the National Institute of Environmental Health Sciences, U07/CCU906162 from the National Institute for Occupational Safety and Health, and RD-83241401 from the U.S. Environmental Protection Agency.

The authors declare they have no competing financial interests.

Received 8 September 2008; accepted 2 February 2009.

and 30–70% relative humidity. All procedures were performed under an Institutional Animal Care and Use Committee approved protocol. All animals used in this study were treated humanely and with regard for alleviation of suffering.

Methods for intratracheal instillation of 50-µL suspensions of known amounts of PM into mice and evaluation of lung inflammation are described in detail elsewhere (Wegesser and Last 2008). Bronchoalveolar lavage (BAL) and tissue were collected 24 hr after PM instillation. Whole cell counts were performed with whole lavage fluid and a hemocytometer. Cells were separated from supernatant by centrifugation at 2,000 rpm in a benchtop centrifuge and stained with Diff-Quick (Fisher Scientific; Kalamazoo, MI) for differential cell counts. Protein content of lavage fluid supernatant was determined by a colorimetric reaction with the Micro BCA Protein Assay Reagent Kit (Pierce Biotechnology, Rockford, IL). Lavaged lungs were fixed at 30 cm pressure with 1% paraformaldehyde in PBS for 1 hr for histopathologic assessment, after staining with Harris' hematoxylin and eosin, with an Olympus BH2 microscope connected to an OLY-750 Color Camera (Olympus; Center Valley, PA). Endotoxin in PM preparations was assayed by the Limulus amoebocyte lysate (LAL) assay (Wegesser and Last 2008).

Statistical analysis of data was performed with Prism 4.0 and 5.0 (GraphPad Software, San Diego, CA). All values are expressed as mean ± SE. Parametric analysis of data was conducted using analysis of variance with Tukey's post-test for multiple comparisons. Differences were considered significant if the *p*-value (two-tailed) was < 0.05. Welch's correction was applied if variances were found to be unequal.

Results

We found no significant differences in total cells recovered by lung lavage between either untreated (data not shown) or saline-instilled controls and mice instilled with 10, 25, or 50 µg wildfire $PM_{10–2.5}$ (Figure 1A). There was a significant increase in total lavageable cells with instillation of 100 µg $PM_{10–2.5}$ from the wildfire sample. In prior studies we have seen significant dose-related increases (more than twice as many cells) in total lavageable cells from mice instilled with 25 or 50 µg $PM_{10–2.5}$ from ambient air samples collected from this geographic area (Wegesser and Last 2008), so the lack of increase in total lavageable cells seen between 10 and 50 µg $PM_{10–2.5}$ from the wildfire samples is unusual.

The cells lavaged from lungs of control mice were 95–100% macrophages, whereas lavage fluid from mice instilled 24 hr earlier with 50 µg $PM_{10–2.5}$ from ambient air contained about 30% macrophages and 70% neutrophils (Wegesser and Last 2008). We found 49 ± 15, 47 ± 18, and 57 ± 23% neutrophils for mice instilled with 10, 25, or 100 µg wildfire $PM_{10–2.5}$, respectively (Figure 2A). Thus, despite the lack of apparent increase in total cell numbers in the lung lavage from the mice exposed to 10 or 25 µg $PM_{10–2.5}$ in Figure 1A, the mice responded to the wildfire PM at the lowest and the highest doses tested. The cell populations had shifted to about half neutrophils, which is not normal, despite total cell numbers remaining more or less constant. On an equal-dose basis, the wildfire lavage samples contained significantly lower numbers of macrophages than did lavage fluid from mice instilled with $PM_{10–2.5}$ collected from normal ambient air (AA) during the same period 1 year earlier (Figure 2B; compare the responses to 25 and 50 µg wildfire $PM_{10–2.5}$ with 25 AA and 50 AA, where 25 AA and 50 AA signify the samples of 25 and 50 µg $PM_{10–2.5}$ from normal ambient air). Direct LAL assay shows < 1 endotoxin unit (EU) of endotoxin/50 µg $PM_{10–2.5}$ preparation, ruling out a significant role for lipopolysaccharide (LPS) in the generation of the observed neutrophilic inflammation, as Balb/C mice respond normally to endotoxin (Silvia and Urosevic 1999).

The lung inflammatory response to $PM_{10–2.5}$ from the wildfire differs from the response to $PM_{10–2.5}$ from ambient air. Because the total number of lavageable cells did not increase in the mice exposed to 10–50 µg $PM_{10–2.5}$ (Figure 1A), and half of the total cells were neutrophils, the wildfire $PM_{10–2.5}$ must have caused a decrease in the numbers of macrophages in the lungs (Figure 1B). Note also that in the 100 µg wildfire $PM_{10–2.5}$ sample, the total cells in the lavage were significantly increased and this increase was made up primarily of neutrophils (57% of the total cells). When compared with the 25 µg and 50 µg samples from normal AA, all animals dosed with $PM_{10–2.5}$ from the wildfire (10, 25, 50, and 100 µg groups) had significantly fewer macrophages in their lung lavage fluid (Figure 2B). Thus, the most striking aspect of the cell differential counts in the mice exposed to the wildfire-derived $PM_{10–2.5}$ is the relative absence of alveolar macrophages in their lungs, compared with PBS-instilled controls (Figure 2B). This accounts for the lower total cell count in these wildfire PM-exposed lungs, suggesting that either the

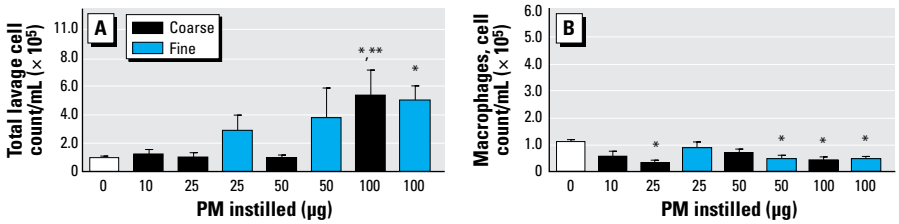

Figure 1. Number (mean ± SE) of total cells (*A*) and macrophages (*B*) recovered in lung lavage fluid from mice intratracheally instilled with different doses of $PM_{10–2.5}$ (coarse) or $PM_{2.5}$ (fine) from the wildfire samples (PBS-instilled controls, 0 µg). Note difference in *y*-axis scale in *A* and *B*.
*$p < 0.001$ compared with control. **$p < 0.01$ compared with 25 or 50 µg.

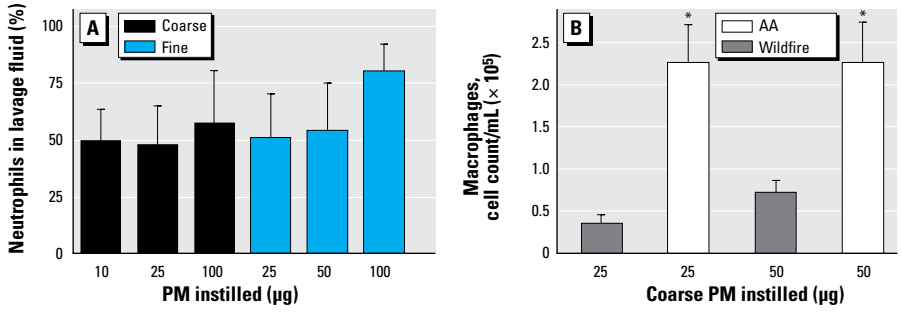

Figure 2. (*A*) Percentage of neutrophils in the lung lavage fluid from mice instilled with the indicated amounts of $PM_{10–2.5}$ or $PM_{2.5}$ wildfire PM. All of the indicated values are significantly greater than PBS-instilled controls, which contained 0% polymorphonuclear leukocytes. (*B*) Number of macrophages in the lung lavage fluid of mice instilled with either 25 or 50 µg PM: comparison of wildfire PM and normal AA PM collected 1 year earlier from the same area. Values shown are mean ± SE.
*$p < 0.05$ compared with either 25 or 50 µg wildfire PM samples.

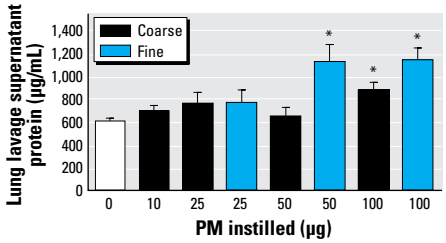

Figure 3. Protein content of lung lavage fluid supernatant of mice instilled with the indicated amounts of wildfire $PM_{10–2.5}$ or $PM_{2.5}$.
*$p < 0.05$ compared with control.

wildfire $PM_{10-2.5}$ may be especially toxic to pulmonary alveolar macrophages or that these wildfire $PM_{10-2.5}$ create a condition in which macrophages are difficult to extract from the lungs by lavage, perhaps because of enhanced adherence to alveolar surfaces.

As shown in Figure 3, there was significantly more protein in the lavage fluid supernatant from mice instilled with 100 µg of the $PM_{10-2.5}$ fraction, and a trend toward higher amounts of protein in all of the groups examined compared with controls. The lack of a clear dose–response relationship in the data suggests that there is either a threshold in the observed response or that the lavage supernatant protein content is measuring a phenomenon more complex than simple fluid transudation across the airway epithelial barrier in a damaged lung (Witschi and Last 2001). In contrast, we have not observed any significant increase in lung lavage supernatant content of protein in mice exposed to $PM_{10-2.5}$ preparations from normal AA collected from the San Joaquin Valley.

We performed similar experiments with $PM_{2.5}$ preparations from the wildfire samples (Figures 1 and 3) collected simultaneously to facilitate direct comparisons of the two size fractions. We found significantly more total cells in the mice instilled with 100 µg wildfire $PM_{2.5}$, with an apparent trend for dose response between 25 and 100 µg $PM_{2.5}$ (Figure 1A). We observed significantly fewer macrophages in the lung lavage fluid from mice instilled with either 50 or 100 µg wildfire $PM_{2.5}$ and comparable decreases of macrophages in mice instilled with 100 µg wildfire $PM_{10-2.5}$ or $PM_{2.5}$ (Figure 1B). Both the 50 µg and 100 µg samples caused significant increases in the concentration of lung lavage supernatant protein in mice exposed to wildfire $PM_{2.5}$ preparations (Figure 3), with an apparent dose-related difference in response to the 25 µg dose versus the 50- and 100-µg doses of $PM_{2.5}$ tested. The increase in amount of protein in the lung lavage supernatant was not significantly different between the mice instilled with 100 µg $PM_{10-2.5}$ or $PM_{2.5}$.

As shown in Figure 4, a marked influx of cells composed of monocytes and neutrophils was observed in mice instilled with 100 µg wildfire $PM_{10-2.5}$ within the peribronchial tissues of the airways, along with an increased cellularity of septal tissues in the lung parenchyma with notable accumulation of inflammatory cells in the centriacinar airspaces of the lungs. Occasional extravasation of red blood cells, along with patchy edema fluid, was also noted in the alveolar airspaces. Increased lung tissue damage was noted with increasing doses of instilled particles for both $PM_{2.5}$ and $PM_{10-2.5}$ (Figures 4–7). In addition, wildfire $PM_{10-2.5}$ particles induced greater histologic changes to the lungs for both the airways and the alveoli when compared with $PM_{10-2.5}$ particles collected under normal ambient conditions.

Discussion

There exists extensive literature on epidemiologic studies and a much smaller literature on whole-animal studies of the heath effects of exposure to woodsmoke from stoves, agricultural burning, wildfires, and other sources (Naeher et al. 2007; Zelikoff et al. 2002). Dubick et al. (2002) presented evidence of oxidative stress (lipid peroxidation) in lungs of rats acutely exposed (16 min) to whole woodsmoke by inhalation. Li et al. (1997) intratracheally instilled $PM_{10-2.5}$ collected from AA in Scotland into rats and observed neutrophilic inflammation, increase in protein content, and oxidant stress (less glutathione) in lung lavage fluid from these animals, similar to our findings in this study. Many authors have examined the toxicity and proinflammatory activity of $PM_{10-2.5}$ and/or $PM_{2.5}$ by examination of their effects on cultured cells *in vitro* (e.g., Jiménez et al. 2002; Monn and Becker 1999; Veranth et al. 2004). Others have examined the toxicity of fractionated PM components to cultured cells (e.g., Adamson et al. 1999; Carter et al. 1997; Imrich et al. 2000). Specific toxicologic studies with PM isolated from wildfire smoke (e.g., Leonard et al. 2000; Jalava et al. 2006) are rare in the literature, presumably because of difficulty in collecting such PM fractions.

The lungs of mice exposed to wildfire $PM_{10-2.5}$ or $PM_{2.5}$ in the present study showed significant damage, as measured by histologic evaluation of inflammatory cell influx or by relative neutrophil content or total protein content of lung lavage fluid, compared with mice exposed to 10-fold higher doses of normal AA PM from the same area. The relative toxicity of $PM_{10-2.5}$ and $PM_{2.5}$ seemed similar in these experiments, but we should note that use of the intratracheal instillation route would mask differences in actual PM dosage to the lung of these different size fractions when they were inhaled. Based on the responses of mice to the 10 µg dose of wildfire $PM_{10-2.5}$ or $PM_{2.5}$ compared with the response to

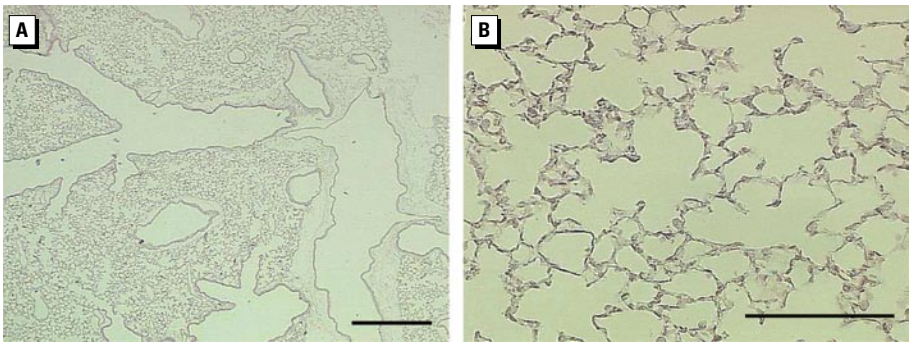

Figure 5. Representative lung sections from control mice instilled 24 hr with 50 µL PBS solution. (*A*) Whole lung (low magnification) showing airways, blood vessels, and parenchyma; bar = 500 µm. (*B*) Lung parenchyma (high magnification) showing thin delicate alveolar septal tissues; bar = 100 µm.

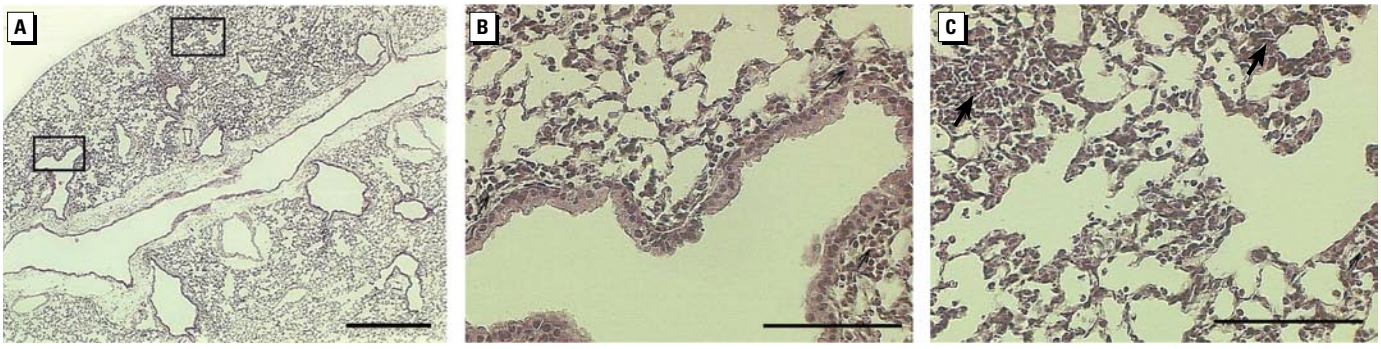

Figure 4. Representative lung sections from mice instilled 24 hr with 100 µg wildfire $PM_{10-2.5}$. (*A*) Whole lung; bar = 500 µm. Boxes indicate areas shown in higher magnification in (*B*) and (*C*). (*B*) Proximal lung with conducting airways; bar = 100 µm. (*C*) Distal lung with centriacinar region; bar = 100 µm. Arrows indicate typical areas with inflammatory cell infiltrates. Sections from control animals are shown in Figure 5.

50–100 μg PM from normal AA, we can estimate the relative toxicity of the wildfire PM on an equal-dose basis as about 10-fold more damaging than normal PM. Based on daily average PM mass collected with our high-volume sampler, there was about 2.2 times more $PM_{10–2.5}$ and about 3.4 times more $PM_{2.5}$ concentration in the air than on normal days in the region. Thus, a mouse exposed to the smoke-laden air from the wildfires would have been exposed to a relative risk of lung inflammation on the order of > 30 times the risk of breathing ordinary air in this region, which has some of the highest reported concentrations of $PM_{2.5}$ in ambient air in the United States. In addition, the underlying mechanisms of toxicity may differ for the wildfire and for normal PM. The severity of the actual damage is masked during routine analysis of lung lavage parameters (cellularity or protein content) by the concomitant killing of pulmonary alveolar macrophages by the wildfire particles. The extent of damage to the lungs cannot be appreciated by *in vitro* analyses of $PM_{10–2.5}$ or $PM_{2.5}$ in cultured cells because of participation of extravasated blood and influx of inflammatory cells and edema fluid into the lung during pathologic changes. These observations highlight the critical importance of bioassays of toxicity of inhaled pollutants in whole animals as a component of a balanced scientific approach to estimating their toxicity.

Preliminary experiments suggest that active pro-inflammatory agent(s) in the wildfire $PM_{10–2.5}$ fraction is heat labile and extractable into an organic solvent, suggesting its organic nature. This is a reasonable hypothesis, given that the genesis of wildfire PM is from the incomplete combustion of biomass at relatively low temperatures. Others have suggested that aromatic chemical compounds, which can redox cycle, in PM derived from diesel exhaust or AA are able to damage lung cells and organelles by oxidative stress and are responsible for PM toxicity (Goldsmith et al. 1997; Laks et al. 2008; Xia et al. 2004). Consistent with this suggestion, we found dose-related increased staining for nitrotyrosine in the lungs of mice instilled with the wildfire $PM_{10–2.5}$ but not the wildfire $PM_{2.5}$. The active pro-inflammatory agent(s) in the wildfire $PM_{2.5}$ need not be the same agent(s) responsible for the activity of the $PM_{10–2.5}$. Studies with $PM_{10–2.5}$ and $PM_{2.5}$ collected from Alaska wildfire sites also implicate oxidative stress, in this case derived from free radicals arising (at least in part) from reactive metals in particles ($PM_{10–2.5}$ and $PM_{2.5}$), as a major source of carbon-centered free radicals responsible for their toxicity (Leonard et al. 2007).

Pulmonary alveolar macrophages may be a preferred target for PM toxicity, which affects macrophage function and specifically suppresses nitric oxide production by the macrophages (Antonini et al. 2002). Based on our results, there was no striking difference in the toxicity of the $PM_{2.5}$ and $PM_{10–2.5}$ fractions from the wildfire. $PM_{10–2.5}$ was reported to have greater toxicity than $PM_{2.5}$ from other sources (Kleinman et al. 2003), but our results suggest that relative toxicity of the two PM sizes may be assay dependent.

The use of intratracheal instillation as an exposure route may be criticized as unphysiologic because of the delivery of a bolus dose rather than a more gradual dose by inhalation exposure (Witschi and Last 2001). However, recent studies (Costa et al. 2006) suggest that if the total dose of PM instilled intratracheally remains in the physiologic range (i.e., equivalent to total dose achieved by acute inhalation exposure), then responses of laboratory animals to intratracheal administration are comparable with results found after inhalation exposure. Thus, intratracheal injection is an acceptable experimental approach to studying PM toxicity in whole animals.

Due to the sporadic and unpredictable nature of wildfires and the tendency for air pollution monitors to be situated in predominantly

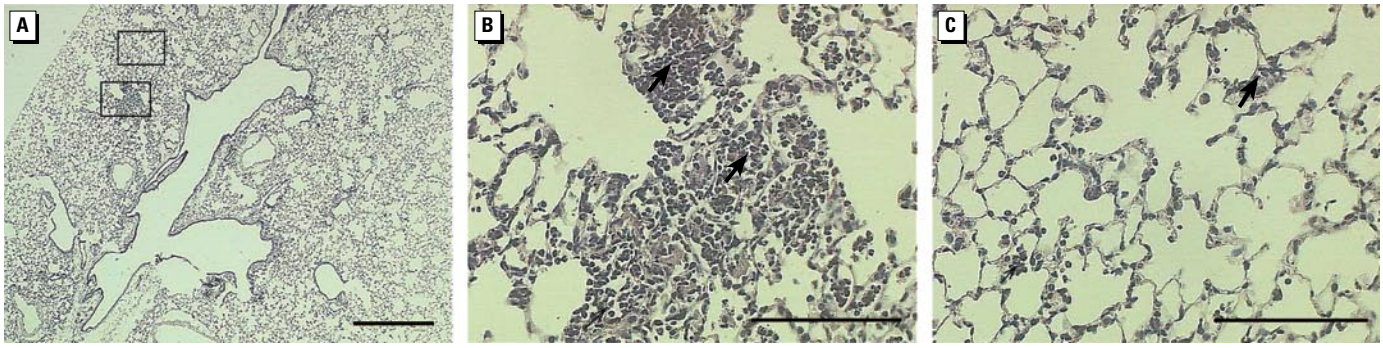

Figure 6. Representative lung sections from mice instilled 24 hr with 100 μg wildfire $PM_{2.5}$. (*A*) Whole lung (low-magnification; bar = 500 μm); boxes indicate areas shown in higher magnification in (*B*) and (*C*). (*B*) Centriacinar lung region showing the prominent accumulation of numerous inflammatory cells within alveolar airspaces. (*C*) Distal alveolar region with a diffuse increase in septal cellularity and occasional inflammatory cells within the alveolar airspaces. Arrows indicate areas of inflammatory cell influx. Bar = 100 μm in (*B*) and (*C*).

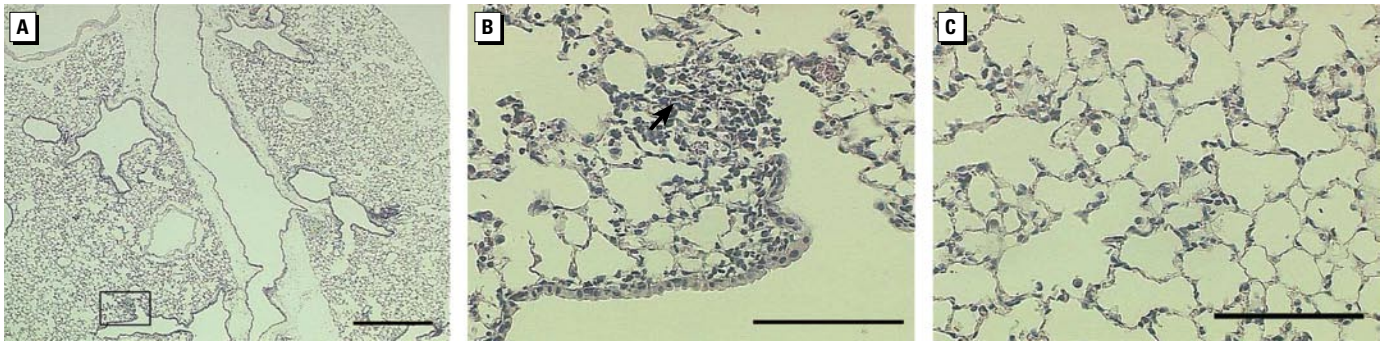

Figure 7. Representative lung sections from mice instilled 24 hr with 10 μg wildfire $PM_{10–2.5}$. (*A*) Whole lung (low-magnification; bar = 500 μm); box indicates area shown in higher magnification in (*B*) and (*C*). (*B*) Centriacinar region with accumulations of inflammatory cells in the alveolar airspaces; arrow indicates area of cellular influx. (*C*) Distal alveolar region with subtle markings of pulmonary edema and increased abundance of alveolar macrophages. Bar = 100 μm in (*B*) and(*C*).

urban areas where population is concentrated, there has been relatively little systematic study of the toxicity of PM from wildfires in the literature. Hänninen et al. (2008) estimated population exposures in southern Finland to wildfire PM from a series of fires in Russia and the former Soviet Union in 2002. Their article reviews the existing epidemiologic data on exposures to PM from woodsmoke combustion, including wildfires, and concludes that within a large range of uncertainty, the effect of wildfire PM seems to be consistent with the effects of similarly sized PM from other sources of urban PM on an equal-exposure basis. However, total PM mass is higher during the wildfire episodes, so total toxicity would be greater. This conclusion is consistent with regulatory guidance from the World Health Organization and the U.S. EPA, where all PM of a given size class are assumed to be equally toxic regardless of source or chemical composition. Cell culture assays of wildfire-derived PM in mouse macrophages (Jalava et al. 2006) suggest that these size-fractionated PM preparations elicit similar or lesser toxicity on an equal-mass basis than normal ambient PM from the same sources. However, our comparative results testing PM in mice from normal AA and from AA during the wildfire suggest that the assumption that all particles of a given size class in the AA have the same toxicity (which is the basis for regulation of PM in the atmosphere) is an oversimplification.

We can conclude from these studies that the lungs of mice exposed to wildfire $PM_{10–2.5}$ or $PM_{2.5}$ show significant damage, as measured by histologic evaluation of inflammatory cell influx or by relative neutrophil or total protein content of lung lavage fluid, compared with mice exposed to 10-fold higher doses of normal AA PM from the same area. Thus, the inherent toxicity on an equal-dose basis is greater for the wildfire PM than that of PM from normal AA in this region. This is a novel and unexpected observation. Thus, a mouse exposed to the smoke-laden air from the wildfires with peak hourly $PM_{10–2.5}$ and $PM_{2.5}$ concentrations about three times higher than normal peak PM concentrations in AA in this region, would be exposed to a relative risk on the order of > 30 times the risk of breathing ordinary air in the region. The relative toxicity of the $PM_{10–2.5}$ and $PM_{2.5}$ seemed to be similar in these experiments, but we should note that use of intratracheal instillation route would mask differences in actual PM dosage to the lung of these different size fractions on inhalation. Our observations in mice suggest that further research is required to test the assumption that all particles of a given size class in the ambient air have the same toxicity, the current regulatory approach paradigm.

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CORONAVIRUS

Air pollution and COVID-19 mortality in the United States: Strengths and limitations of an ecological regression analysis

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Assessing whether long-term exposure to air pollution increases the severity of COVID-19 health outcomes, including death, is an important public health objective. Limitations in COVID-19 data availability and quality remain obstacles to conducting conclusive studies on this topic. At present, publicly available COVID-19 outcome data for representative populations are available only as area-level counts. Therefore, studies of long-term exposure to air pollution and COVID-19 outcomes using these data must use an ecological regression analysis, which precludes controlling for individual-level COVID-19 risk factors. We describe these challenges in the context of one of the first preliminary investigations of this question in the United States, where we found that higher historical $PM_{2.5}$ exposures are positively associated with higher county-level COVID-19 mortality rates after accounting for many area-level confounders. Motivated by this study, we lay the groundwork for future research on this important topic, describe the challenges, and outline promising directions and opportunities.

INTRODUCTION

The suddenness and global scope of the coronavirus disease 2019 (COVID-19) pandemic have raised urgent questions that require coordinated investigation to slow the disease's devastation. A critically important public health objective is to identify key modifiable environmental factors that may contribute to the severity of health outcomes [e.g., intensive care unit (ICU) hospitalization and death] among individuals with COVID-19. Numerous scientific studies reviewed by the U.S. Environmental Protection Agency (EPA) have linked fine particles ($PM_{2.5}$; particles with diameter, ≤ 2.5 μm) to a variety of adverse health events (*1*) including death (*2*). It has been hypothesized that because long-term exposure to $PM_{2.5}$ adversely affects the respiratory and cardiovascular systems and increases mortality risk (*3*–*5*), it may also exacerbate the severity of COVID-19 symptoms and worsen the prognosis of this disease (*6*).

Epidemiological studies to estimate the association between long-term exposure to air pollution and COVID-19 hospitalization and death is a rapidly expanding area of research that is attracting attention around the world. Two studies have been published using data from European countries (*7*, *8*), and many more are available as preprints. However, because of the unprecedented nature of the pandemic, researchers face serious challenges when conducting these studies. One key challenge is that, to our knowledge, individual-level data on COVID-19 health outcomes for large, representative populations are not publicly available or accessible to the scientific community. Therefore, the only way to generate preliminary evidence on the link between $PM_{2.5}$ and COVID-19 severity and outcomes using these aggregate data is to use an ecological regression analysis. With this study design, publicly available area-level COVID-19 mortality rates are regressed against area-level air pollution concentrations while accounting for area-level potential confounding factors. Here, we discuss the strengths and limitations of conducting ecological regression analyses of air pollution and COVID-19 health outcomes and describe additional challenges related to evolving data quality, statistical modeling, and control of measured and unmeasured confounding, paving the way for future research on this topic. We discuss these challenges and illustrate them in the context of a specific study, in which we investigated the impact of long-term $PM_{2.5}$ exposure on COVID-19 mortality rates in 3089 counties in the United States, covering 98% of the population.

Illustration of an ecological regression analysis of historical exposure to $PM_{2.5}$ and COVID-19 mortality rate

We begin by describing how to conduct an ecological regression analysis in this setting. COVID-19 death counts (a total of 116,747 deaths) were obtained from the Johns Hopkins University Coronavirus Resource Center and were cumulative up to 18 June 2020. We used data from 3089 counties, of which 1244 (40.3%) had reported zero COVID-19 deaths at the time of our analysis. Daily $PM_{2.5}$ concentrations were estimated across the United States on a $0.01° \times 0.01°$ grid for the period 2000–2016 using well-validated atmospheric chemistry and machine learning models (*9*). We used zonal statistics to aggregate $PM_{2.5}$ concentration estimates to the county level and then averaged across the period 2000–2016 to perform health outcome analyses. Figure 1 illustrates the spatial variation in 2000–2016 average (hereafter referred to as "long-term average") $PM_{2.5}$ concentrations and COVID-19 mortality rates (per 1 million population) by county.

We fit a negative binomial mixed model using COVID-19 mortality rates as the outcome and long-term average $PM_{2.5}$ as the exposure of interest, adjusting for 20 county-level covariates. We conducted more than 80 sensitivity analyses to assess the robustness of the findings to various modeling assumptions. We found that an increase of 1 $\mu g/m^3$ in the long-term average $PM_{2.5}$ is associated with a statistically significant 11% (95% CI, 6 to 17%) increase in the county's COVID-19 mortality rate (see Table 1); this association continues to be stable as more data accumulate (fig. S3). We also found that population density, days since the first COVID-19 case was reported, median household income, percent of owner-occupied housing, percent of the adult population with less than high school

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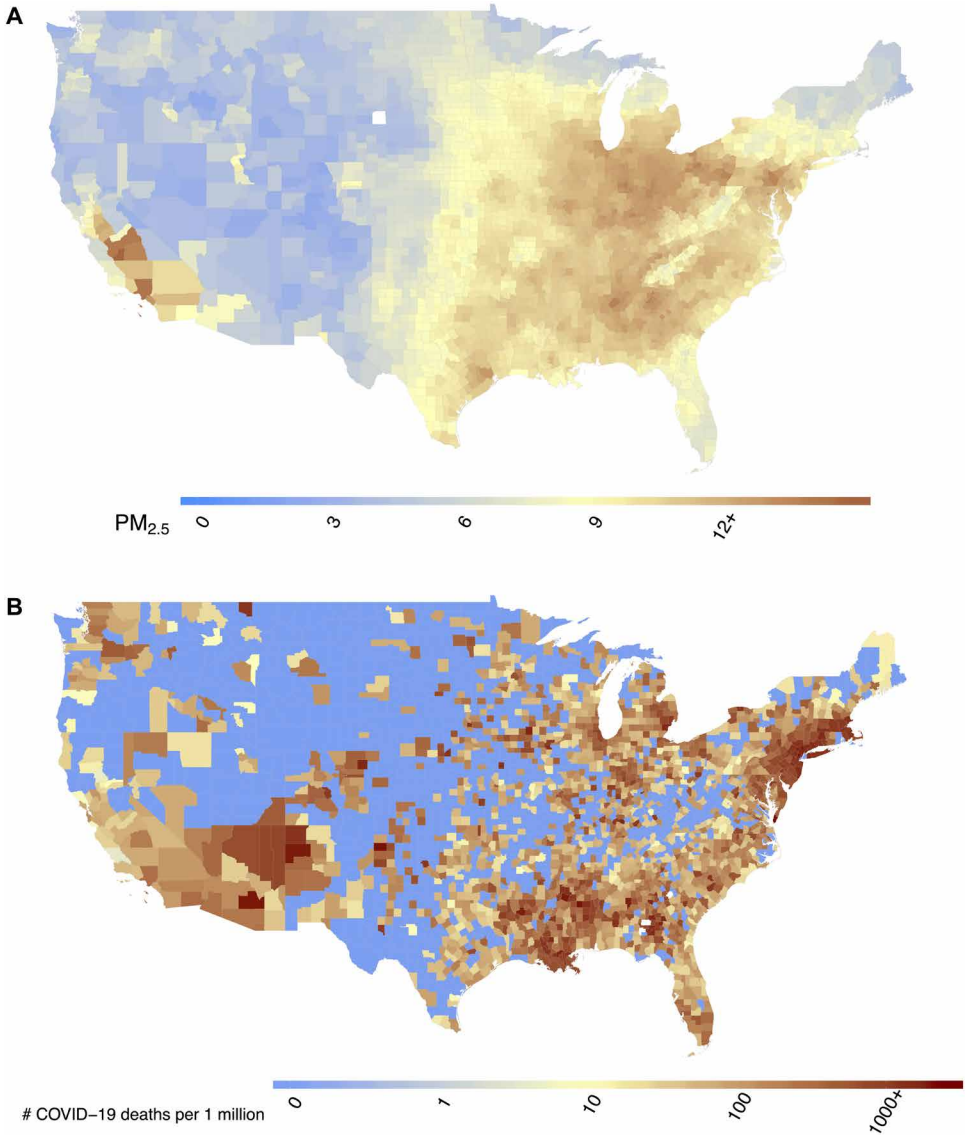

Fig. 1. National maps of historical $PM_{2.5}$ concentrations and COVID-19 deaths. Maps show (**A**) county-level 17-year long-term average of $PM_{2.5}$ concentrations (2000–2016) in the United States in $\mu g/m^3$ and (**B**) county-level number of COVID-19 deaths per 1 million population in the United States up to and including 18 June 2020.

education, age distribution, and percent of Black residents are important predictors of the COVID-19 mortality rate in the model. We found a 49% (95% CI, 38 and 61%) increase in COVID-19 mortality rate associated with a 1-SD (per 14.1%) increase in percent Black residents of the county. Details on the data sources, statistical methods, and analyses are summarized in the Supplementary Materials. All data sources used in the analyses, along with fully reproducible code, are publicly available at https://github.com/wxwx1993/PM_COVID.

Strengths and limitations of an ecological regression analysis

Ecological regression analysis provides a simple and cost-effective approach for studying potential associations between historical exposure to air pollution and increased vulnerability to COVID-19 in large representative populations, as illustrated in our study in the previous section. This approach is regularly applied in many areas of research (*10*). Using our study as an example, we summarize in Table 2 the strengths, limitations, and opportunities considering (i) study design, (ii) COVID-19 health outcome data, (iii) historical exposure to air pollution, and (iv) measured and unmeasured confounders, with the goal of paving the way for future research.

Among the key limitations, by design, ecological regression analyses are unable to adjust for individual-level risk factors (e.g., age, race, and smoking status); when individual-level data are unavailable, this approach leaves us unable to make conclusions regarding individual-level associations. In the context of COVID-19 health outcomes, this is a severe limitation, as individual-level risk factors are known to affect COVID-19 health outcomes. It is important to note that confusion between ecological associations and individual associations may present an ecological fallacy. In extreme

cases, this fallacy can lead to associations detected in ecological regression that do not exist or are in the opposite direction of true associations at the individual level. However, ecological regression analyses still allow us to make conclusions at the area level, which can be useful for policy-making (*11*). For the association between COVID-19 health outcomes and $PM_{2.5}$ exposure, we argue that area-level conclusions are valuable, as they can inform important immediate policy actions that will benefit public health, such as (i) prioritization of precautionary measures [e.g., personal protective equipment (PPE) allocations and hospital beds] to areas with historical higher air pollution and (ii) further strengthening the scientific argument for lowering the U.S. National Ambient Air Quality Standards for $PM_{2.5}$ and other pollutants. To completely avoid potential ecological bias, a representative sample of individual-level data is necessary. While this may not be feasible in the near future, as some COVID-19 outcome data become available at the individual level, existing approaches that augment county-level data with individual-level data (*12*) could be used to correct for ecological bias.

Furthermore, air pollution exposure misclassification, due to between-area mobility and within-area variation, is another potential source of bias that could affect the ecological regression results described in our example study. Methods to account for the propagation of exposure error into the ecological regression model (*13*) could be applied to help mitigate the impact of measurement error. Outcome misclassification is another limitation that can be partially overcome by accessing nationwide registry data with the validated cause of death (*14*). As in all observational studies, adjustment for measured and unmeasured confounding presents another key challenge in ecological regression analyses, which may be exacerbated when dealing with dynamic pandemic data, as in our study. Conducting studies using both traditional regressions and methods for causal inference as in Wu *et al.* (*2*) is necessary to assess the robustness of the findings.

Increasing the scientific rigor of research in this area requires access to representative, individual-level data on COVID-19 health outcomes, including information about patients' residential address, demographics, and individual-level confounders. This is an enormous challenge that will require consideration of many privacy, legal, and ethical trade-offs (*14*). Future areas of research also include the application of statistical methods to quantify and correct for ecological bias and measurement error, reproducible methods for causal inference, and sensitivity analysis of measured and unmeasured confounding bias as suggested above. These strengths and limitations are illustrated further in the context of our own study (see the Supplementary Materials).

Table 1. Mortality rate ratios (MRR), 95% confidence intervals (CI), and *P* values for all variables in the main analysis. Details of the statistical models are available in section S2. Q, quintile.

	MRR	95% CI	*P* value
$PM_{2.5}$	1.11	(1.06–1.17)	0.00
Population density (Q2)	0.91	(0.71–1.15)	0.42
Population density (Q3)	0.91	(0.71–1.16)	0.45
Population density (Q4)	0.74	(0.57–0.95)	0.02
Population density (Q5)	0.92	(0.69–1.23)	0.56
% In poverty	1.04	(0.96–1.12)	0.31
Log(median house value)	1.13	(0.99–1.29)	0.07
Log(median household income)	1.19	(1.04–1.35)	0.01
% Owner-occupied housing	1.12	(1.04–1.20)	0.00
% Less than high school education	1.20	(1.10–1.32)	0.00
% Black	1.49	(1.38–1.61)	0.00
% Hispanic	1.06	(0.97–1.16)	0.23
% ≥ 65 years of age	1.04	(0.93–1.17)	0.46
% 45–64 years of age	0.77	(0.67–0.90)	0.00
% 15–44 years of age	0.76	(0.68–0.85)	0.00
Days since stay-at-home order	1.18	(0.92–1.52)	0.20
Days since first case	2.40	(2.05–2.80)	0.00
Rate of hospital beds	1.00	(0.93–1.08)	0.95
% Obese	0.96	(0.90–1.03)	0.32
% Smokers	1.13	(1.00–1.28)	0.05
Average summer temperature (°F)	1.11	(0.95–1.30)	0.20
Average winter temperature (°F)	0.86	(0.69–1.07)	0.19
Average summer relative humidity (%)	0.93	(0.80–1.09)	0.38
Average winter relative humidity (%)	0.97	(0.87–1.07)	0.52

DISCUSSION

Ecological regression analyses are crucial to stimulate innovations in a rapidly evolving area of research. Ongoing research has already focused on overcoming some aspects of these limitations (*8*, *15*). For example, ecological regression analysis of air pollution and COVID-19, using data with finer geographic resolution, is being conducted for different countries and regions around the world. Cole *et al.* (*8*) published an ecological regression analysis using data in Dutch municipalities and found results consistent with our own investigation; the California Air Resources Board (CARB) is planning to conduct a similar study at the census tract level (*15*). Although an ecological regression analysis cannot provide insight into the mechanisms underlying the relationship between $PM_{2.5}$ exposure and COVID-19 mortality, studies are starting to shed light on the potential biological mechanisms that may explain the relationship between air pollution and viral infection outcomes (*16*). For example, it has been hypothesized that chronic exposure to $PM_{2.5}$ causes alveolar angiotensin-converting enzyme 2 (ACE-2) receptor overexpression and impairs host defenses (*17*). This could cause a more severe form

Table 2. Strengths and limitations of ecological regression analyses applied to research on air pollution and COVID-19 and opportunities for future research.

	Strengths	Limitations	Future research
Study design: ecological regression	Feasible, timely, and cost-effective	Cannot be used to make inference about individual-level associations, doing so leads to ecological fallacy	Augment county-level data with individual-level data to adjust for ecological bias (*12*)
	Data are representative of the entire U.S. population	Cannot adjust for individual-level risk factors such as age, gender, and race (*19*–*21*)	Conduct studies of individual-level health records using traditional regression and causal inference methods as in Wu *et al.* (*2*)
	Allows inference at the area level, which can be useful for policy-making (*11*)	Results are sensitive to the assumptions of the statistical model (*11*)	
	Computationally efficient and can be conducted daily to allow for the dynamic nature of the data and observe temporal trends; see fig. S3		
	Facilitates comparison of results across countries		
Outcome: COVID-19 deaths aggregated at the county level	Publicly available data updated almost daily	Potential for outcome misclassification (*22*), particularly differential misclassification over time and space, which could bias results	Access to nationwide registry data with the validated cause of death (*14*)
			Analyses using county excess deaths as the outcome (*23*)
Exposure: 2000–2016 average exposure to $PM_{2.5}$ at the county level	Use of well-validated atmospheric chemistry models and machine learning models (*9*, *24*)	Aggregation assumes that everyone in a county experiences the same exposures, leading to exposure misclassification, especially for the largest counties	Individual-level data on COVID-19 deaths with geocoded addresses to link to air pollution data at the place of residence
	$PM_{2.5}$ exposure estimated at fine grids, which can be aggregated to the county level to assess exposure even in unmonitored areas (*24*)	Can be used to assess historical exposures to air pollution but not real-time exposures	Additional statistical methods to account for the propagation of exposure error into the ecological regression model (*13*)
	As opposed to using monitor data, aggregation of modeled estimates ensures that county $PM_{2.5}$ exposure estimates represent the distribution across the entire area		
Measured confounders	More than 20 area-level variables capture age distribution, race distribution, socioeconomic status, population density, behavioral risk factors, epidemic stage, and stay-at-home orders (see tables S1 and S2)	County average features may not represent the features of COVID-19 patients, leading to inadequate adjustment	Causal inference approaches to adjust for measured confounding bias, producing results that are less sensitive to statistical modeling assumptions
	These overlap with the confounder sets used in much of the previous literature on air pollution and health (*25*, *26*)	Difficult to formalize the notion of "epidemic stage," which may be an important confounder	
		The threat of unmeasured confounding bias still present	Causal inference approaches to assess covariate balance (*2*)
		Sensitive to the form of the statistical model specified (i.e., assumptions of linearity and no effect modification)	Individual-level data on key measured confounders such as smoking and body mass index
Unmeasured confounders	Leverage existing approaches, such as the calculation of the E-value (*27*), to assess how strong the effect of an unmeasured confounder would need to be to explain away the associations detected (see section S3)	The most important threat to the validity of any observational study Even measures like the E-value cannot inform us about the likelihood that a strong unmeasured confounder exists; this must be evaluated on the basis of subject matter knowledge	Natural experiment designs and instrumental variables can be used to reduce the threat of unmeasured confounding but are less common

of COVID-19 in ACE-2–depleted lungs, increasing the likelihood of poor outcomes, including death (*18*).

The associations detected in ecological regression analyses provide strong justification for follow-up investigations as more and higher-quality COVID-19 data become available. Such studies would include validation of our findings with other data sources and study types, as well as investigations into mediating factors and effect modifiers, biological mechanisms, impacts of $PM_{2.5}$ exposure timing, and relationships between $PM_{2.5}$ and other COVID-19 outcomes such as hospitalization. Research on how modifiable factors may exacerbate COVID-19 symptoms and increase mortality risk is essential to guide policies and behaviors to minimize fatality related to the pandemic. Such research could also provide a strong scientific argument for revision of the U.S. Ambient Air Quality Standards for $PM_{2.5}$ and other environmental policies in the midst of a pandemic.

SUPPLEMENTARY MATERIALS

Supplementary material for this article is available at http://advances.sciencemag.org/cgi/content/full/6/45/eabd4049/DC1

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Acknowledgments: The computations in this paper were run on (i) the Odyssey cluster supported by the FAS Division of Science, Research Computing Group at Harvard University and (ii) the Research Computing Environment supported by Institute for Quantitative Social Science in the Faculty of Arts and Sciences at Harvard University. We gratefully acknowledge support from the 2020 Star-Friedman Challenge for Promising Scientific Research, the Climate Change Solutions Fund at Harvard University, and the Fernholz Foundation. We would like to thank L. Goodwin and S. Tobin for editorial assistance in the preparation of this manuscript. **Funding:** This work was made possible by support from NIH grants R01 ES024332-01A1, P50MD010428, RO1 ES026217, RO1 ES028033, R01 ES030616, R01 AG066793-01, and R01 MD012769; Health Effects Institute grant (HEI) 4953-RFA14-3/16-4: and US EPA grant 83587201-0. The funding sources did not participate in the design or conduct of the study; collection, management, analysis, or interpretation of the data; or preparation, review, or approval of the manuscript. The research described in this article was conducted under contract to the HEI, an organization jointly funded by the EPA (Assistance Award No. R-83467701), and certain motor vehicle and engine manufacturers. The contents of this article do not necessarily reflect the views of HEI or its sponsors, nor do they necessarily reflect the views and policies of the EPA or motor vehicle and engine manufacturers. **Author contributions:** X.W. and R.C.N. contributed equally to the paper. X.W. and R.C.N. contributed to formulation of the idea, data preparation, data analysis, data interpretation, and writing of the manuscript. M.B.S. and D.B. contributed to data preparation, data interpretation, and review of the manuscript. F.D. contributed to formulation of the idea, study design, data interpretation, funding, and writing of the manuscript. All authors contributed to the interpretation of the results and critical revision of the manuscript for important intellectual content and approved the final version of the manuscript. The corresponding author attests that all listed authors meet authorship criteria and that no others meeting the criteria have been omitted. F.D. is the guarantor. **Competing interests:** The authors declare that they have no competing interests. **Data and materials availability:** All data needed to evaluate the conclusions in the paper are present in the paper and/or the Supplementary Materials. Data and code are publicly available at https://github.com/wxwx1993/PM_COVID. Additional data related to this paper may be requested from the authors.

Submitted 25 June 2020
Accepted 18 September 2020
Published 4 November 2020
10.1126/sciadv.abd4049

Citation: X. Wu, R. C. Nethery, M. B. Sabath, D. Braun, F. Dominici, Air pollution and COVID-19 mortality in the United States: Strengths and limitations of an ecological regression analysis. *Sci. Adv.* **6**, eabd4049 (2020).

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Air pollution and COVID-19 mortality in the United States: Strengths and limitations of an ecological regression analysis

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Sci. Adv., 6 (45), eabd4049. • DOI: 10.1126/sciadv.abd4049

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SPECIAL REPORT

Wildfires, Global Climate Change, and Human Health

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The world has already observed many devastating effects of human-induced climate change.[1] A vivid manifestation is the several large wildfires that have occurred recently — in some cases, fires of unprecedented scale and duration — including wildfires in Australia in 2019 to 2020, the Amazon rainforest in Brazil in 2019 and 2020, the western United States in 2018 and 2020, and British Columbia, Canada, in 2017 and 2018. Since August of this year, record-breaking wildfires have burned 2.7 million hectares (as of September 18, 2020) along the West Coast of the United States, killing more than 30 people and leaving tens of thousands homeless.[2] Robust projections indicate that the risk of wildfires will continue to increase in most areas of the world as climate change worsens[3-6] and that the fires will increase excess mortality and morbidity from burns, wildfire smoke, and mental health effects.[7-9]

Substantial greenhouse-gas emissions and forest loss from wildfires are likely to accelerate climate change further and possibly lead to a reinforcing feedback loop.[3] This report summarizes the status of wildfires under climate change, current knowledge and gaps about the health risks of wildfires, and challenges of developing and implementing strategies for reducing associated health risks.

CLIMATE CHANGE AND WILDFIRES

For a wildfire to start, three essential conditions (known as the fire triangle) are needed: fuel, oxygen, and an ignition source.[10] Climate change can increase the chances that each of these will be present.

Climate change–related rainfall anomalies can intensify drought in tropical and subtropical areas.[1] Rainfall is becoming more concentrated in winter, making other seasons, especially summer, hotter and drier.[1,3,11] An increase in the evaporation of moisture in soil during dry periods leads to an increase in flammable vegetation that can fuel wildfires, under the assumption that forest management is unchanged.

The global surface wind speed has increased substantially since 2010, after three decades of decrease. This shift is driven mainly by ocean–atmosphere oscillations, such as El Niño events, which might be related to climate change.[12,13] Climate change is projected to enhance differences in temperature between the land and the sea, resulting in greater land–sea differences in air pressure, which boost wind power in tropical and southern subtropical areas.[14] Strong winds provide more oxygen for wildfires and encourage their spread, potentially outstripping firefighting capability.[10]

Increases in the frequency and intensity of heat waves under climate change provide more ignition sources for wildfires.[6,10] Climate change also affects lightning strikes, another important ignition source.[3,15] A study of cloud ice fluxes — changes in the mass of ice particles in clouds over time, which are positively correlated with lightning strikes — projected an overall decrease in lightning strikes, especially in tropical regions, but a likely increase over North America and Siberia.[15]

Furthermore, the wildfire season is starting much earlier and ending later because of a warming climate.[3,6] Consequently, there is a wider window in which wildfires can occur and a narrower window for prescribed burning — deliberate burning of available vegetation during cooler seasons, which is an essential strategy to reduce the risk of wildfires.[3]

Fire suppression and the conversion of tropical savannas and grasslands to agricultural lands have resulted in a decline of approximately 30% in the overall global area of land burned by wild-

fires since 1930, but the area of land burned in dense forests has increased.[16] Deliberate setting of fires to convert tropical forest to open lands (e.g., agricultural lands, cattle ranches, and lands for real-estate speculation) contributes to climate change and to the associated disease burden through large emissions of greenhouse gases and air pollutants.[1,3,16] Although wildfires and climate change could reduce the availability and growth of vegetation, the risk and severity of wildfires in forests (often alongside human activities) and the area of land burned are expected to increase in the future.[3-6]

The interplay between climate change and wildfires could be reinforcing and synergistic (Fig. 1). From 1997 to 2016, the global mean carbon dioxide emissions from wildfires equated to approximately 22% of the carbon emissions from burning fossil fuels.[3] Forest loss in tropical areas due to wildfires damages the Earth's ability to absorb carbon dioxide and to cool the climate.[18] Wildfires in the Arctic and boreal forest ecosystem could melt the permafrost in that region directly and lead to the release of previously frozen carbon and methane, which is a stronger greenhouse gas than carbon dioxide.[3]

HEALTH RISKS ASSOCIATED WITH WILDFIRES

The health risks associated with wildfires include direct risks from exposure to fires or involvement in wildfire events, as well as risks from wildfire smoke (Fig. S1 in the Supplementary Appendix, available with the full text of this article at NEJM.org).

DIRECT HEALTH RISKS FROM WILDFIRE EVENTS

For firefighters and people living near wildfires, direct health effects include burns, injuries,

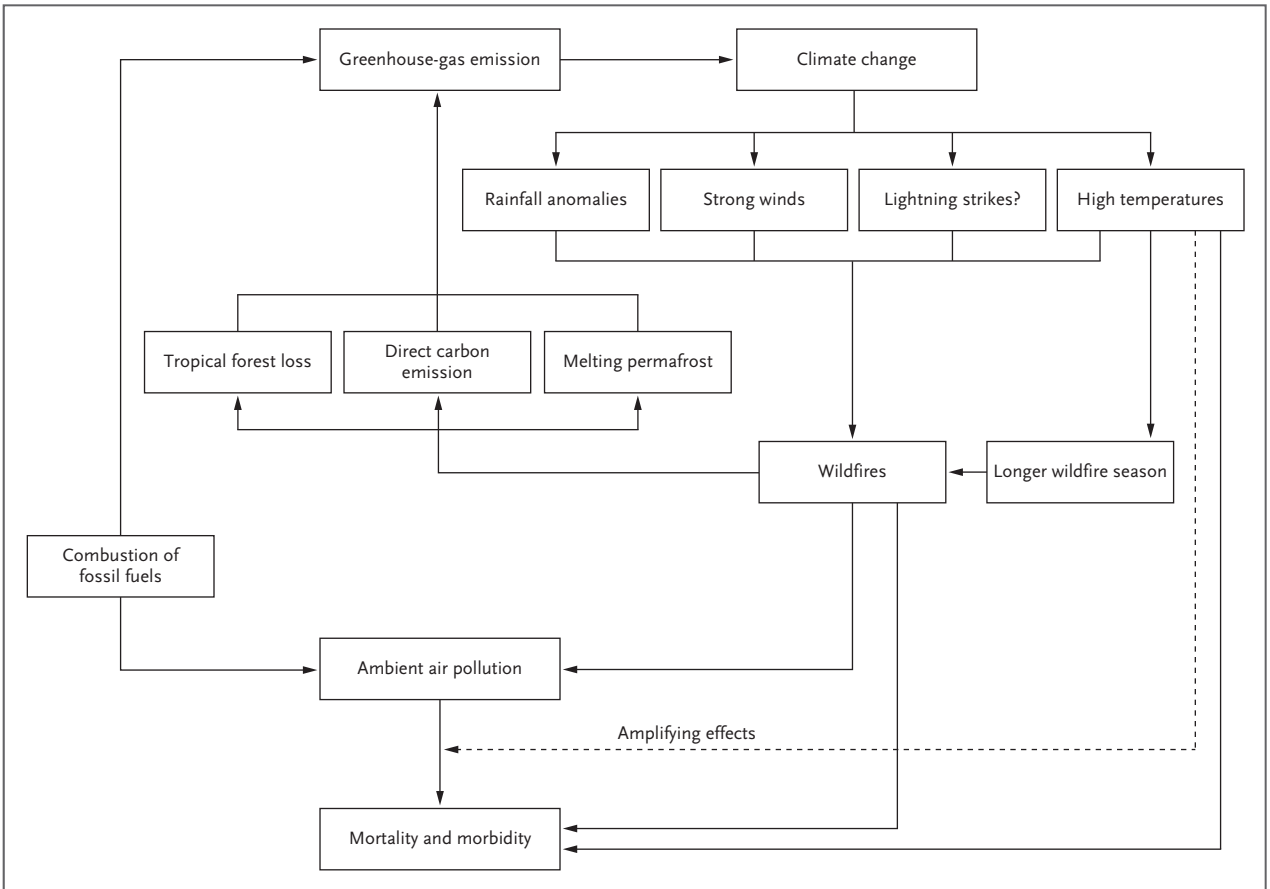

Figure 1. Potential Reinforcing Feedback Loop of Climate Change, Wildfires, and Health Risks.

The dashed line indicates that high temperatures could amplify, or enhance, the effects of ambient air pollution on mortality and morbidity.[17]

mental health effects, and death due to exposure to flames or radiant heat.[7] For example, the 2009 "Black Saturday" wildfires in Australia killed 173 people directly; in the first 72 hours, 146 patients with burns and 64 with physical trauma presented to local emergency departments.[19] In addition, firefighters are at high risk for heat-related illnesses ranging from dehydration-induced heat cramps to life-threatening heat stroke.[20]

Owing to traumatic experiences, property loss, and displacement, residents in areas affected by wildfires are at an increased risk for mental illness, including post-traumatic stress disorder, depression, and insomnia.[21] The psychological consequences of wildfire events can persist for years,[22] and children and adolescents are particularly vulnerable.[23] A 20-year follow-up study showed that exposure to wildfires in childhood was associated with an increased likelihood of mental illness in adulthood.[24] Furthermore, wildfire events have been associated with a subsequent decrease in academic performance in children.[25]

HEALTH RISKS FROM WILDFIRE SMOKE

In areas surrounding a wildfire, heavy smoke can cause eye irritation and corneal abrasions and can substantially reduce visibility, increasing the risk of traffic accidents.[7] As far as 1000 km away, wildfire smoke can increase ambient air pollution,[26] along with associated risks of illness and death.

Air Pollutants from Wildfire Smoke

The primary air pollutants from wildfire smoke are particulate matter; carbon monoxide; nitrogen oxides, including nitrogen dioxide and nitric oxide; and volatile organic compounds.[27,28] A photochemical reaction between volatile organic compounds and nitrogen oxides under sunlight generates a secondary pollutant, ground-level ozone.[27] Peat fires, such as those that occurred in Indonesia during the 2015 El Niño event, may extend up to 20 m underground and result in an extraordinarily high level of air pollution, including high emissions of carbon dioxide and many potentially toxic compounds, such as formaldehyde and hydrogen cyanide.[29]

The major pollutants of public health concern during wildfire events are carbon monoxide, ozone, and particulate matter.[10] Increases in carbon monoxide are usually restricted to the areas that are directly affected by the fire, but ozone and particulate matter spread much farther.[28] Wildfire smoke is an increasingly important source of ambient air pollution in the United States, where industrial emissions of air pollutants are declining.[30] In the United States between 1997 and 2016, wildfires were a contributing factor on approximately 10% of the days that the surface ozone level exceeded the 8-hour standard (70 parts per billion).[28] Most studies evaluating the health effects of wildfire smoke have focused on the health risks associated with wildfire particulate matter with a diameter of 10 μm or less (PM_{10}) (Table 1). PM_{10} includes fine particles (diameter, ≤2.5 μm [$PM_{2.5}$]), submicronic particles (diameter, ≤1 μm [PM_{1}]), and ultrafine particles (diameter, ≤0.1 μm [$PM_{0.1}$]); smaller particle size is correlated with a greater toxic effect.[35] Although it is clear that urban background $PM_{2.5}$ has major effects on human health, the evidence specifically for wildfire $PM_{2.5}$ is more limited.

Short-Term Health Effects of Wildfire Smoke

Studies suggest a consistent association between the level of particulate matter during wildfire events and the risk of death from any cause or nonaccidental death, but the association between the level of wildfire particulate matter and the risk of death from specific causes (e.g., respiratory or cardiovascular causes) remains uncertain, possibly because of limited sample sizes (details are provided in Table S1).[8,9,36] In the vicinity of the 2020 California wildfires, the daily mean $PM_{2.5}$ level has often reached 350 to 500 μg per cubic meter, far exceeding the 24-hour standard in the United States (35 μg per cubic meter); as far as 1000 km away from the fires, the daily mean $PM_{2.5}$ level has reached 35 to 150 μg per cubic meter.[2] During wildfire events, each increase of 10 μg per cubic meter in the daily $PM_{2.5}$ level and in the daily PM_{10} level has been associated with an increase of 0.8 to 2.4% and 0.8 to 3.5%, respectively, in the risk of death from any cause or nonaccidental death for up to 4 days after the exposure.[8,9,36] In comparison, in a recent global study, the same change in the daily $PM_{2.5}$ level and the daily PM_{10} level (regardless of the source, with mainly urban sources) was associated with an increase of 0.68% and 0.44%, respectively, in the daily risk of death from any cause.[37] Although this comparison does not account for location-specific modifying factors (e.g., socioeconomic and climatic factors),[37] it suggests that wildfire particulate

Table 1. Characteristics and Health Risks of Wildfire Particulate Matter.*

Feature	Description
Source	Wildfire particulate matter results from combustion of biomass.[27,28]
Particle size	The particles are smaller than those in particulate matter from urban sources (i.e., with a higher proportion of $PM_{2.5}$ and PM_1 in PM_{10}).[31]
Contribution to ambient particulate matter	In the continental United States in 2000 to 2016, wildfires were a contributing factor on 20% of the days that the daily $PM_{2.5}$ level exceeded the 24-hour standard (35 μg per cubic meter).[30] During the 2019–2020 Australian wildfire, the daily $PM_{2.5}$ level reached 600 μg per cubic meter in Sydney.[32]
Components and toxic effects	As compared with urban background particulate matter, wildfire particulate matter that reaches urban areas may contain more oxidative components (e.g., oxygenated PAHs and quinones) and proinflammatory components (e.g., aldehydes and oxides of nitrogen) and may have greater oxidative potential.[33] As wildfire smoke ages, the oxidative potential can more than double.[34] When wildfire particulate matter reaches urban areas, toxic effects on macrophage cells could be 5 times as intense as effects with the same dose of urban particulate matter, but the effects may vary according to combustion conditions and type of burned vegetation.[35]
Short-term health effects	
Mortality	There is consistent evidence of an increased risk of death from any cause but uncertain evidence of an increased risk of death from specific causes.[8,9,36] Wildfire particulate matter may have a stronger effect on mortality than urban particulate matter,[8,9,36,37] owing to the smaller particle size,[31] more abundant oxidative and proinflammatory components,[33] and amplifying effects of high temperature[17] and ozone.[38]
Morbidity	There is consistent evidence of an increased risk of respiratory events, including hospitalizations and emergency department visits due to asthma, chronic obstructive pulmonary disease, and respiratory infection.[8,9,36,39] Wildfire particulate matter has a stronger effect on the risk of asthma-related events than urban particulate matter.[33,40,41] Data are inconsistent regarding the risk of cardiovascular events,[8,9,36] but the effect may be similar to that of urban particulate matter.[41]
Risk of other health effects	Risks of low birth weight and preterm birth are increased.[8,9] Rates of influenza are increased.[42] Ambulance dispatches among people with diabetes are increased.[43]
Long-term health effects	Effects are largely unknown; wildfire particulate matter might impair lung capacity, self-reported general health, and physical functioning several years later.[44]
Vulnerable populations	Older adults, children, and pregnant women are more susceptible. People with preexisting cardiac or respiratory conditions (or both) have increased risks. People living in low-income areas have increased risks. Outdoor workers have increased exposure.

* Details regarding the short-term health effects of wildfire particulate matter are provided in Table S1 in the Supplementary Appendix, available at NEJM.org. Particulate matter with a diameter of 10 μm or less (PM_{10}) includes fine particles (diameter, ≤2.5 μm [$PM_{2.5}$]), submicronic particles (diameter, ≤1 μm [PM_1]), and ultrafine particles (diameter, ≤0.1 μm [$PM_{0.1}$]). PAH denotes polycyclic aromatic hydrocarbon.

matter could be more lethal than urban particulate matter.

As compared with urban background particulate matter, which results mainly from the combustion of fossil fuels, wildfire particulate matter tends to have a smaller particle size[31] and to contain more oxidative components (e.g., oxygenated polycyclic aromatic hydrocarbons and quinones) and proinflammatory components (e.g., aldehydes and oxides of nitrogen),[33] features that potentially lead to stronger toxic effects.[35] In addition, the high temperatures that often accompany wildfires and the oxidant gases from wildfires (ozone and nitrogen dioxide) can amplify the health risks of wildfire particulate matter.[17,38]

Exposure to wildfire particulate matter is associated with an increased risk of respiratory events, including impaired lung function and hospitalizations, emergency department visits, physician visits, and medication use for asthma, chronic obstructive pulmonary disease, and respiratory infection (Table S1).[8,9,36,39] The associa-

tion with the risk of asthma-related events has been the strongest and most consistent.[39] Studies also suggest that exposure to wildfire particulate matter might have a stronger effect on the risk of asthma-related events than exposure to urban particulate matter, probably because of the more abundant oxidative and proinflammatory components in wildfire particulate matter.[33,40,41]

There is an inconsistent association between wildfire particulate matter and cardiovascular events (Table S1). Observational studies showed that the association was often not significant, but in many of the studies, power was limited by a relatively small number of cardiovascular events during wildfire periods.[8,9,36] In a large study that analyzed 2.5 million hospitalizations for cardiovascular diseases among Medicare recipients (≥65 years of age) in the United States who were living within 200 km of large wildfires, increases in cardiovascular risk associated with wildfire particulate matter were similar to those associated with urban particulate matter.[41] A small randomized, double-blind, crossover trial showed adverse effects of acute (3-hour) exposure to woodsmoke on central arterial stiffness and heart-rate variability.[45]

Limited data support associations between wildfire particulate matter and adverse pregnancy outcomes (e.g., low birth weight and preterm birth; Table S1),[8,9] increased rates of influenza,[42] and increased ambulance dispatches for patients with diabetes mellitus.[43] Other short-term health effects of exposure to wildfire particulate matter remain largely unexplored.

Few studies have evaluated the health effects of gaseous air pollutants from wildfire smoke other than particulate matter, mainly ozone and carbon monoxide.[8,9,36,39,46] Carbon monoxide poisoning is a potential concern for residents and firefighters during wildfire events.[28,47] The secondary pollutant ozone can travel much farther[28] and should be considered when evaluating the health risks of wildfire smoke.[46]

Long-Term Health Effects of Wildfire Smoke

Data are lacking to quantify the long-term health risks of wildfire smoke. In one study with follow-up data obtained 10 years after the 1997 Indonesian forest fires,[44] people who had been exposed to wildfire smoke had poorer results for lung capacity, self-reported general health, and physical functioning than those who had not been exposed.[44]

Vulnerable Populations Affected by Wildfire Smoke

Populations that are particularly vulnerable to adverse effects of wildfire smoke include people 65 years of age or older, who have an increased risk of short-term respiratory events[40,48]; people with preexisting cardiac or respiratory conditions (or both) and people living in low-income areas, who have an increased risk of short-term cardiopulmonary events[48-50]; and pregnant women, who have a risk of adverse pregnancy outcomes.[8,9] Outdoor workers are also a high-risk group, owing to their increased exposure to wildfire smoke. It is hypothesized that children are more susceptible to harm from wildfire smoke than adults because they have less mature respiratory and immune systems, have a higher breathing rate relative to body size, and spend more time outdoors.[51] Priority should be given to these vulnerable populations when implementing strategies to reduce the health risks of wildfire smoke (e.g., staying indoors or using air cleaners).

PROTECTING HEALTH AGAINST WILDFIRES

It is important for residents in areas affected by wildfires to keep track of reliable information and community evacuation plans during the wildfire season and to gather emergency supplies (e.g., food, water, medication, and N95 or P100 face masks) before wildfires occur.[10] When evacuation is required, it is important to drive with caution in conditions of low visibility.[7] People who present with eye irritation should be screened for corneal abrasions, if possible.[7] Careful triage and planning for each patient before hospitalization can improve the ability of surrounding hospitals to manage increased patient loads.[19]

Personal protective equipment, rest periods, adequate hydration, and health awareness are vital for preventing heat-related illnesses in firefighters.[7,20] Psychological support services are important for addressing mental health effects during and after wildfires, especially in children and the most affected communities.[7,21-24] Wildfire ash, which contains polycyclic aromatic hydrocarbons and heavy metals, can heavily pollute the water and land in affected communities, and these areas must be cleaned after the event, in accordance with guidelines.[7,10] During and after wildfire events, residents in affected areas should avoid drinking from water supplies that could be contaminated by wildfire ash, fire retardant, dead

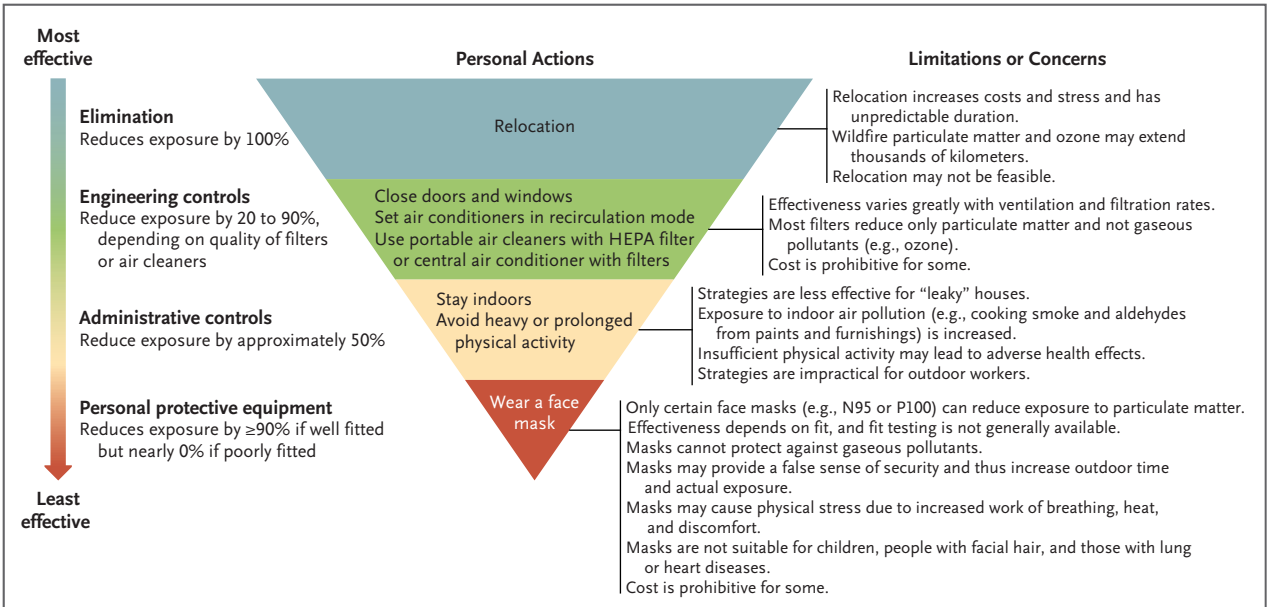

Figure 2. Main Actions That Individual People Can Take to Reduce Exposure to Wildfire Smoke and Its Health Risks.

Data are adapted from the Centers for Disease Control and Prevention,[10] Vardoulakis et al.,[32] and Laumbach.[53] The strategies are organized according to the hierarchy of controls proposed by the National Institute for Occupational Safety and Health (NIOSH).[53] The use of N95 or P100 face masks certified by the NIOSH or their potential equivalents (e.g., KN95 or P95 masks) is recommended. Recommendations regarding the use of face masks, air conditioning, and air cleaners are provided in the Supplementary Appendix, available at NEJM.org. HEPA denotes high-efficiency particulate air.

animals, or damaged water pipes, until testing confirms that the water is safe to drink.[52]

Public agencies are responsible for releasing accurate and clear information regarding air quality and advice regarding health protection against wildfire smoke.[10,32] Residents should keep track of the air quality and adjust their behavior accordingly.[10] When air-quality data are not available, residents should "trust their senses" — that is, use risk-reduction strategies when smoke can be smelled or seen or when visibility is substantially reduced, even when a wildfire is at a distance.[32] Key strategies that individual people can use to minimize health risks associated with wildfire smoke are summarized in Figure 2.[10,32,53]

However, all these strategies have limitations. For example, wearing an N95 or P100 face mask can cause physical stress from increased work of breathing, heat, and discomfort, particularly in the hot weather that is common during wildfire events.[53] Both central air conditioners with high-efficiency filters and portable air cleaners with high-efficiency particulate air (HEPA) filters can reduce indoor levels of $PM_{2.5}$ efficiently, but neither can remove gaseous pollutants, and some electronic air cleaners (e.g., some electrostatic precipitators and ionizers) could even generate ozone.[10] Air cleaners or filters that are designed for removing gaseous pollutants remain limited. The most widely used activated carbon filters can clean volatile organic compounds and odors but not ozone (details are provided in the Supplementary Appendix). Cost is also a concern, especially in the low-income population, given that air cleaners that cost less than $200 are often ineffective in removing air pollutants.[10]

It has been proposed that the use of rescue medications might decrease the respiratory effects of wildfire smoke among children with asthma.[54] However, data are lacking to inform the effectiveness of such medications in this population or in other people with chronic conditions (e.g., asthma, chronic obstructive pulmonary disease, or heart diseases) after exposure to wildfire smoke.

MITIGATING WILDFIRE RISKS BY LIMITING GLOBAL TEMPERATURE INCREASE

Projections indicate that, in a scenario of high greenhouse-gas emissions, the frequency of wild-

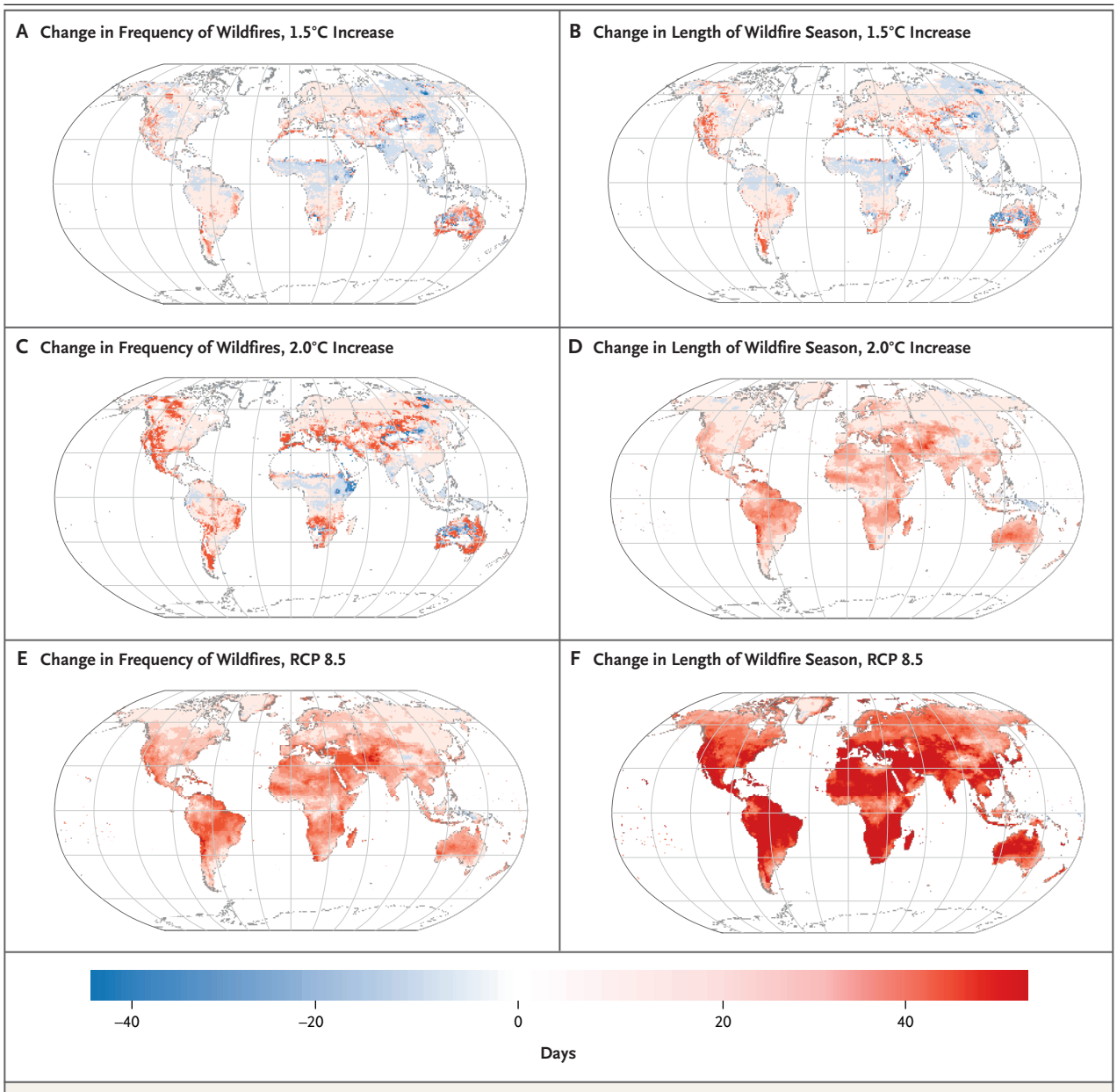

Figure 3. Projected Change from 1981–2000 to 2080–2099 in Frequency of Wildfires and Length of Wildfire Season, According to Global Mean Surface-Temperature Increase.

Adapted from Sun et al.[6] Shown is the projected change from 1981–2000 to 2080–2099 in the frequency of wildfires (days with wildfire events per year) and the length of the wildfire season (days with a normalized daily fire danger index value above a threshold of 50 per year) with an increase in the global mean surface temperature of 1.5°C (Panels A and B, respectively) and with an increase of 2.0°C (Panels C and D, respectively) relative to the preindustrial level. Also shown is the projected change under the conditions of representative concentration pathway (RCP) 8.5 (Panels E and F, respectively), which is a future scenario of high greenhouse-gas emissions and no climate change–mitigation policy, with an increase in the global mean surface temperature of 3.2°C to 5.4°C relative to the preindustrial level (corresponding to an increase of 2.2°C to 4.4°C relative to the 2019 level). Details are provided in the Supplementary Appendix.

fires will substantially increase over 74% of the global land mass by the end of this century.[6] However, if immediate climate change–mitigation steps are taken to limit the global mean temperature increase to 2.0°C or 1.5°C above the preindustrial level, then 60% or 80%, respectively, of the increase in wildfire exposure could be avoided (Fig. 3).[6] Reaching the 1.5°C target

would require reducing global net anthropogenic carbon dioxide emissions from 2010 levels by approximately 45% by 2030 and reaching "net zero" by around 2050.[1] The 1.5°C target remains achievable if carbon dioxide emissions decline by 7.6% per year from 2020 to 2030.[55]

Cutting carbon emissions may appear to be difficult and costly, but its near-term benefits outweigh its costs in many areas.[56] Even only accounting for the improved air quality due to the reduction in burning fossil fuels, the cost savings associated with reduced mortality and morbidity from exposure to $PM_{2.5}$ and ozone is estimated to be 1.40 to 2.45 times as high as the cost of reducing carbon emissions, albeit with considerable regional variation.[57] The long-term benefits of avoiding health and other risks of climate change, including those associated with wildfires, are additional motivations for urgent climate actions.

As a trusted source, health professionals are responsible for educating the public about the health risks of wildfires and risk-reduction strategies. They can also focus on reducing the carbon intensity of health care systems and advocate for lifestyles, actions, and policies with low environmental impact, such as the rapid transition to renewable energy.[56]

CONCLUSIONS

Wildfires are associated with increased morbidity and mortality, but there are many gaps in knowledge regarding their health effects. At the individual level, people can do little to reduce the adverse health consequences of exposure to wildfires. Societal action is requisite. Without immediate actions to limit the global temperature increase, the interplay between wildfires and climate change is likely to form a reinforcing feedback loop, making wildfires and their health consequences increasingly severe.

Supported by China Scholarship Council funds (201806010405 to Dr. Xu and 201906210065 to Dr. Yu), the Australian National Health and Medical Research Council (Early Career Fellowship APP1109193 to Dr. Li and Career Development Fellowship APP1107107 to Dr. Guo), and a Select Foundation Fellowship (to Dr. Johnston).

Drs. Li and Guo contributed equally to this article.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

We thank Dr. Chiyuan Miao for providing original data for Figure 3 and Tingting Ye for assistance with an earlier version of Figure 3.

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This article was published on October 9, 2020, at NEJM.org.

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DOI: 10.1056/NEJMsr2028985

Int J Cardiol. 2017 Jan 1;226:110-117. doi: 10.1016/j.ijcard.2016.10.053. Epub 2016 Oct 25.

The impact of short-term exposure to air pollutants on the onset of out-of-hospital cardiac arrest: A systematic review and meta-analysis.

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Abstract

BACKGROUND: Acute exposure to outdoor **air** pollution was considered to be associated with the incidence of out-of-hospital cardiac arrest (OHCA). But the relation between specific **air pollutants** and OHCA remains controversial. We conducted a systematic review and meta-analysis to quantitatively assess the acute effects of **air pollutants**, including particulate matter (PM_{10} and $PM_{2.5}$), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO) and ozone (O_3) on OHCA onset.

METHODS: Six databases were searched to identify studies analyzing the association between OHCA and the main **air pollutants**. We summarized the pooled estimates using random-effect models. Heterogeneity within studies was assessed using Cochran's Q and I^2 statistics. Funnel plots, Egger's regression test and Begg's rank correlation method were constructed to evaluate publication bias. Subgroup analyses and sensitivity analyses were also conducted to evaluate the potential sources of heterogeneity.

RESULTS: A total of 15 studies met the inclusion criteria. PM_{10}, $PM_{2.5}$, NO_2 and O_3 were found to be significantly associated with increase in OHCA risk (PM_{10} 1.021, 95%CI: 1.006-1.037; $PM_{2.5}$ 1.041, 95%CI: 1.012-1.071; NO_2 1.015, 95%CI: 1.001-1.030 and O_3 1.016, 95%CI: 1.008-1.024).

The acute exposure to SO_2 and CO was not associated with the incidence of OHCA. Additional analyses verified the findings in the overall analyses except SO_2 and NO_2. Population attributable fractions for PM_{10}, $PM_{2.5}$, and O_3 were 2.1%, 3.9% and 1.6%, respectively.

CONCLUSION: The current evidence confirmed the associations between short-term exposure to $PM_{2.5}$, PM_{10} and O_3 and a high risk of OHCA, with the strongest association being observed for $PM_{2.5}$.

KEYWORDS: **Air** pollutant; Meta-analysis; Out-of-hospital cardiac arrests

PMID: 27806308 DOI: 10.1016/j.ijcard.2016.10.053

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