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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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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.

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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	