FINAL REPORT

Wildland Fire Smoke Health Effects on Wildland Firefighters and the Public

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Abstract

Wildland fire smoke is a complex mixture of air contaminants that have the potential cause adverse health effects. Individuals can be exposed occupationally if they work as wildland firefighters or public exposure from ambient air that is contaminated with smoke from a nearby or distant wildland fire. Previous studies of public exposure to smoke have suggested that wildland fire smoke can cause adverse respiratory health effects and possibly increased mortality and cardiovascular health effects. What the research means for healthy workers is less clear. For example, in wildland firefighters, minor decrements in lung function may be at least partially reversible after periods of recovery (no exposure), but even temporary clinically significant adverse effects from occupational exposures are unacceptable outcomes. This work combines a comprehensive literature review with extensive smoke exposure concentration data for wildland firefighters to estimate health risks specific to wildland fire smoke. First, we conducted a literature review to identify smoke components that present the highest health hazard potential, the mechanisms of their toxicity, and reviewed epidemiological studies to identify the current gaps in knowledge about the health impacts of wildland fire smoke exposure for firefighters and the public. Next, we examined wildland firefighter exposures, explored predictors of smoke exposures to determine factors influencing smoke exposure for wildland firefighters and estimated exposure to air pollutants using carbon monoxide (CO) as an indicator pollutant. Lastly, we estimated disease risk in wildland firefighters for exposure to particulate matter from smoke using firefighter specific breathing rates with existing exposure response relationship information for risk of lung cancer, ischemic heart disease and cardiovascular disease from cigarette smoking, which produces particulate matter with a similar size range. From the literature review, we found that respiratory events measured in time series studies as incidences of disease-caused mortality, hospital admissions, emergency room visits and symptoms in asthma and chronic obstructive pulmonary disease patients are the health effects that are most commonly associated with public exposure to wildland fire smoke. A few recent studies have also determined associations between acute wildland fire smoke exposure and cardiovascular health end-points. From this review, we concluded that there remains a need for research on acute and longer term effects of wildland fire smoke exposure. The health effects of acute exposures beyond susceptible populations and the effects of chronic exposures experienced by the wildland firefighter are largely unknown. Longitudinal studies of wildland firefighters during and/or after their firefighting career could help elucidate some of the unknown health impacts of cumulative exposure to wildland fire smoke, establish occupational exposure limits, and help determine the types of exposure controls that may be applicable to the occupation. We found that among 83 firefighters at prescribed burns, and 417 at project wildfires, the Occupational Safety and Health Administration (OSHA) 8-hour exposure level of 50 ppm for CO was exceeded 3.5 percent of the time at prescribed fires and 5.6 percent of the time at project fires. Adjustments recommended for altitude, work rate and duration of exposure would increase these percentages. We identified that crew type, main work task and duration, and downwind position from the fire were factors that influenced exposure to CO and respirable particulate matter, but only main task and duration were important predictors for respirable crystalline silica exposure. Linear regression results indicated that CO measurement is a reasonably useful real-time gauge of the inhalation hazard from smoke-derived particulate matter. The exposure data also found that respirable crystalline silica was a soil-derived hazard that exceeded shift-average OSHA Permissible Exposure Limits from 6.7% to as much as 28% of the time, depending on incident type. Using epidemiology studies to understand the exposure-response relationship for PM, we found that firefighters were at an increased risk for long-term health effects from smoke exposure. The risk for lung cancer mortality increases nearly linearly with exposures over time and is more strongly influenced by exposure duration than are the risks of death from cardiovascular or ischemic heart disease. On the other hand, the risk of cardiovascular mortality rises steeply for doses in the range we estimated for firefighter exposures but flattens out at higher exposures to PM. The data presented in this paper clearly identify the crews and activities most likely to exceed occupational exposure limits and firefighters may have a an increased health risk from smoke exposures.

Objectives

Aim 1 - Review of the Health Effects of Wildland Fire Smoke on Wildland Firefighters and the Public

Review the existing peer-reviewed literature from five major databases to identify smoke components that present the highest health hazard potential, the mechanisms of toxicity, and review epidemiological studies for health effects to identify the current gap in knowledge on the health impacts of wildland fire smoke exposure for firefighters and the public.

Aim 2 – Smoke Exposure Among Wildland Firefighters

Examine exposures versus occupational exposure limits, explore predictors of smoke exposures to analyze the source of exposure variability and improve understanding of the factors that determine smoke exposure for wildland firefighters and estimate exposure to air pollutants using carbon monoxide as an indicator pollutant

Aim 3 – Wildland Fire Smoke Exposure and Cardiovascular Disease Outcomes

Estimate disease risk in wildland firefighters for exposure to particulate matter from smoke using firefighter specific breathing rates with existing exposure response relationship information for risk of lung cancer, ischemic heart disease and cardiovascular disease.

Aim 1 - Review of the health effects of wildland fire smoke on wildland firefighters and the public

Introduction

Although smoke from burning wildland vegetation (wildland fire smoke) is known to be composed of many potentially harmful components, its impacts on human health are relatively understudied and inadequately understood. Vegetative biomass smoke under different exposure scenarios has been associated with various adverse health effects. However, fewer studies have investigated the adverse health effects of wildland (natural vegetation including forests, grasslands, chaparral etc.) fire smoke compared with those experienced in association with residential combustion of wood or other vegetation based fuels; fewer still have examined the effects of occupational exposure among wildland/forest firefighters.

The current review of vegetative biomass smoke exposure specifically examines adverse health effects of exposure to smoke emissions from forest fires or prescribed burns. Wildland fire smoke exposure is typically experienced on two levels: the community/general public level and occupationally among wildland firefighters. A majority of the investigation into the community level health effects of wildfire smoke exposure has been conducted in association with ambient air particulate matter concentrations, while a few have also studied associations with other criteria air pollutants. However, wildland fire smoke contains many other potentially harmful substances such as mono- and polycyclic aromatic hydrocarbons, aldehydes and metals for which dose-response data are not always available (Naeher et al., 2007).

Additionally, it is important to note that wildland fire smoke is a dynamic mixture, changing temporally and spatially in composition as it is dispersed from the source. Its composition at the source is dependent on combustion conditions, while its variation across space from the source is highly influenced by atmospheric and weather factors. Consequently, the exposures experienced by wildland firefighters deployed to the fire line would be expected to be rather different from those experienced within communities downwind from wildland fires. Due to their proximity to the source, wildland firefighters may be exposed to elevated concentrations of the more harmful constituents of wildland fire smoke such as particulate matter and aldehydes when compared to what is experienced by the public. They are also expected to be more frequently exposed.

Accordingly, we review the literature on and assess the evidence for the health effects of wildland fire smoke exposure on both wildland firefighters and the general public, and discuss the needs for research considering both exposure scenarios.

As part of an effort to characterize health risks of wildland fire smoke exposure to wildland firefighters and the public, we review the literature to identify the components that present the highest hazard potential to both populations. We also review the literature for evidence of the health effects of wildland fire smoke and for possible underlying mechanisms of toxicity. The specific objectives of the current review are to:

1. Discuss the composition of wildland fire smoke. Since a primary objective of this review is the evaluation of health hazards of wildland fire smoke exposure to wildland firefighters and the general public, focus is placed on wildland fire smoke components for which good exposure estimates can be obtained (either from the exposure assessment or emission factor literature), and for which relevant exposure standards are available. This discussion also highlights specific characteristics of wildland fire smoke derived particulate matter in terms of its chemical composition and size distribution.
2. Identify the components presenting the highest hazard ratios to wildland firefighters and the public based primarily on reported occupational exposure or ambient air concentrations.
3. Review the evidence for the adverse health impacts of wildfire smoke on wildland firefighters and the public.
4. Discussion of the possible mechanisms for wildland fire smoke toxicity.
5. Identification of research needs for determining the health effects of occupational and community level wildfire smoke exposure.

This report provides a brief review of this literature review to highlight important information that will be discussed in Aim 2 and 3. The comprehensive literature review that was performed can be found in Appendix I.

Methods

Wildland fire smoke components that are considered harmful based on available occupational or general population regulatory or recommended exposure limits were identified from the literature. Concentrations or emission factor data were then abstracted from the selected papers. Emission factors were used to calculate concentrations if the emission factor for carbon monoxide or carbon dioxide was available in the same study as these are indicators of incomplete and complete combustion respectively. Molar ratios of the components relative to carbon monoxide or carbon dioxide were then obtained from the emissions factor data and multiplied by the maximum mean concentration of fire line exposure to carbon monoxide or carbon dioxide reported in the most comprehensive published wildland firefighters exposure assessment study that is available (Reinhardt & Ottmar, 2004) The maximum estimate or reported average and/or individual concentrations were then used to determine hazard indices based on the most stringent occupational or general population regulatory or recommended exposure limits.

The review of the health effects of wildland fire smoke exposure was conducted using both epidemiological and experimental studies. The evidence analysis protocol of the Academy of Nutrition and Dietetics was adapted for conducting the review (Academy of Nutrition and Dietetics, 2012). Three databases: PubMed, SportsDiscus and Medline were used for a comprehensive literature search for the review of health effects of wildland fire smoke exposure. The terms used for the searches are presented in Table I in Appendix I. Environmental Sciences and

Pollution Management (ProQuest) and ACS Symposium Series, in addition to the first three databases were used for literature searches for emission factor or concentration data for components of wildland fire smoke.

Results and Discussion

Wildland Fire Smoke Composition

Smoke from wildland fires is a complex mixture containing hundreds of non-toxic and toxic air compounds in both particulate and gaseous phases, and its composition often varies spatially and temporally depending on combustion conditions (especially the relative amounts of flaming and smoldering combustion). These in turn are a function of fuel characteristics such as its chemistry, bulk density, arrangement and moisture content (CA Alves et al., 2010b; Burling et al., 2010; Urbanski, 2014). The emissions can have significant impact on the earth's atmosphere by significantly altering the concentrations of some of its constituents, shifting radiative forcing, and negatively impacting air quality on a regional and continental scale (Akagi et al., 2013; Anttila et al., 2008; Ferek, Reid, Hobbs, Blake, & Liousse, 1998; Heil & Goldammer, 2001; Urbanski, 2014; Yokelson et al., 2013).

Wildland fuels have a relatively consistent carbon content with dry matter carbon content ranging between 35% and 55% (Urbanski, 2014). By far, most of the carbon is released as carbon dioxide (CO_2) which together with carbon monoxide (CO) and methane (CH_4) constitutes approximately 95% of carbon released during wildland fires (Urbanski, 2014). According to the National Emissions Inventory (NEI) estimate from the United States Environmental Protection Agency (USEPA), wildland fires (wildfires and prescribed burns) are the largest source of $PM_{2.5}$ emissions in the United States, accounting for 29% of total emissions compared to 9.2% from transportation sources (Aurell and Gullett, 2013). The classes of compounds/components that have been observed in biomass smoke include major inorganic gases, hydrocarbons, oxygenated hydrocarbons, trace metals and particulate matter. Wildland fire smoke could also contain exotic persistent organic compounds such as dioxins and furans. It may also, with possibly less potential impacts, contain radon-derived daughter radionuclides, and absorbed accumulations of abiotic contaminants such as polychlorinated biphenyls (PCBs) and pesticides/herbicides. Based on the maximum reported mean or individual TWA fixed area ground or personal exposure measurements and relevant regulatory or recommended occupational or general population exposure limits for acute and chronic exposures, the components of most concern are respirable (PM_4) or fine ($PM_{2.5}$) particulate matter and carbon monoxide. Other components of concern identified based on the stated criteria are acrolein, nitrogen dioxide, benzene, and formaldehyde, which are fully discussed in Appendix I

Particulate Matter

Particulate matter has been identified as the best single indicator of the health hazards of smoke from biomass combustion sources (Naeher et al., 2007). The size and composition of the particles are two of the characteristics that determine its toxicity (Bølling et al., 2009). Both unimodal and bimodal size distribution have been observed for particles emitted in vegetative biomass smoke (Barregard et al., 2008; Chakrabarty et al., 2006; Iinuma et al., 2007; Keywood, Ayers, Gras, Gillett, & Cohen, 2000; Tesfaigzi et al., 2002). However, results indicate that the particulate matter emission is dominated by smaller particles in the accumulation mode (aerodynamic diameter of 0.1-2 µm) (Barregard et al., 2008; Chakrabarty et al., 2006; Iinuma et al., 2007; Keywood et al., 2000). Additionally, greater increases in concentrations of particles in the accumulation mode have been observed in studies of ambient air during periods of wildland fire compared to periods without such events (Alonso-Blanco, Calvo, Fraile, & Castro, 2012; Cashdollar, Lee, & Singer, 1979; Portin et al., 2012; Sillanpää et al., 2005; Verma et al., 2009).

Sub-micrometer airborne particles, which as noted are relatively abundant in vegetative biomass smoke, are transported by diffusion and penetrate deeper into the lungs compared to larger particles (Araujo & Nel, 2009; Invernizzi et al., 2006; Kristensson, Rissler, Löndahl, Johansson, & Swietlicki, 2013; Schwarze et al., 2006). They are also deposited more efficiently in the pulmonary region compared to the more proximal regions of the lungs (Alföldy, Giechaskiel, Hofmann, & Drossinos, 2009).

The above observations are important as they indicate that wildland fire smoke-derived particulate matter is comparable, in terms of its size, to particles in traffic exhaust or smoke particles from other combustion sources. It possesses more similarities to fumes or diesel particulate matter than to comminution-derived inert dust that is regulated for the workplace (OSHA, 1987). The regulatory standard for inert or nuisance dust is based on its perceived low toxicity due to low solubility (and low quartz content), and its toxicity is thought to result from injury in the terminal airways and proximal alveoli due to accumulation from high levels of exposure (Cherrie, Brosseau, Hay, & Donaldson, 2013). However, wildland fire smoke-derived particles contain water soluble components, and redox reactive metals and polar organic compounds (Célia Alves et al., 2011; Balachandran et al., 2013; Lee et al., 2005; Lee et al., 2008; Leonard et al., 2007; Leonard et al., 2000; Wegesser, Franzi, Mitloehner, Eiguren-Fernandez, & Last, 2010). Such particles may also induce measurable acute pulmonary and systemic responses at lower exposure levels (Naeher et al., 2007).

Although, the current occupational standard for particulate matter would therefore appear to be inadequate for particles in wildland fire smoke, ambient air concentrations in the immediate vicinity of fires (up to 12.5 mg/m^3) (CA Alves et al., 2010a; CA Alves et al., 2010b) and personal wildland firefighter exposures (up to 10.5 mg/m^3) (Reinhardt & Ottmar, 2004) can both exceed the lowest occupational exposure limit (3 mg/m^3) recommended for nuisance dust by the American Conference of Governmental Industrial Hygienists (ACGIH). These levels even exceed the Occupational Safety and Health Administration's (OSHA) nuisance dust Permissible Exposure Limit (PEL) of 5 mg/m^3. These levels are of course well above the current 24-hour National Ambient Air Quality Standard (NAAQS) for $PM_{2.5}$ in ambient air (98th percentile 24-hour average of 35 µg/m^3). Although typically a lot lower than wildland firefighter exposure, ambient air concentrations at least two to three times higher than the NAAQS are not uncommon in urban areas downwind of wildland fire. These levels have been associated with various adverse health outcomes (Ralph J Delfino et al., 2008).

Carbon Monoxide

Carbon monoxide, along with particulate matter, has the most comprehensive exposure data from personal monitoring and area/ground measurements in the literature among the air pollutants emitted during wildland fires. Published study average TWA personal occupational exposures at wildfires or prescribed burns are lower than the lowest OEL occupational exposure limit (OEL) of 25 ppm (ACGIH) indicating that exposures of most wildland firefighting personnel are relatively low (Adetona, Simpson, Onstad, & Naeher, 2013a; Dunn, Shulman, Stock, & Naeher, 2013; Miranda et al., 2012; Reinhardt & Ottmar, 2004; Reisen & Brown, 2009). Nonetheless, the maximum TWA personal occupational exposures in the literature exceeded 50 ppm (Reinhardt & Ottmar, 2004), the CO PEL issued by OSHA. Similarly, the reported maximum instantaneous peak personal exposure of 1085 ppm was about 5.5 times the NIOSH and California OSHA recommended ceiling value of 200 ppm (Reinhardt & Ottmar, 2004). Exposure of the public during wild fire events is usually much lower than the published occupational exposures because of dilution of CO in air during transport from the fire to public receptor locations. v

The toxicity of CO is partly due to its ability to bind hemoglobin more strongly than oxygen (~ 240 times) causing the formation of carboxyhemoglobin (COHb) (J. Raub, 1999). This results in tissue hypoxia since the formation of COHb reduces the oxygen carrying capacity of the blood. COHb levels beginning at 5% saturation in the blood results in decreased work capacity in healthy young adults, while levels below 5% but greater than 2% have been associated with cardiovascular effects in persons with pre-existing cardiovascular diseases (J. Raub, 1999). Higher COHb concentrations could result in headache, dizziness, weakness, disorientation and impair decision making (J. Raub, 1999; J. A. Raub, Mathieu-Nolf, Hampson, & Thom, 2000). The elimination half-life of COHb is 4 to 5 hours without any intervention, and treatment of CO poisoning involves speeding up the elimination rate (Annane et al., 2011; Guzman, 2012; Quinn et al., 2009; Wolf, Lavonas, Sloan, & Jagoda, 2008). Treatment with normobaric 100% oxygen, for example, reduces the half-life by up to 80%; treatment with hyperbaric (2.5 atmospheres) 100% oxygen further reduces the half-life to 20 minutes (Quinn et al., 2009; Wolf et al., 2008). Such treatments reverse hemoglobin binding of carbon monoxide and improve tissue oxygenation (Quinn et al., 2009). Apart from the formation of COHb, other mechanisms at the cellular level are also thought to be involved in CO toxicity (Guzman, 2012; J. A. Raub et al., 2000). This is evidenced by the poor correlation between COHb levels at the time of hospital admission and the symptoms and signs of acute CO poisoning (J. Raub, 1999).

Health Impacts of Wildland Fire Smoke Exposure

Virtually all of the health studies of wildland fire smoke have focused on the more immediate effects of acute exposures on the general public. Furthermore, a very limited number of health studies have been conducted among wildland firefighters, and most of the investigation has focused on acute physiological changes in response to exposures during the work shift at wildfires or prescribed burns. Therefore, little is known about the effects of more chronic cumulative exposures experienced by wildland firefighters. The primary information discussed in this section is from studies investigating the effects of exposures directly related to wildland/vegetation fire events.

Health Effects of Exposure Directly Related to Wildland Fire Smoke in the General Public

The study of the effects of wildland fire smoke exposure is complicated by the sporadic unpredictable nature of wildfires. Consequently, most of the knowledge about the health impacts of exposures directly related to wildland fire smoke on the general public has come from retrospectively conducted ecological time series studies: 25 of the 36 (69%) of the articles that were identified were ecological studies with only population level measures for exposure and outcomes.

Acute cardiovascular and/or respiratory impacts with lagged effects mostly restricted to within six days of exposure were the focus of most (35/36) of the studies that were identified. Furthermore, outcomes in many of the studies were defined as the incidences of mortality, hospital admission, physician or emergency room visits due to events or symptoms resulting from diseases such as chronic obstructive pulmonary disease (COPD), asthma and cardiovascular episodes such as stroke, heart failure and cardiac dysrhythmia. Accordingly, health effects that have been examined have largely been those most relevant to people who are susceptible due to pre-existing diseases. Therefore, very little is known about the effects of wildland fire smoke exposure in individuals who are otherwise healthy. Knowledge is also lacking regarding the delayed effects of exposure over the longer term. The summary of all identified studies involving the general public are presented in Table IV in Appendix I. For this final report, we will highlight cardiovascular effects of wildland fire smoke in the general public. Appendix I contains additional discussion of respiratory effects and other health effects of wildland smoke in the general public.

In all, 13 peer-reviewed papers reporting on the possible cardiovascular effects of wildland fire smoke were identified. The ecological time series design was employed in all the studies except for a cohort study that was conducted in British Columbia, Canada (Henderson et al., 2011). Mostly, null findings were reported for the associations between wildland fire exposure and cardiovascular health end points among the general public. No positive association was reported for hospital admissions, physician or emergency room visits due to all cardiovascular diseases combined in ten studies conducted in North America, Asia or Australia (Crabbe, 2012; Delfino et al., 2008; Duclos et al., 1990; Hanigan et al., 2008; Henderson et al., 2011; Johnston et al., 2007; Moore et al., 2006; Morgan et al., 2010; Mott et al., 2005; Rappold et al., 2011).

However, positive association was reported for the association between cardiovascular mortality in Athens, Greece and the size of forest fires occurring in areas adjacent to the city (Analitis et al., 2011). An apparent dose-response effect was also observed with fires classified as medium being associated with a non-significant 6.0% (-

0.3, 12.6%) rise in cardiovascular mortality, while fires classified as large were associated with a 60% (43.1, 80.3%) increase. Additionally, the effect was observed to be more pronounced in the older population (> 75 years). Conversely, no positive association was observed between mortality among all age groups and visibility which was used as a surrogate measure for particulate matter ambient air pollution in two Malaysian cities, Kuala Lumpur and Kuching, during the 1997 forest fire related haze in Southeast Asia (Sastry, 2002). Nevertheless, cardiovascular mortality was observed to increase among 65-74 year olds (RR: 2.016) in Kuala Lumpur and persons who were 75 years and older (RR: 3.060) in Kuching on days with forest fire related reduced visibility in the same study.

Some of the identified papers report findings from studies investigating the associations between wildland fire smoke exposure and specific cardiovascular health end points among the general public. These end points include hospital admission and/or emergency room visits for hypertension, ischemic heart disease, cardiac dysrhythmia, myocardial infarction, stroke and heart failure. Of these, positive associations have only been reported for hospital admission due to hypertension in relation to exposure to smoke from the burning of sugar cane fields in Brazil (Arbex et al., 2010), emergency room visits due to heart failure in relation to exposure to peat forest fire smoke in North Carolina (Rappold et al., 2011), and hospital admissions due to ischemic heart disease among indigenous people in Darwin, Australia (Johnston et al., 2007). However, these results should be interpreted with caution. No other study of the association between wildland fire smoke exposure and hypertension was identified. Two studies, one in Australia in relation to bushfires and another in the United States with respect to forest fires, report null findings for hospital admissions due to heart failure (Delfino et al., 2008; Morgan et al., 2010). A null finding was reported for non-indigenous people in the Australian study which reported a positive finding for indigenous persons for hospital admissions for ischemic heart disease (Johnston et al., 2007). Additionally, four other studies – one from Malaysia in relation to the 1997 forest fire haze episode in Southeast Asia, one from the United States in relation to forest fires, and two from Australia in relation to bushfires – report null findings for hospital admissions due to ischemic heart disease (Delfino et al., 2008; Hanigan et al., 2008; Morgan et al., 2010; Mott et al., 2005). Null findings were reported for cardiac dysrhythmia in two studies from the United States (Delfino et al., 2008; Rappold et al., 2011), myocardial infarction in one study from the United States (Rappold et al., 2011), and stroke from two studies, one from the United States and the other from Australia (Delfino et al., 2008; Morgan et al., 2010).

Health Effects of Occupational Exposure to Wildland Fires among Wildland Firefighters

Few studies of the health effects of occupational wildland fire smoke exposure have been conducted among wildland firefighters. The comprehensive review of the health effects of wood smoke by Naeher et al. (2007) included six studies of health effects among wildland firefighters. Nine studies investigating the health effects of occupational wildland fire smoke among wildland firefighters have since been published. None of these studies has investigated direct linkages to diseases, and all have focused on various adverse physiological responses in the airways or blood.

Declines in lung function measures across the workshift have been observed in a few studies. Betchley et al (1997) reported declines of 65 ml, 150 ml and 497 ml/sec in forced vital capacity (FVC), forced expiratory volume in 1 sec (FEV_1) and maximum mid-expiratory flow (FEF_{25-75}) across the work shift for wildland firefighters working at wildland fires in the states of Oregon and Washington. Similarly, corresponding declines of 59 ml, 53 ml and 53 L/min were also observed at the end of the first firefighting activity compared to baseline measurements collected before deployment of a group of firefighters in Corsica, France (Jacquin et al., 2011). A smaller non-significant cross-shift decline in FEV_1 of 30 ml was reported by Gaughan et al. (2008). No association was observed between cross-shift declines in lung function measures and work shift exposure to $PM_{3.5}$, carbon monoxide, acrolein or formaldehyde in the studies conducted in Oregon and Washington (Slaughter, Koenig, & Reinhardt, 2004). However, Gaughan et al. (2014a) reported an association between cross-shift declines in FEV_1 and work shift exposure to particulate levoglucosan.

Nonetheless, the results of the cross-shift studies referenced above are limited by the lack of comparisons to control days when the firefighters were not exposed to wildland fires. This is especially important because of the large variability that is associated with lung function measures (Borsboom et al., 1999; Troyanov, Ghezzo, Cartier, & Malo, 1994), and the probable confounding effect of physical exertion that accompanies working at wildfires or prescribed burns. Moreover, Adetona et al. (2011b) did not detect any differences in changes in lung function measures between days when a crew of wildland firefighters in Southeastern United States worked at prescribed burns and days when they did not.

However, there is evidence that continuous occupational wildland fire smoke exposure may have a cumulative effect on lung function. Adetona et al. (2011b) observed decreases of 24 ml in FVC and 24 ml in FEV_1 for each additional day that the firefighters worked at a prescribed burn during the dormant winter burn season in the Southeastern United States (Adetona, Hall, & Naeher, 2011b). Declines in lung function measures have also been observed across periods encompassing one or two burn seasons (Betchley, Koenig, van Belle, Checkoway, & Reinhardt, 1997; Gaughan et al., 2008; Liu, Tager, Balmes, & Harrison, 1992; Miranda et al., 2012). Significant cross-season declines of 90 ml, 150 ml and 440 ml/sec in FVC, FEV_1 and FEF_{25-75} respectively were found in wildland firefighters in the state of California (Liu et al., 1992). Corresponding cross-season declines in these measures in another study of wildland firefighters in the states of Oregon and Washington were 33 ml, 104 ml and 275 ml/sec (Betchley et al., 1997). Gaughan et al. (2008) also observed a cross-season decline of 224 ml in FEV_1 in hot-shot firefighters working at wildfires in the states of Alaska and California. Additionally, Rothman et al. (1991) reported cross-season declines of 1.2% in FEV_1 and 0.3% in FVC that were mostly associated with hours of recent firefighting activities among wildland firefighters in California. However, it is unclear whether declines through the prescribed burn/wildfire season are sustained through non-exposure periods/months. No difference was observed in pre-season lung function measurements of a small number of wildland firefighters ($n = 9$) across two years in one study (Adetona et al., 2011b), while Betchley et al (1997) reported that cross-season declines in lung function measures tended to resolve over non-exposure periods ranging between 5.5 and 13 months among the subjects in their study. However, declines which had been observed across a work shift among wildland firefighters in Corsica, France persisted over a three month non-exposure period (Jacquin et al., 2011). FVC, FEV_1 and FEF_{25-75} remained 280 ml, 340 ml and 45 L/min below their baseline measurements respectively. Wildland firefighters in Sardinia, Italy also had lower measurements for various lung measurements including FVC, FEV_1 and the FEV_1/ FVC ratio compared to policemen on the island after controlling for known confounding factors such as age, height and smoking (Serra, Mocci, & Randaccio, 1996). It should be noted that the authors considered that the two groups were similar with respect to their level of physical fitness and the non-sedentary nature of their jobs.

Acute airway and systemic inflammation among wildland firefighters have also been investigated in a few studies. No significant cross-shift changes in eosinophilic cationic protein and myeloperoxidase in induced sputum were observed among two Type 1 (IHC) “hot-shot” crews fighting wildfires in Alaska and California (Gaughan et al., 2008). However, concentrations of these inflammatory biomarkers were increased in their nasal lavage across the work shift. Furthermore, Swiston et al. (2008) reported cross-shift increases in percentages of granulocytes, mostly neutrophils, in induced sputum among wildland firefighters in British Columbia. On the other hand, exhaled nitric oxide did not increase immediately after the end of a second season of firefighting compared to baseline measurements in a group of firefighters in another study that was conducted in Portugal (Miranda et al., 2012). Although, the investigators were surprised by this result because it indicated reduction in airway inflammation consequent upon exposure to wildland fire, they noted that their observation was similar to results observed in smokers. They noted that cigarette smoke may induce such effect due to the inhibition of nitric oxide synthetase; this in turn could contribute to increased risks of chronic and respiratory diseases in cigarette smokers since endogenous nitric oxide is important for protecting the respiratory tract and counteracting bronchoconstriction, vasoconstriction and platelet aggregation (Miranda et al., 2012).

Acute systemic inflammation consequent upon occupational wildland fire exposure among wildland firefighters has been investigated in two studies (Hejl et al., 2013; Swiston et al., 2008). Significant cross-shift changes in circulating band cells and serum concentrations of pro-inflammatory cytokines, interleukin-6 (IL-6) and IL-8, were observed after exposure to wildland fire smoke in British Columbia (Swiston et al., 2008). Increases in the cytokine concentrations were not observed across a work shift when the firefighters were engaged in strenuous physical activities but had no wildland fire smoke exposure. A similar finding was observed among wildland firefighters in Southeastern United States (Hejl et al., 2013). Post-shift concentrations of IL-8 in dried blood spot samples were 1.7 times higher than the pre-shift levels. Cross-shift differences were not observed for adhesion molecules (VCAM-1 and ICAM-1), IL-1β, serum amyloid A (SAA), and C-reactive protein (CRP). Comparisons to changes on days when there were no wildland fire smoke exposures were not made in this study.

Although wood smoke particles have been shown to generate reactive oxygen species (ROS),(Leonard et al., 2007; Leonard et al., 2000) only two studies of oxidative stress among wildland firefighters were identified (Adetona et al., 2013b; Gaughan et al., 2014b). Cross-shift changes were not observed for oxidative stress biomarkers, urinary 8-hydroxy-2'-deoxyguanosine (8-OHdG) and 8-isoprostane, in all wildland firefighters that were included in one study. However, cross-shift increases were observed for subjects who had worked as wildland firefighters for less than two years, while cross-shift decreases were observed for those with longer careers (Adetona et al., 2013b). The authors hypothesized that the acute oxidative stress response due to wildland fire smoke may be modified by the cumulative exposure of the wildland firefighter. The study was limited due to its very small sample size. Although many repeated measurements were collected, the total number of subjects was 17 and the number of subjects per career length group was five or less. Gaughan et al. (2014b) also observed a positive association between urinary 8-OHdG and aortic augmentation among two hot shot crews in Colorado. Aortic augmentation is a measure of arterial stiffness which is involved in the pathogenesis of cardiovascular disease.

None of the studies of wildland firefighters that were identified investigated the effect of occupational wildland fire smoke exposure over the longer term. Consequently, very little to nothing is known about the health effects of continuing occupational wildland fire smoke exposure across years among career wildland firefighters. However, such information is needed since the exposure of wildland firefighters, unlike that of the public, is more persistent and typically much higher. The summary of all identified studies involving wildland firefighters are presented in Table V in Appendix I.

Mechanisms of Toxicity

Most mechanistic studies of wood smoke toxicity relate to its adverse effects in the airways with one study involving both intratracheal instillation and oral lavage of wood smoke particles reporting that the strongest effects were exerted in the organ closest to the port of entry (Danielsen et al., 2010). However, systemic effects after inhalation exposures are reported in a few *in-vivo* and human studies. The majority of the mechanistic studies investigated the effects of wood smoke particle exposure on oxidative stress, inflammation and cell toxicity. A few of the studies that attempt an elucidation of the toxicity pathways indicate that these effects are largely due to the endogenous generation of ROS. This indicates that toxicity by wood smoke particles may be induced in a way similar to the hierarchical cellular response model that has been proposed for the toxicity of diesel and ambient air particles (Li et al., 2002; Li, Xia, & Nel, 2008; Xiao, Wang, Li, Loo, & Nel, 2003). It should be noted again at this point that particulate matter has been identified as the chief indicator of the adverse effects of pollution from combustion sources (Naeher et al., 2007). A few studies have also reported that wood smoke inhalation may induce adverse effects through the action of its component pollutants on cells in the autonomic nervous system. It seems that these effects could be mediated without or together with particles in wood smoke, and that the generation of ROS is at least partially involved.

Cardiovascular Effects

Various indicators of cardiovascular health in association with wood smoke exposure have been studied in a few human experimental studies. Non-smoking healthy human subjects had higher central arterial stiffness measures (augmentation index, augmentation pressure and pulse wave velocity) and decreased variability in the time domain of the electrocardiogram one hour after exposure to wood smoke with an average PM_1 concentration of ~ 300 $\mu g/m^3$ for three hours compared to filtered air exposure (Unosson et al., 2013). There were no changes immediately or 20 hours after wood smoke exposure in both the time domain and repolarization variables of the electrocardiogram in another human experimental study (two hour exposure to particulate matter concentration of ~ 400 $\mu g/m^3$) (Ghio et al., 2012). Marginally significant minimal changes were observed in the frequency domain measures, while a significant 16.8% increase in maximal heart rate was observed in this second study. Compared to exposure to filtered air, there was no change in central arterial stiffness measures over a 24 hour period following experimental one hour exposure of firefighters to wood smoke with an average PM_1 concentration of 1,115 $\mu g/m^3$ (Hunter et al., 2014). No change was observed in vascular function as measured by venous occlusion plethysmography with intra-arterial infusion of vasodilators 4-6 hours after wood smoke exposure among the firefighters. Similarly, no change in vascular function as measured by peripheral arterial tonometry was observed among non-smoking healthy subjects immediately, six or 20 hours following 3 hour exposures to average $PM_{2.5}$ concentrations of 200 $\mu g/m^3$ and 354 $\mu g/m^3$ (Forchhammer et al., 2012). Timing of measurements and the healthy worker effect in the case of the firefighter study were given as possible reasons for the negative findings and the inconsistent results between the studies (Hunter et al., 2014).

Three possible mechanisms that have been proposed for the cardiovascular effects of particulate matter inhalation exposure could apply to wood smoke. These include the spilling over of local airway inflammation into the lungs, translocation of ultrafine particles into circulation from the airways, and the interaction with the autonomic nervous system through the stimulation of pulmonary vagal afferents by wood smoke constituents (Brook et al., 2002; Robert D Brook et al., 2010; Ghelfi, Rhoden, Wellenius, Lawrence, & Gonzalez-Flecha, 2008; Kido et al., 2011; Mills et al., 2009). The first two pathways could also be involved in systemic oxidative stress and inflammation resulting from inhalation exposure to wood smoke.

Conclusions

The summary of evidence for the hazard associated with wildland fire smoke is presented in Table VI. The evidence that acute wildland fire smoke exposure adversely impacts respiratory health among the general public is strong. Although most of the evidence is from ecological studies without individual level measurements of exposure and outcomes, positive findings have been reported in cohort studies for COPD symptoms and various indicators of worsening of health in persons with asthma (Henderson, Brauer, MacNab, & Kennedy, 2011; Johnston et al., 2006; Sutherland et al., 2005). Results from studies from different regions of the world (North and South America, Southeast Asia and Australia) are mostly consistent for positive findings for acute responses in persons with pre-existing diseases or for the development of respiratory infections resulting in hospital admissions, emergency room or physician visits. Dose-response relationships between exposure during wildland fire events to particulate matter, a major health hazard in wood smoke, and respiratory end-points were also determined in many of the studies. Furthermore, persons who are more susceptible to adverse effects of wildland fire smoke due to pre-existing conditions would more likely take preventive measures to reduce their exposures during wildfire events. Such behavior would result in exposure misclassification which would bias estimates for effects sizes towards the null. This might have contributed to the null findings in some of the ecological studies.

The available research on wildland firefighter occupational exposure is currently very limited, and there is not enough information to make conclusions with regards to cardiovascular and chronic respiratory effects. Only acute physiological responses have been investigated without any determination of the clinical significance of findings. Therefore, a conclusion could only be made with respect to acute respiratory effects. The evidence for wildland fire being an acute respiratory hazard is weak. The pattern of wildland firefighter occupational exposure is very different from those of the populations from which evidence of chronic effects are available. Their exposure is more frequent than that of the general public to wildland fire smoke but more intermittent than the exposure experienced by individuals in the case of household air pollution. Additionally, the healthy worker effect makes the extrapolation of results difficult. Consequently, there is need to conduct studies of clinically significant health end-points among this population. Investigating such effects in association with the intermittent seasonal nature of wildland firefighters may help elucidate possible associations between exposure and disease initiation and/or progression. Experimental models with exposure patterns, fuel mix, and combustion conditions similar to the populations of interest in this review could also help inform on the health effects of wildland fire smoke exposure.

Aim 2 - Smoke Exposure Among Wildland Firefighters

Introduction

The USDA Forest Service (USFS), National Technology & Development Program (NTDP) collected breathing zone measurements of occupational exposure to smoke and dust at wildland fire activities across the U.S. between 2009 and 2012. This portion of the report presents the methods and results of the initial comprehensive analysis of these data. From 2009-2011, a variety of wildland fire crew types were included in the exposure monitoring program. In 2012, only line management staff were selected for exposure monitoring at a variety of wildfires.

Wildland fires can be summarized by the objective of the fire management activity. Fire Type is a convenient subdivision of the exposure data, because it translates readily to operationally meaningful descriptions of wildland fire management activities. The four basic fire types at which data were obtained by NTDP were:

- Prescribed Burns—intentionally-ignited burns of designated areas to achieve land management objectives, these are commonly bounded by pre-established firelines, roads and natural features, may have extensive hose lays to provide water along the firelines which are usually manned to prevent escape of the fire, and are ignited by ground-based personnel or aerial incendiary devices. Pile burning is a specific type of prescribed burning that involves ignition of machine-or hand-piled forest debris, left over one or more winters to cure, and typically covered during.

- Prescribed Natural Fires—these are naturally-ignited wildland fires that are allowed to continue to burn because they are achieving land management objectives. In that sense they are prescribed fires, and like prescribed burns the fires may require fire suppression intervention to stop their progress when they approach the boundary of the intended burn area. However, they do not benefit from the completely planned nature of prescribed burn ignition and firing rate management. Because the pattern and rate of ignition is not managed to a set plan like a prescribed burn, the ad hoc nature of the fire progress forces firefighters to engage in suppression efforts that are similar to wildfires.

- Initial Attack—these types of fires are characterized by the suppression efforts begun as soon as a wildfire is reported and accessible. The fire may be put out quickly with the initially-responding resources. The shift may include substantial time spent doing unexposed tasks while waiting for a fire to occur.

- Project Wildfires—When initial attack efforts fail, the fire typically expands in size with every day, requiring more personnel, resources and time to contain. Project fires can last over a month, and have a management and logistical support team operating out of a fire camp, often supplemented by rudimentary spike camps if terrain and logistics make end of shift return to a central fire camp infeasible.

The objective of this aim was to examine exposures versus occupational exposure limits, explore predictors of smoke exposures to analyze the source of exposure variability and improve understanding of the factors that determine smoke exposure for wildland firefighters and estimate exposure to air pollutants using carbon monoxide as an indicator pollutant.

Methods

Data Collection

The air sampling is described briefly in this section; for more detail reference Appendix II. The 2009 data consisted of personal measurements of exposure to carbon monoxide (CO), measured using CO dosimeters (MSA Altair Pro) consistent with Method 6604, published by the National Institute for Occupational Safety and Health (NIOSH). Beginning in 2010, data were also collected for exposure to respirable particulate matter (PM_4) with a median diameter of 4 micrometers (µm), generally consistent with NIOSH Method 0600, with analysis of crystalline silica content using NIOSH Method 7500. Pre-weighed 37-millimeter diameter PVC filters with 1 µm pore size in 3-piece cassettes were obtained from a commercial laboratory accredited by AIHA in the Industrial Hygiene Laboratory Accreditation Program (RJ Lee Group, Monroeville, PA). SKC Airchek pumps were used at a target flow rate of just over 1 liter per minute. The cyclone selected for the PM_4 sampling was the BGI SCC1.062 (Triplex), constructed of aluminum to minimize wall losses from electrostatic effects. The Triplex sampler obtains a PM_4 curve approximation at only 1.05 liters per minute, half that of alternative samplers, and according to manufacturer literature achieves a reasonably good match to the consensus sampling efficiency curve for respirable particulate established by the International Standards Organization, the European Standards Committee, and the American Conference of Governmental Industrial Hygienists (ACGIH). Field protocols were developed to provide a rich data set that could be used to analyze exposure against a variety of variables.

At most wildfire events, an extra CO dosimeter and PM_4 sampling train were left in a central location of the incident command post, or at a spike camp that the crew was assigned to. They were primarily intended to be started upon initial visit to the camp and ended every 24 hours. These samplers were left unsupervised but otherwise handled in the same way as the personal exposure samples. Because the study had only personal sampling equipment and methods designed for occupational exposures, which are not as sensitive as the methods required for assessing public health hazards, it is likely that the statistical metrics for these “camp” results were biased by the detection limit censoring, and so indicate higher exposures than actually occurred.

The CO dosimeters were activated at the start of the workshift and removed from the crew prior to their departure back to fire camp. General there was little or no smoke at camp. The dosimeters were programmed to record the average and peak CO concentration every minute. The average value was used for analysis. The filter flow rates through the PM_4 samplers were calibrated on site with a primary standard frictionless piston (BIOS DC-Lite) before sampling, and checked again after sampling, using the calibration adapter provided by the manufacturer. After sampling, all filters were capped and transported under chain of custody to the laboratory, accompanied by field blanks prepared each day. CO dosimeters were calibrated at the end of each shift, and the data downloaded to field computers before the instruments were reset.

Because CO data was collected on a minute by minute basis during operational shifts it is possible to calculate several different occupational exposure metrics to evaluate the exposure to acute toxins as well as those that accumulate over longer periods. Spreadsheets that had been developed for the fireline rate construction project were modified and expanded to automatically calculate the following metrics:

- The highest 1-minute CO exposure value provides the peak exposure level for each firefighter on every shift. This is a good value to compare against the NIOSH Immediately Dangerous to Life or Health (IDLH) standard of 1200 ppm.
- The highest 5-minute CO exposure value was obtained by rolling average through each firefighter’s shift, incrementing each minute of the day. This metric is a good value to compare against the typical state-jurisdiction (e.g., California and Washington) short term exposure limit (STEL) of 200 ppm.

- Eight-hour CO exposures were also calculated for each firefighter. A rolling eight-hour exposure was calculated (incrementing forward in time with each minute of monitoring data), and the highest value was automatically selected for comparison to 8-hour duration OELs. When the firefighter was on the line for less than eight hours the eight-hour exposure was calculated by adding the appropriate amount of time at zero exposure to the time at the measured fireline exposure, so the calculation can be made on an eight-hour exposure. In these cases, field notes were consulted to confirm that no known exposure took place during the additional time in the eight hours, and an 8-hour time-weighted average (TWA) was calculated. A convenient metric for this is the TLV of 25 ppm that should be health-conservative for healthy workers, even when firefighting occurs at high elevation. A suggested effort would be to confirm this assumption through application of one of the pharmacokinetic models to predict COHb from exposure conditions.

- Fireline exposures were also calculated to represent the TWA exposure during all time on the fireline. Because the dosimeters were measuring CO the entire time these firefighters were on the fireline, it is a direct measurement for these data. The PM_4 and respirable crystalline silica samples began and ended with the fireline time, and therefore these only represent exposure during activities on the fireline. If the time on the fireline is more than eight hours, a variety of methods are available to adjust an 8-hour OEL downward to account for the longer time.

- Shift exposures were also determined for each firefighter by using the total exposure during fireline operations and adding the total shift time to the calculations. The shift exposure is a TWA that includes exposure (or zero exposure) off the fireline. If fire camp data had shown that the firefighters were in an inversion and were exposed even though they were not on the fireline, this would be included in the shift average. However, the fire camp measurements indicated uniformly there was no appreciable CO exposure off the fireline at these events, so the TWA uses zero ppm CO for this unexposed time in the TWA formula.

The field data collection attempted to collect detailed data for a wide variety of potentially explanatory variables, in addition to fire type.

- Crew type (Type I, Type II, Type II(IA), Fuels, Engine, Dozer)
  - Fire management personnel were sampled in 2012.
- Work Activity
  - Fireline Construction –direct and indirect, lighting burnouts, dozer operation, dozer boss
  - Fireline Defense – holding and gridding
  - Post Flaming Phase Tasks – mop-up
  - Prescribed burning tasks – lighting and holding
- Environmental Conditions
  - Fuel Model
  - Slope and slope position
  - Wind speed and wind position
  - Temperature
  - Fire behavior
  - Flame height
  - Canopy cover
  - Inversion
  - Region, State and Land Owner

Factors that might control exposure were noted by observers at the start of time on the fireline, and roughly hourly through the remainder of the day—each interval comprising a period of time in the day defined by a start and end time.

Data Analysis

Because of the variability in smoke exposures, modeling was undertaken to analyze the source of variability and improve understanding of the factors that determine smoke exposure. For the following exposure response variables, the analyses were performed on the log of the exposure because each distribution approximated a lognormal distribution:

- Short-term (5-minute STEL and 1-minute peak) CO exposure,
- Fireline-average CO exposure,
- Fireline-average PM_4 exposure, and
- Fireline-average respirable crystalline silica (quartz) exposure.

General modeling approaches and tests are summarized here. Unique aspects of modeling for specific response variables are discussed in Appendix II. The exposures of fire managers were summarized separately from firefighters, and other than indicating an apparently lower average exposure, a model for their exposures has not yet been developed. For the firefighters, the data structure of the observational data set was considered and hierarchical “multilevel” mixed-effects models (MLMs) were developed and fit to the observed exposures. In the resulting hierarchical structure of the MLM, the individual “firefighter” was omitted as a clustering factor because there were relatively few replicates by firefighter (specifically, the firefighters were unique for 70% of the 621 shifts. Of the remainder, 72 firefighters were monitored on two days, and 14 were monitored on 3 or more incidents.

Because crews are deployed as teams and were almost always assigned to tasks as a group, with close areal proximity within the group being the norm rather than the exception, the crew was a natural grouping factor. And because a given day presents a unique set of environmental conditions in terms of incident type, weather, general incident behavior and so forth, the day was another factor influencing the exposure for the crew. On each day of exposure monitoring, 2-3 firefighters were typically monitored from within each of two crews. The crews containing the instrumented firefighters were often assigned to different areas of the fire, resulting in usually 4-6 firefighters per day from among two crews. At four prescribed burns, 6-9 firefighters were monitored from just one crew. The grouping factor selected was for any given day a combination of the day and crew variables (factor “daycrew”). As such, our conception is that it captures the random effects of the environmental and site conditions of the day, as well as the unique assignment and characteristics of the crew, if more than one crew was observed that day. This random “daycrew” factor was a random clustering factor which averaged zero in the model, but accounted for each fire being a unique combination of environmental and individual crew characteristics, and of course is simply a sample from a larger population of crews and daily conditions and sites that will continually increase.

Alternative grouping factors were explored (Crew within Day within Fire Name, for example, since there were many instances of multiple days at a given named project fire), but in these data, daycrew effectively captured cluster variation as it had the largest intraclass correlation (ICC) of the alternatives explored. For these reasons, two-level MLMs were developed for the analyses, where fixed effects of explanatory variables were tested after grouping by the daycrew factor. A null model using the daycrew factor was developed, and the significance of the MLMs were compared using analysis of variance (ANOVA). We developed only a random-intercept model, which utilized the same average slope for each fixed effect across all instances of “daycrew”, because different slopes for each level of the daycrew factor would not be useful for predicting future exposures. All data analysis was performed in the R System for Statistical Computing. The R packages NLME and LME4 were used for most of the data analyses. The R package lmerTest was used for model simplification. Parameter estimates for a given model used restricted maximum likelihood (REML) estimates to be reduce potential bias. The significance of fixed effect parameters was tested using degrees of freedom approximations via both Satterthwaite and Kenward-Roger in LME4 and lmerTest.

Overall model comparisons via ANOVA were performed after refitting the models using maximum likelihood estimates of fixed effects, per current guidance. Continuous variables (windspeed, slope, and percentage of fireline time represented by a given activity) were tested using grand mean centering to reduce collinearity effects and aid interpretation (Finch, Bolin and Kelley, pg. 34). Graphics were mainly produced in the R Package Lattice.

Interpollutant correlation modeling

Measurement pairs by firefighter consisting of the PM_4 and respirable crystalline silica data (all found to be only as quartz) were obtained among a subset of 119 firefighters who participated in the CO exposure monitoring. These PM_4 and respirable quartz data covered essentially the entire time the firefighters were on the fireline. The time-matched CO data were paired with the PM_4 and respirable quartz data, along with explanatory factors aggregated over sub-intervals that together summed to the entire fireline time. Work activity was the only explanatory factor with predictive power for PM_4 exposure that was available for each record. Thus, we selected for each firefighter the single activity record representing the most time within each firefighter's day, resulting in 119 records with PM_4 and respirable quartz data, and the associated major activity within them, which represented from 18.5 to 100% of the fireline time. We developed a linear regression between CO and PM_4, after adjusting the PM_4 data to subtract the detectable respirable quartz (considered an indicator of a soil component) and focus on smoke-derived PM_4.

Results and Discussion

In all, monitoring was completed for 83 person-days at prescribed burns, 83 at prescribed natural fires, 50 at initial attack events (of which 60 total events occurred) and 417 person-days were monitored at multi-day project wildfires. In 2012, data were only collected from wildland fire management personnel who had supervisory duties at wildland fire operations. Thirty-one shifts of fire operations supervisors were monitored in that year.

When compared to traditional workplaces, wildland firefighters typically work much longer hours. National Wildfire Coordinating Group (NWCG) and federal agency policy limits firefighters to 14 days on assignment with a mandatory 2-day break. Fireline time is the duration at a fire in the shift. For initial attack days, the fireline time was the sum of hours at each fire when there were multiple events in a day. During their work shift time away from the fireline (in transit or staging) firefighters were usually unexposed to smoke, though this is not always true, especially under inversion conditions in complex terrain. In 2012, only fire management personnel were monitored, so project fire management personnel are a separate category from firefighting crewmembers at project fires.

Table 1: Summary Metrics for Occupational Exposures among U.S. Wildland Firefighters (2009–2012)

Distribution Metrics	CO 1-Min Avg. (ppm)	CO 5-Min Avg. (ppm)	CO 8-hr Avg. (ppm)	CO 8-hr Avg. (ppm)	CO Fireline Avg. (ppm)	CO Shift Avg. (ppm)	PM4 Shift Avg. (mg/m^3)	Quartz Shift Avg. (mg/m^3)	Quartz Shift PEL (%)
OEL Criterion	**1200**	**200**	**50**	**35**	**25**	**16**	**0.7**[c]	**0.057**	**100**
Initial Attack (n)	60[a]	60[a]	50	50	50	50	18	18	18
UTL (95%/95% UCL)	337	208	29	29	41	17	2.2	0.567	176[d]
95th percentile	153	132	15	15	23	9.5	0.69[b]	0.153[b]	280[b]
95% UCL of mean	89	34	3.1	3.1	4.3	2.1	0.24[b]	0.042[b]	72[b]
Arithmetic Mean	62	28	2.4	2.4	3.5	1.6	0.17[b]	0.027[b]	44[b]
Geometric Mean	29	14	0.9	0.9	1.6	0.65	0.07	0.008	11
GSD (unitless)	3.5	3.9	5.6	5.6	5.1	5.1	4.1	6.0	7.2
Nondetects (%)	1.7	1.7	2.0	2.0	2.0	2.0	61	44	44
Exposures > OEL (%)	0.14	2.6	1.0	1.7	4.4	2.5	0.0[b]	28[b]	28[b]
95% UCL of Exceedances (%)	0.65	5.7	3.1	4.5	9.2	6.0	15[b]	50[b]	50[b]
Project Fire Crews (n)	417	417	417	417	417	417	80	80	80
UTL (95%,95% UCL)	610	341	50	50	45	32	2.3	0.303	517
95th percentile	518	287	40	40	36	26	1.7	0.132[b]	211[b]
95% UCL of mean	164	90	13	13	12	8.4	0.67	0.034[b]	56[b]
Arithmetic Mean	142	77	10	10	9.4	6.7	0.53	0.026[b]	43[b]
Geometric Mean	60	29	2.2	2.2	1.8	1.4	0.32	0.007	12
GSD (unitless)	3.7	4.0	5.8	5.8	6.1	5.9	2.7	6.9	7
Nondetects (%)	1.7	1.7	1.7	1.7	1.9	1.7	10	38	38
Exposures > OEL (%)	1.1	8.3	3.8	5.9	7.4	8.4	22	10[b]	10[b]
95% UCL of Exceedances (%)	1.7	10	5.1	7.4	9.2	10	29	17[b]	17[b]

Distribution Metrics	CO 1-Min Avg. (ppm)	CO 5-Min Avg. (ppm)	CO 8-hr Avg. (ppm)	CO 8-hr Avg. (ppm)	CO Fireline Avg. (ppm)	CO Shift Avg. (ppm)	PM4 Shift Avg. (mg/m^3)	Quartz Shift Avg. (mg/m^3)	Quartz Shift PEL (%)
OEL Criterion	1200	200	50	35	25	16	0.7[c]	0.057	100
Project Fire Managers (n)	31	31	31	31	31	31	31	31	31
UTL (95%/95% UCL)	830	496	42	42	53	25	1.1	0.044	100
95th percentile	164 [b]	111 [b]	7.1 [b]	7.1 [b]	7.9 [b]	4.2 [b]	0.35 [b]	0.020 [b]	40 [b]
95% UCL of mean	66 [b]	40 [b]	2.6 [b]	2.6 [b]	3.6 [b]	1.5 [b]	0.22 [b]	0.011 [b]	21 [b]
Arithmetic Mean	48 [b]	28 [b]	1.7 [b]	1.7 [b]	2.2 [b]	1.0 [b]	0.17 [b]	0.009 [b]	17 [b]
Geometric Mean	18	8.6	0.31	0.31	0.30	0.19	0.11	0.007	12
GSD (unitless)	6.2	6.8	10	10	12	10	3.0	2.4	2.7
Nondetects (%)	6.5	6.5	6.5	6.5	9.7	6.5	33	27	27
Exposures > OEL (%)	0.00 [b]	0.00 [b]	0.00 [b]	0.00 [b]	0.00 [b]	0.00 [b]	3.3 [b]	0.00 [b]	0.00 [b]
95% UCL of Exceedances (%)	9.2 [b]	9.2 [b]	9.2 [b]	9.2 [b]	9.2 [b]	9.2 [b]	15 [b]	9.5 [b]	9.5 [b]
Prescribed Natural Fires (n)	83	83	83	83	83	83	16	16	16
UTL (95%/95% UCL)	801	453	91	91	64	53	2.4	0.049	94
95th percentile	523 [b]	285 [b]	49 [b]	49 [b]	35 [b]	29 [b]	0.88 [b]	0.025 [b]	45 [b]
95% UCL of mean	102 [b]	51 [b]	8.0 [b]	8.0 [b]	6.5 [b]	4.8 [b]	0.31 [b]	0.011 [b]	18 [b]
Arithmetic Mean	87 [b]	43 [b]	6.2 [b]	6.2 [b]	5.0 [b]	3.8 [b]	0.21 [b]	0.008 [b]	14 [b]
Geometric Mean	44	20	1.4	1.4	1.1	0.9	0.11	0.006	9.3
GSD (unitless)	4.5	5.1	8.9	8.9	8.3	8.5	3.6	2.4	2.6
Nondetects (%)	3.6	3.6	3.6	3.6	4.8+	3.6	44	38	38
Exposures > OEL (%)	0.0 [b]	0.0 [b]	0.0 [b]	1.2 [b]	4.8 [b]	7.2 [b]	6.3 [b]	0.0 [b]	0.0 [b]
95% UCL of Exceedances (%)	3.6 [b]	3.5 [b]	3.5 [b]	5.6 [b]	11 [b]	14 [b]	26 [b]	17 [b]	17 [b]
Prescribed Burns (n)	83	83	83	83	83	83	15	15	15
UTL (95%/95% UCL)	476	314	60	60	55	36	3.8 [b]	0.180	308
95th percentile	360	206	45	45	49	29	1.7 [b]	0.047 [b]	69 [b]
95% UCL of mean	150	92	15	15	14	9.3	0.74 [b]	0.025 [b]	37 [b]
Arithmetic Mean	123	72	10	10	10.4	6.5	0.49 [b]	0.012 [b]	17 [b]
Geometric Mean	80	42	3.2	3.2	4.4	2.6	0.28	0.004	3.9
GSD (unitless)	2.5	2.9	4.6	4.6	3.7	3.9	3.0	4.8	5.7
Nondetects (%)	0	0	0	0	0	0	20	53	53
Exposures > OEL (%)	0.17	6.7	3.5	5.8	9.2	9.0	20 [b]	6.7 [b]	6.7 [b]
95% UCL of Exceedances (%)	0.62	11	6.5	9.7	14	14	44 [b]	28 [b]	28 [b]

Fireline-average CO exposure

Among the activities that made up most of the time on the fireline, those performing Handline/sawyer (direct) tasks had significantly higher CO exposures than those doing mainly lighting tasks (lighting and lighting boss), pump operations and mop up. Exposures among those mainly holding fireline were significantly higher than those doing the lighting tasks, and those doing mop up. As might be expected, when ancillary tasks predominated (hiking, standby/staging, briefing), exposures were low. The fireline-average CO data indicate that management interventions will be most effective if focused on Type II and Type I (IHC) crews, especially when they are performing direct handline/sawyer assignments and holding fireline, and unavoidably downwind of the fire.

A substantial amount of the variation in fireline-average CO exposures among firefighters was due to which crew and day they happened to be observed on, a "random" factor that was not useful as a future predictor of exposure. The best null model (clustering by the factor daycrew) had an intraclass correlation coefficient (ICC) of 0.67. For the 621 observations of firefighters' fireline-average CO exposure, the final multilevel mixed model that resulted was a two-level model, with each firefighter being nested within a given crew for each day (factor "daycrew", representing clustering within 208 unique crew-days. The daycrew factor accounted for about 42% of the variation in fireline-average CO, while the fixed effects explained 34% (via partial r^2 calculations—Finch et al, 2014. Pp. 47-48). The model can be summarized as:

logFireline.CO=CrewType+Activity2.1.1+Ctr.PctFireline1+WindPosition1+Activity1:Ctr.PctFireline1

Where:

logFireline.CO = the log of the fireline-average carbon monoxide concentration (adjusted for the method detection limit),

CrewType = The firefighter crew category,

Activity2.1.1 = The most-performed activity represented in the time on the fireline (for a given state of all the other final factors in the model),

Ctr.PctFireline1 = The grand mean centered percentage of fireline time represented by the most-performed activity,

WindPosition1 = The wind field position of the firefighter relative to the fire; and

Activity2.1.1:Ctr.PctFireline1= The interaction between the work activity and the percentage of fireline time that it represents.

Similar exposure groups can be established from these data. For example, estimated fireline-average exposures could be specifically examined among Type I (IHC), Type II(Fuels) or Type II(IA) crews performing fireline holding or Handline/Sawyer(Direct) tasks in downwind situations. A long-term CO surveillance project might be appropriately focused on tracking and assessing the effectiveness of mitigation strategies for fireline-average CO exposure among these crews doing these tasks in these conditions, as the model indicates that based on these data, they will have the highest fireline-average CO exposures. By grouping future data by these crew, task and wind position categories, the inherent variability of the results may be reduced, thereby improving the ability to detect a real reduction of exposure from a given mitigation strategy.

Fireline-average PM_4 exposure

There were 128 observations of PM_4 exposure among firefighters in 2010-2011 (data were not collected for PM_4 in 2009). For PM_4 exposure, project fire crews and prescribed burning are both likely to exceed a hypothetical PM_4 criterion of 1.0 mg/m^3 due to smoke alone, especially for certain tasks such as holding fireline and during mop-up. Shift-average exposures to PM_4 are likely to exceed a 14-hour adjusted OEL of 0.7 mg/m^3 among project wildfire crews, and during prescribed burning, though few prescribed burn days last 14 hours.

Main work activity, position up- or downwind of the fire, and crew type were the only significant factors. In terms of main activity during the shift, PM_4 exposures among those doing mainly mop up were significantly higher than those doing mainly Handline/sawyer(Indirect) line construction, and those performing ancillary tasks. PM_4 exposures for those doing mainly mop-up and holding fireline were also higher than Handline/sawyer(direct or indirect), and dozer operations, but not significantly so. The average PM_4 exposures for those doing mainly mop up was higher, but not significantly higher than those holding firelines. PM_4 exposure management implications from these findings indicate that the most effective opportunities to reduce PM_4 exposures would be among Type II and Type I crews downwind of the fire, doing mop up and holding firelines. Dozer crews also present opportunities to reduce PM_4 exposures.

Because of lack of replication within a crew on a given day, the "daycrew" strategy used for CO could not be applied to the PM_4 data. The best null model hierarchy was obtained by using *Crew.Name* within *Fire.Name*, which produced an ICC of 0.60. This formed the null model for comparing fixed effects. The final multilevel mixed model that resulted was a three-level model, with each firefighter being nested within a given crew for a given fire. The fixed effects for the model can be summarized as:

logFireline.PM4=CrewType+Activity2.1.1+WindPosition1

Where:

logFireline.PM4 = the log of the fireline-average PM4 concentration

CrewType = The firefighter crew category,

Activity2.1.1 = The most-performed activity represented in the time on the fireline

WindPosition1 = The wind field position of the firefighter relative to the fire.

Fireline-average respirable crystalline silica (quartz) exposure

There were 128 observations of respirable crystalline silica exposure among firefighters in 2010-2011 (the PM_4 samples were subsequently analyzed for crystalline silica—all was found to be quartz). As for the PM_4 data, for respirable quartz the lack of replication within a crew meant that "daycrew" could not be used as a clustering factor. For respirable quartz, the best hierarchy was obtained by using Crew.Name within Fire.Name, which produced an ICC of 0.70. This formed the null model for comparing the addition of fixed effects.

For respirable quartz, the main work activity and the proportion of time it represented were the only significant factors. Those doing mainly dozer operations had higher respirable quartz exposures than all other tasks, but significantly higher than only holding, lighting and ancillary operations. Mop-up respirable quartz exposures were significantly higher than those doing mainly holding or lighting. Differences in these patterns for respirable quartz versus PM_4 make sense when considering the source—respirable quartz arising only from soil disturbance, and PM_4 representing mainly smoke. Management implications for respirable quartz are that dozer operations and mop up tasks present the best opportunities to control dust exposures.

The fireline-average respirable quartz model included all 128 observations of firefighters' fireline-average respirable quartz exposure, clustered among 50 fire names, and 104 crew names within them (some crews were sampled on more than one day within a given project wildfire or PNF). Simplification dropped several factors that had been significant for CO and PM4. The final model only kept the following categorical and continuous fixed-effect factors:

$$logFireline.Quartz = Activity2.1.1 + Ctr.PctFireline1$$

Where:
logFireline.Quartz = the log of the fireline-average respirable crystalline silica concentration
Activity2.1.1 = The most-performed activity represented in the time on the fireline (for a given state of all the other final factors in the model)
Ctr.PctFireline1 = The grand mean centered percentage of fireline time represented by the most-performed activity.

For establishing similar exposure groups for exposure to respirable quartz, the model results indicate that it makes sense to focus on personnel performing mop-up tasks, and the crews performing handline construction (as they were generally higher in exposure, although they weren't significantly higher than other tasks). Clearly dozer crews are likely to be a similar exposure group while performing dozer operations. A long-term respirable quartz surveillance project should track and assess the effectiveness of mitigation strategies for fireline-average quartz exposure among these crews doing these tasks. Grouping future data into SEGs by these task categories should improve the ability to detect a real reduction of exposure from a given mitigation strategy.

In summary, for respirable quartz, the majority activity and the proportion of time it represented were the only significant factors. Those doing mainly dozer operations had higher respirable quartz exposures than all other tasks, but significantly higher than only holding, lighting and ancillary operations. Mop-up respirable quartz exposures were significantly higher than those doing mainly holding or lighting.

Exposure at Fire Camps

Data collected at wildfires throughout the US between 2010 and 2012 shows that highly-elevated smoke incidents in fire camps are rare. Ambient air quality was measured over 24-hour periods during 80 days at 21 incidents. Results were validated for 79 days of PM_4 measurement and 80 days of CO measurement.

Of the 80 days with CO data, about 69 percent of the rolling 8-hour average CO exposures never rose above the detection limits of the instrumentation (about 1 ppm), and the 24-hour CO levels reached 5 ppm during only one day in one ICP, and exceeded 2 ppm at only one spike camp. Based on these data, the 95 percent upper confidence level estimate of the frequency of exceeding the 9 ppm CO NAAQS is less than 3 percent. In other words, for 100 wildfires, only three would exceed that level, and less than 0.7 percent (less than 1 in 1000) would exceed the PEL.

For PM_4, 79 percent of the 79 daily 24-hour average PM_4 results were below measurement detection limits (typically the detection limit was in the range of 14-40 $\mu g/m^3$, depending on sample duration). Using methods appropriate for censored lognormal distributions, the Kaplan-Meier estimate of the arithmetic mean of these daily average concentrations was 35 $\mu g/m^3$ in ICPs and 50 $\mu g/m^3$ in spike camps. This is a statistically-derived estimate that may be biased high because of the relatively high method detection limits and effect on censoring levels. We note that this average concentration is about equal to the 98th percentile 24-hour average $PM_{2.5}$ NAAQS, and we have the understanding that these were fire camps that did not report unusually smoky or concerning conditions.

Correlations among atmospheric hazards

The fireline-average data provided an opportunity to test whether results were consistent with previous work showing a relationship between CO and PM_4 in smoke. The relationship between CO and quartz-corrected respirable particulate matter (adjusted PM_4) can be summarized as a simple linear regression:

$$Adjusted.PM4 = 0.31\ (\pm 0.06) + 0.085\ (\pm 0.0073)\ \times COPPM$$

Where:

Adjusted.PM_4 = the soil-corrected PM_4 exposure of the firefighter (in milligrams per cubic meter, adjusted by subtracting the respirable quartz in the respirable particulate matter samples); and
COPPM = the TWA CO exposure of the firefighter (in parts per million) over the period corresponding to the PM_4 sample.

In summary, we recognized that PM_4 at the breathing zone of the wildland firefighter would be due to two main sources: smoke and soil-derived dust. Other sources of inhalation hazards may come into play at times, such as when firefighters are exposed to exhaust from diesel and gasoline engine operation and smoke from drip torches and fusees. We were able to adjust the PM_4 concentration downward to correct for soil quartz by subtracting the mass of quartz measured in the sample. This likely represents the minimum effect of soil dust, because soils may not always include quartz. From this analysis, there appears to be a reasonably consistent correlation between exposure to PM_4 from smoke and exposure to CO.

Especially for tasks such as holding line, where almost all the CO and PM_4 are due to the fire, the CO to adjusted PM_4 exposure relationship is a valuable estimator of how much of the PM_4 is due to fire emissions. Should toxicology and risk assessment identify PM_4 from smoke as the critical pollutant to manage, this relationship is a ready means to estimate the PM_4 exposure. When tasks generate soil dust, the total PM_4 exposure will reflect the additional respirable portion of the dust created by these tasks (such as mop up, handline construction, bulldozer operations, hiking in dusty conditions, and driving on dusty roads). With the recent reduction of the OSHA respirable crystalline silica standard to 0.05 $\mu g/m^3$ (scheduled to take effect in September, 2017), attention to that soil dust hazard is warranted where soils contain quartz or other forms of crystalline silica. But for estimating exposure to fire-derived PM_4, these data show a reasonably strong relationship over all USFS regions and during most tasks.

Conclusions

We found that among 83 firefighters at prescribed burns, and 417 at project wildfires, the Occupational Safety and Health Administration (OSHA) 8-hour exposure level of 50 ppm for CO was exceeded 3.5 percent of the time at prescribed fires and 5.6 percent of the time at project fires. Mop-up was a statistically significant cause of exposure during fire operations. Reducing the amount of time firefighters perform mop-up operations will be crucial to reducing their exposure to CO, respirable particulate matter, and quartz. Crew leaders, division supervisors, and other overhead personnel must understand that excessive mop-up places firefighters at increased risk of exposure. Mopup guidelines should be guided by the principal of the right tool, at the right time, at the right place, for the right duration and the right reason. The data collection gathered substantial information about many factors that, together, influence occupational exposures among wildland firefighters. These included crew type, experience of the firefighter and their supervisor, work activity, fuel type and canopy percentage, fire behavior, flame length, wind speed and position relative to the fire, slope and position relative to the fire, USFS Region, and other variables. The data were gathered to determine whether exposures observed across the country were similar to results obtained in the western US in the 1990s, and to help determine factors or conditions that drive high occupational exposures to smoke and dust. By identifying these factors or conditions, management interventions might be developed to reduce exposures among the workforce.

Aim 3 - Wildland Fire Smoke Exposure and Disease Outcomes

Introduction

There is wide agreement that exposure to the mixture of components that we characterize as urban air pollution can be unhealthy and that such exposures worsen the frequency and severity of lung disease episodes (including respiratory tract infections, chronic diseases such as bronchitis or emphysema and inflammatory diseases such as asthma) and can directly or indirectly affect people's hearts. Epidemiological studies have been major contributors to our understanding of the relationship between ambient particulate matter (PM) and cardiac morbidity and mortality with both short- and long-term exposure (Beckerman et al., 2012; Cesaroni et al., 2013; Coogan et al., 2012; R. J. Delfino et al., 2009; Dominici et al., 2006; Lipsett et al., 2011; Simkhovich, Kleinman, & Kloner, 2008; Zhang et al., 2009).

PM cardiovascular health risks have been evaluated in various epidemiologic studies looking at fatal and non-fatal outcomes (Robert D. Brook et al., 2010). Miller et al, examined postmenopausal women without prior CVD, and noted an association between long-term $PM_{2.5}$ exposure and cardiovascular events (Miller et al., 2007). In their study, they showed that an additional 10 ug/m^3 of $PM_{2.5}$ was associated with a 24% increase in all first cardiovascular events (Miller et al., 2007). Several PM cohort studies have yielded results pertaining to specific CV diseases such as heart failure, peripheral vascular disease, and ischemic heart disease which may help mechanistically understand the potential overall toxic effects of PM (Robert D. Brook et al., 2010).

The American Cancer Society (ACS) Cancer Prevention Study associated heart failure deaths with prolonged $PM_{2.5}$ exposures (Pope et al., 2004). Daily hospitalizations for heart failure are associated with short term PM exposures. A 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 1.28% increase in heart failure admissions. Additionally, a reduction of $PM_{2.5}$ by 10 $\mu g/m^3$ was estimated to reduce heart failure admissions in 204 counties (Dominici et al., 2006). Additionally, regarding peripheral vascular disease, a study from Italy by Baccarelli et al., found a 70% increase in risk of deep vein thrombosis per 10 ug/m3 elevation in long-term PM_{10} level (Baccarelli et al., 2008). Exposure to elevated PM levels are associated with acute increases in systemic arterial blood pressure (BP), by about 1 to 4 mmHg per 10 $\mu g/m^3$ elevation in PM (Ibald-Mulli, Stieber, Wichmann, Koenig, & Peters, 2001; Zanobetti et al., 2004). Additionally, Dvonch et al., demonstrated increases in systolic BP and daily elevations in $PM_{2.5}$ in a study of 347 adults living in three communities within metropolitan Detroit, Michigan (Dvonch et al., 2009). The relationship between exposures and the risk of deaths from lung cancer or heart disease varies in a non-linear manner going from low to high doses. For CVD mortality the exposure–response function is relatively steep at very low levels of exposure (in the range associated with exposure to SHS and ambient air pollution) and flattens out at higher doses. For lung cancer mortality, excess risks increase in a nearly linear manner over the full range of exposure and exposure duration appeared to have a much larger impact (C. A. Pope et al., 2011).

The objective of this study was to estimate lifetime disease risk in wildland firefighters for exposure to particulate matter from smoke using firefighter-specific breathing rates with existing exposure response relationship information for risk of lung cancer, ischemic heart disease and cardiovascular disease. We hypothesized that $PM_{2.5}$ health effects data can be used as a surrogate to estimate health risks from wildfire smoke exposure for firefighters. After correcting for the mass contributions of coarse PM, $PM_{2.5}$ and the respirable $PM_{3.5}$ (or PM_4) measured for firefighter occupational exposure, the respirable and fine particulate metrics are similar enough that we can use $PM_{2.5}$-derived health outcome data to estimate wildland firefighter health risks.

Methods

Wildland Firefighter Estimated Shift Dose of $PM_{2.5}$

To assess the exposure-response relationship, we calculated the estimated shift dose of $PM_{2.5}$ using the following equation to calculate a shift dose of $PM_{2.5}$ for wildland firefighters at different fire events:

Eq 1 - $Shift\ dose\ PM_{2.5}(mg) = Duration\ \left(\frac{hrs}{shift}\right) \times\ Exposure\ Concentration\ \left(\frac{mg}{m^3}\right) \times\ Breathing\ Rate\ \left(\frac{L}{min}\right) \times\ CF\ \times\ f$

CF – Conversion Factors ($\frac{60\ min}{hr}$) and ($\frac{m^3}{1000\ liters}$)

f – frequency of exposure ($\frac{shift\ days\ per\ year}{365\ days\ per\ year} \times \frac{years\ of\ firefighting\ career}{45\ years}$)

Shift Duration and Frequency

For this assessment we used wildland firefighter shift duration measurements from the field sampling campaign summarized in Aim 2. Days worked per year were extracted from Booze et al. 2004, for Type 1 crews. Although not all firefighters will have a career only working on a Type 1 crew, we decided this metric would be the most health conservative. Career years were chosen based on the comparison groups used in Semmens et al. 2016 and were adjusted by 45 years, which is the working career of an individual accoarding to OSHA. Manager risk was only calculated for 10 years, as it a position that is generally held at the end of someone's wildland firefighting career. Below is the average shift duration and exposure frequency scenarios at each wildland fire event.

Wildland Fire Event	Duration (hrs)	Days worked per Year	Career Duration
Prescribed Burn	10.5 ± 2.2	5	10, 15, 20 years
Prescribed Natural Fire	13.6 ± 2.2	64	10, 15, 20 years
Fire Project Crew	13.6 ± 1.5	64	10, 15, 20 years
Initial Attack	12.4 ± 3.6	64	10, 15, 20 years
Project Fire Manager	14.5 ± 2.2	64	10years

Exposure Concentration of PM

To use the exposure response relationship that has been developed from air pollution and cigarette epidemiology studies we had to make some assumptions to complete this study. Data from many combustion studies have demonstrated that the particle size of combustion-generated particles is on the order of 300 nm. Thus, even though conventional occupational PM samples collect particles with aerodynamic cut size of 3.5 µm, most of the wildfire smoke exposure is to submicron particles. This compares well to smoke from burning cigarette tobacco. When the size distribution of particles from wildfire smoke was measured during an aerosol study in Yosemite National Park (McMeeking et al., 2006) using both an optical particle counter and a differential mobility analyzer, the mass median particle diameter was about 300 nm. Concentrations of commonly used wood smoke tracers and backward trajectory analyses are used to identify periods when the influence of wood smoke dominated the aerosol concentrations at the park and those when other sources were dominant. Volume geometric mean diameters ranged from ~200 nm during non-smoke periods to between 300 and 400 nm during periods of highest fine aerosol mass concentrations associated with smoke-impacted times(McMeeking et al., 2006). Composition of $PM_{2.5}$ was determined to be dominated by organic carbon for most of the study period. Kleeman et al. (Kleeman, Schauer, & Cass, 1999) measured the particle sizes of smoke aerosol from several different types of wood, under laboratory conditions, and found that particles ranged from about 90 to about 300 nm in mass median diameter and that smoke from conventional cigarettes ranged from 300 to 400 nm. The data from personal PM_4 aerosol samples collected on firefighters during typical firefighting activities would however contain some contribution from other, mostly crustal materials, which would bias our estimates of smoke-related risks that would be based on mortality-exposure relationships for individuals exposed to $PM_{2.5}$ from tobacco smoke and conventional air pollution (Pope et al., 2009; C. A. Pope et al., 2011).

For this analysis, we used measured smoke concentration of PM from the extensive field campaign that was completed by George Broyles and was discussed in Aim 2. To estimate the amount of $PM_{2.5}$ from smoke, we subtracted out the respirable crystalline silica which accounts for soil dust exposure and estimates the measured PM_4 concentrations for smoke alone. This results in a concentration measurement that should also have a size profile that is similar to $PM_{2.5}$, and so would result in similar actual mass concentrations.

Wildland Fire Event	**Shift Exposure Concentration (mg/m^3)**	
	Mean	95% UCL
Prescribed Burn	0.48	0.72
Prescribed Natural Fire	0.20	0.30
Project Fire Crew	0.51	0.64
Initial Attack	0.15	0.20
Project Fire Manager	0.16	0.21

Breathing Rates for Wildland Firefighters

NTDP measured heart rate and respiratory rate on wildland firefighters performing various work tasks. Generalized estimating equations were used to estimate mean (95%CI) heart rate and respiratory rate, accounting for clustering of repeated observations on the same wildland firefighter. An exchangeable correlation structure was selected for this analysis although results were not sensitive to this decision. The reported heart and respiratory rates are lower than one would suppose for most individuals presumably performing reasonably heavy labor. By comparison, they were comparable to those measured for a trained athlete performing exercise at a relatively mild level (e.g. walking on a treadmill at 10.8 km hr^{-1})(Sracic, 2016). However, firefighters are expected to work at these levels for the duration of a shift and they are conditioned to be able to perform under the conditions of the job. In order to calculate the dose of inhaled $PM_{2.5}$, it was necessary to develop an estimate of breathing rate (liters of air inhaled/minute). Although we have heart rate and respiratory data from actual measurements with firefighters, minute ventilation data was not measured. Therefore, we developed a linear equation to estimate breathing rates for firefighters working at different fire events using using HR and Ve data published by Webb et al. (Webb et al., 2010) to estimate Ve from the measured firefighter HR data. Table 4 and 5 in Appendix III present the results of generalized estimating equations and breathing rate estimation by various work tasks performed by wildland firefighters.

Below are the breathing rates and corresponding work tasks that are commonly performed at each fire type.

Fire Type	**Breathing Rate (L/min)**	**Work Tasks Performed**
Prescribed Burn	21	Holding, Lighting, Standby
Prescribed Natural Fire	25	Handline (indirect), Handline Sawyer (indirect), Holding, Lighting, Standby
Project Fire Crew	25	Hiking, Handline (direct), Handline Sawyer (direct), Pump Operator, Mop-up, Holding, Lighting, Handline (indirect), Handline Sawyer (indirect), Standby
Initial Attack	27	Hiking, Handline (direct), Handline Sawyer (direct), Mop-up, Standby
Project Fire Manager	16	Briefing, ICP, Driving, Standby

Estimation of Disease Risk

Pope et al. (2011) compared exposure–response relationships of $PM_{2.5}$ with lung cancer and cardiovascular mortality and provided data which we used to extrapolate potential risks for firefighters. They performed a cohort study analysis using data for 1.2 million adults that were collected by the American Cancer Society. They used those data to estimate relative risks (RRs) for increments of cigarette smoking after adjusting for various individual risk factors. They modeled RR as a function of estimated daily dose of $PM_{2.5}$ from smoking, ambient air pollution and second hand tobacco smoke (SHS) (C. A. Pope, 3rd et al., 2011). The average daily dose of inhaled $PM_{2.5}$ was estimated by multiplying the average $PM_{2.5}$ concentration by the average daily inhalation rates (cubic meters per day) and plotted against. They used a non-linear regression model to fit the data, a power function, and quantified the exposure-response relationship.

There are some caveats in using these data and applying them to firefighters. First, Pope et al. used the average daily exposure (mg/m^3) as the exposure metric. They did not try to analyze day to day variations. Most of the current smokers were exposed for 25 to 40 years. However, the data show that smoking duration had a much larger impact on lung cancer mortality than on CVD mortality. Additionally, they found that the relationships for the various disease outcomes were not linear as a function of daily dose and that at low exposure levels (relevant to firefighter exposures), cardiovascular deaths accounted for most of the burden of disease, whereas at high levels of $PM_{2.5}$, lung cancer became more important.

For firefighters working life exposures are variable however for our initial analysis we had to simplify the data though the following assumptions:

1. PM_4 from most fires after correction for coarse mode contribution using the measured concentrations of quartz is assumed to be equivalent size, toxicity and carcinogenicity to $PM_{2.5}$ from tobacco smoke, so that the RR estimates from Pope et al. (2011) can be used.
2. Shift dose of $PM_{2.5}$ will be used in place of estimated daily dose of $PM_{2.5}$
3. Pope et al. (2011) suggested that because relevant biological pathways for CVD may be activated at low levels of exposure and that increasing exposure further increases risk, but at a decreasing marginal rate, even short term exposures can have a long term, adverse outcome.
4. We only used data from the lower part of the exposure-response curve, where we believed that the exposures to be relevant for wildland firefighter exposure. ($PM_{2.5}$ dose from 0.18 – 66 mg)

Using Pope et al. (2011), we modeled the RR for ischemic heart disease (IHD), cardiovascular disease (CVD) and lung cancer (LC) mortality using a power function. The power function had the functional form of:

$$Relative\ Risk = 1 + \alpha(dose)^{\beta}$$

To create a power function for the part of the exposure response curve that we interested in, we first converted the adjusted excess risk (adjusted RR -1) and daily to their natural logarithms and then a linear regression analysis was performed. The linear regression models were then transformed back into power functions for each disease outcome.

Results and Discussion

Estimation of Disease Risk

From the Pope et al (2011) exposure response data we derived the following power functions to calculate risk for each disease outcome:

Power Function Form - $Relative\ Risk = 1 + \alpha(dose)^{\beta}$

Disease Outcome	**α (95%CI)**	**β (95%CI)**
Lung Cancer	0.47 (0.29, 0.78)	0.74 (0.53, 0.96)
Ischemic Heart Disease	0.29 (0.27, 0.32)	0.22 (0.15, 0.30)
Cardiovascular Disease	0.27 (0.22, 0.33)	0.24 (0.13, 0.34)

Calculation of Disease Risk

Using the calculated shift dose of $PM_{2.5}$ (mean and 95% UCL, Table 6 Appendix II) with the exposure response relationship we derived we calculated the following relative risk of disease development for each fire type:

Relative Risk with no adjustment for days worked or career length

Fire Event	**Shift Dose mg/day**		**Relative Risk**					
			Lung Cancer		**Ischemic Heart Disease**		**Cardiovascular Disease**	
	Mean	**95% UCL**	**Mean**	**95% UCL**	**Mean**	**95% UCL**	**Mean**	**95% UCL**
Prescribed Burn	6.05	9.07	2.78	3.40	1.43	1.47	1.42	1.46
Prescribed Natural Fire	3.75	5.63	2.25	2.69	1.39	1.42	1.37	1.41
Project Fire Crew	9.99	12.53	3.58	4.05	1.48	1.51	1.47	1.50
Initial Attack	2.79	3.72	2.00	2.24	1.36	1.39	1.35	1.37
Project Fire Manager	2.23	2.92	1.85	2.04	1.35	1.37	1.33	1.35

Relative lifetime risk was determined for all activities assuming 5 days per year exposure at a prescribed burn and 64 days at all other fire event for a lifetime

10-year wildland firefighting career

Fire Event	**Shift Dose mg/day**		**Relative Risk**					
			Lung Cancer		**Ischemic Heart Disease**		**Cardiovascular Disease**	
	Mean	**95% UCL**	**Mean**	**95% UCL**	**Mean**	**95% UCL**	**Mean**	**95% UCL**
Prescribed Burn	0.02	0.03	1.02	1.03	1.12	1.13	1.10	1.11
Prescribed Natural Fire	0.15	0.22	1.11	1.15	1.19	1.21	1.17	1.19
Project Fire Crew	0.39	0.49	1.23	1.28	1.24	1.25	1.22	1.23
Initial Attack	0.11	0.14	1.09	1.11	1.18	1.19	1.16	1.17
Project Fire Manager	0.09	0.11	1.08	1.09	1.17	1.18	1.15	1.16

15-year wildland firefighting career

Fire Event	**Shift Dose mg/day**		**Relative Risk**					
			Lung Cancer		**Ischemic Heart Disease**		**Cardiovascular Disease**	
	Mean	**95% UCL**	**Mean**	**95% UCL**	**Mean**	**95% UCL**	**Mean**	**95% UCL**
Prescribed Burn	0.03	0.04	1.03	1.04	1.13	1.14	1.11	1.13
Prescribed Natural Fire	0.22	0.33	1.15	1.21	1.21	1.23	1.19	1.21
Project Fire Crew	0.58	0.73	1.32	1.37	1.26	1.27	1.24	1.25
Initial Attack	0.16	0.22	1.12	1.15	1.19	1.21	1.17	1.19
Project Fire Manager	0.13	0.17	1.10	1.13	1.19	1.20	1.17	1.18

20-year wildland firefighting career

Fire Event	**Shift Dose mg/day**		**Relative Risk**					
			Lung Cancer		**Ischemic Heart Disease**		**Cardiovascular Disease**	
	Mean	**95% UCL**	**Mean**	**95% UCL**	**Mean**	**95% UCL**	**Mean**	**95% UCL**
Prescribed Burn	0.04	0.06	1.04	1.06	1.14	1.15	1.12	1.13
Prescribed Natural Fire	0.29	0.44	1.19	1.26	1.22	1.24	1.20	1.22
Project Fire Crew	0.78	0.98	1.39	1.46	1.27	1.29	1.25	1.27
Initial Attack	0.22	0.29	1.15	1.19	1.21	1.22	1.19	1.20
Project Fire Manager	0.17	0.23	1.13	1.16	1.20	1.21	1.18	1.19

Relative lifetime risk was determined for all activities assuming 5 days per year exposure at a prescribed burn and 64 days at all other fire event for a 10, 15 and 20 year firefighting career respectively.

There are several assumptions that need to be addressed. First, these are long term risks associated with exposures over a working life. In future analyses, the values should be adjusted by the demographics and other characteristics. Over an individual's career he or she will serve in many tasks and activities. Thus the weighted average of exposures over the course of careers provides a realistic predictor of overall risk. Additionally, this analysis only considered the size of the PM and how it compared across wood smoke, ambient air pollution and cigarette smoke, we did not address any of the differences in chemical composition of these sources. In future analyses, it would be ideal to characterize the toxicity and health risk of the different chemical components of smoke.

The number of studies of firefighter mortality is relatively small. Other investigators have reported excess mortality for structural firefighters. Baris et al. observed statistically significant excess risks ischemic heart disease (SMR = 1.09)(Baris et al., 2001). Daniels et al. observed statistically significant positive associations between fire-hours and leukemia and lung cancer mortality where the lung cancer associations were nearly linear in cumulative exposure, while the association with leukemia mortality was attenuated at higher exposure levels and greater for recent exposures (Daniels et al., 2015; Daniels et al., 2014). Female firefighters in Florida had similar morality patterns to non-occupationally exposed Florida women except for atherosclerotic heart disease (SMR = 3.85; 95% CI: 1.66-7.58).(Ma et al., 2005).

Conclusions

Despite wildland firefighters being in superb physical condition inhaled particulate matter can increase the risk of premature mortality from heart disease or cancer. The risk for lung cancer mortality increases nearly linearly with exposures over time and is more strongly influenced by exposure duration than are the risks of death from cardiovascular or ischemic heart disease. On the other hand the risk of cardiovascular mortality rises steeply for doses in the range we estimated for firefighter exposures but flattens out at higher doses (C. A. Pope et al., 2011).

Recommendations

There is strong evidence that acute episodic wildland fire smoke exposure is associated with respiratory effects among the general population, while current evidence of an association with cardiovascular effects is weak. Most of the research of health effects among the general population that has been conducted is based on the ecological time series design, and relies on ambient air concentrations of PM as the measure of exposure and medical visits or mortality as the measure of health outcome. The inability to assess exposure on the individual level within this study design limits the power to detect small effect sizes that may be associated with an episodic event such as wildfires. The greater likelihood that protective action will be taken by susceptible persons biases their exposure upwards and effect sizes towards the null. Perhaps accounting for pre-existing disease in such analysis could help ameliorate this problem. The effect windows used in the studies are typically less than six days. However, effects may be delayed and patients may not make medical visits until symptoms become severe. As such effects of wildland fire smoke exposure may be underestimated especially for respiratory outcomes (Delfino et al., 2008). Additionally, cardiovascular and respiratory effects of wildland fire smoke could be due to other components apart from PM (Delfino et al., 2008). Such association could be explored as has been done for typical ambient air pollution studies. Therefore, it is necessary to continue to develop new exposure metrics that can accurately assess the general population’s smoke exposure. Additionally, we should continue to use and develop real-time air monitoring networks that can inform communities that are impacted by wildland fire smoke on exposure mitigation strategies based on the level of smoke exposure.

For the past 25 years many researchers have made recommendations to wildland fire management agencies to minimize exposure. Many of these recommendations are consistent across all studies – (1) Minimize mopup. (2) Develop a medical surveillance program. (3) Develop wildland fire-specific OELs. (4) Train firefighters on smoke hazards. (5) Reduce exposure by limiting shift length, and rotate crews out of heavy smoke areas. Based on the findings from this project there has been no appreciable reduction in firefighter exposure and in some instances unsafe exposures are more severe than observed by previous research. Exposure to wildland smoke has direct consequences on the ability of firefighters to remain safe by compromising their ability to think clearly and function at their highest mental and physical level. Exposure to the harmful constituents in wildland smoke underlies virtually every aspect of risk management and must be addressed effectively in order to assure other risk management decisions are sound. Therefore, it is essential that sound smoke exposure mitigation strategies be developed, implemented, and enforced.

Convene a task group of industrial hygiene experts, interagency incident management team members, firefighters, risk management, and safety representatives to develop a smoke mitigation implementation plan. Training and education about smoke exposure and the associated hazards are not required components in any of the basic and intermediate levels of required wildland firefighting training (Hyde et al. 2011). Every wildland firefighter must understand the risk associated with smoke exposure and learn how to reduce exposure. Training also must be given to agency administrators and incident management team members who make decisions that affect the exposure to firefighters. Suppression response and mop-up guidelines need to be made that are consistent with operational needs and firefighter safety. Otherwise, risk is being transferred to firefighters rather than being shared with all including the incident commander, agency administrators, and the public.

The data presented in this paper clearly identify the crews and activities most likely to exceed OELs. The USFS is currently managing a wildland firefighter smoke exposure surveillance program. This surveillance program can now focus on these specific crews and activities. However, the program does not have long-term funding commitments. This program should be fully supported with funding and resources by the interagency community to assure its long-term success and ability to identify successful exposure mitigation strategies.

The NWCG Training, Operations & Training and Risk Management Committees should work together to assure smoke exposure hazards are included in NWCG courses beginning with basic wildland fire curriculum through advanced courses.

Acronyms

ACGIH – American Conference on Governmental Industrial Hygiensts

ANOVA - Analysis of Variance

CO – Carbon Monoxide

COPD – Chronic Obstructive Pulmonary Disease

CVD – Cardiovascular Disease

FEF$_{25-75}$ - Maximum Mid-Expiratory Flow

FEV$_1$ - Forced Expiratory Volume in 1 Sec

FVC - Forced Vital Capacity

ICC - Intraclass Correlation Coefficient

IDLH - Immediately Dangerous To Life or Health

IHC – Interagency Hotshot Crew

IHD – Ischemic Heart Disease

LC – Lung Cancer

LCL – Lower Confidence Level

MLM - "Multilevel" Mixed-Effects Models

NAAQS – National Ambient Air Quality Standards

NIOSH – National Institute for Occupational Safety and Health

NTDP – National Technology and Development Program

NWCG – National Wildfire Coordinating Group

PM – Particulate Matter

PEL – Permissible Exposure Limit

OSHA – Occupational Safety and Health Administration

OEL – Occupational Exposure Limit

STEL – Short Term Exposure Limit

TWA – Time Weighted Average

UCL – Upper Confidence Level

USEPA – United States Environmental Protection Agency

USFS – United States Forest Service

References

Adetona, O., Hall, D. B., & Naeher, L. P. (2011b). Lung function changes in wildland firefighters working at prescribed burns. *Inhalation Toxicology, 23*(13), 835-841.

Adetona, O., Simpson, C. D., Onstad, G., & Naeher, L. P. (2013a). Exposure of wildland firefighters to carbon monoxide, fine particles, and levoglucosan. *Annals of occupational hygiene*, met024.

Adetona, O., Zhang, J. J., Hall, D. B., Wang, J.-S., Vena, J. E., & Naeher, L. P. (2013b). Occupational exposure to woodsmoke and oxidative stress in wildland firefighters. *Science of the total environment, 449*, 269-275.

Akagi, S., Yokelson, R. J., Burling, I. R., Meinardi, S., Simpson, I., Blake, D. R., Kreidenweis, S. (2013). Measurements of reactive trace gases and variable O 3 formation rates in some South Carolina biomass burning plumes. *Atmospheric Chemistry and Physics, 13*(3), 1141-1165.

Alföldy, B., Giechaskiel, B., Hofmann, W., & Drossinos, Y. (2009). Size-distribution dependent lung deposition of diesel exhaust particles. *Journal of Aerosol Science, 40*(8), 652-663.

Alonso-Blanco, E., Calvo, A., Fraile, R., & Castro, A. (2012). The Influence of wildfires on aerosol size distributions in rural areas. *The Scientific World Journal, 2012*.

Alves, C., Gonçalves, C., Evtyugina, M., Pio, C., Mirante, F., & Puxbaum, H. (2010a). Particulate organic compounds emitted from experimental wildland fires in a Mediterranean ecosystem. *Atmospheric Environment, 44*(23), 2750-2759.

Alves, C., Gonçalves, C., Pio, C., Mirante, F., Caseiro, A., Tarelho, L., . . . Viegas, D. (2010b). Smoke emissions from biomass burning in a Mediterranean shrubland. *Atmospheric Environment, 44*(25), 3024-3033.

Alves, C., Vicente, A., Nunes, T., Gonçalves, C., Fernandes, A. P., Mirante, F., . . . Caseiro, A. (2011). Summer 2009 wildfires in Portugal: emission of trace gases and aerosol composition. *Atmospheric Environment, 45*(3), 641-649.

Annane, D., Chadda, K., Gajdos, P., Jars-Guincestre, M.-C., Chevret, S., & Raphael, J.-C. (2011). Hyperbaric oxygen therapy for acute domestic carbon monoxide poisoning: two randomized controlled trials. *Intensive care medicine, 37*(3), 486-492.

Anttila, P., Makkonen, U., Hellén, H., Kyllönen, K., Leppänen, S., Saari, H., & Hakola, H. (2008). Impact of the open biomass fires in spring and summer of 2006 on the chemical composition of background air in south-eastern Finland. *Atmospheric Environment, 42*(26), 6472-6486.

Araujo, J. A., & Nel, A. E. (2009). Particulate matter and atherosclerosis: role of particle size, composition and oxidative stress. *Particle and Fibre Toxicology, 6*(1), 24.

Baccarelli, A., Martinelli, I., Zanobetti, A., Grillo, P., Hou, L.-F., Bertazzi, P. A., . . . Schwartz, J. (2008). Exposure to particulate air pollution and risk of deep vein thrombosis. *Archives of Internal Medicine, 168*(9), 920-927. doi:10.1001/archinte.168.9.920

Balachandran, S., Pachon, J. E., Lee, S., Oakes, M. M., Rastogi, N., Shi, W., . . . Zhang, X. (2013). Particulate and gas sampling of prescribed fires in South Georgia, USA. *Atmospheric Environment, 81*, 125-135.

Baris, D., Garrity, T. J., Telles, J. L., Heineman, E. F., Olshan, A., & Zahm, S. H. (2001). Cohort mortality study of Philadelphia firefighters. *Am J Ind Med, 39*(5), 463-476.

Barregard, L., Sällsten, G., Andersson, L., Almstrand, A.-C., Gustafson, P., Andersson, M., & Olin, A.-C. (2008). Experimental exposure to wood smoke: effects on airway inflammation and oxidative stress. *Occupational and Environmental Medicine, 65*(5), 319-324.

Beckerman, B. S., Jerrett, M., Finkelstein, M., Kanaroglou, P., Brook, J. R., Arain, M. A., . . . Chapman, K. (2012). The association between chronic exposure to traffic-related air pollution and ischemic heart disease. *Journal of toxicology and environmental health. Part A, 75*(7), 402-411. doi:10.1080/15287394.2012.670899

Betchley, C., Koenig, J. Q., van Belle, G., Checkoway, H., & Reinhardt, T. (1997). Pulmonary function and respiratory symptoms in forest firefighters. *American journal of industrial medicine, 31*(5), 503-509.

Bølling, A. K., Pagels, J., Yttri, K. E., Barregard, L., Sallsten, G., Schwarze, P. E., & Boman, C. (2009). Health effects of residential wood smoke particles: the importance of combustion conditions and physicochemical particle properties. *Particle and Fibre Toxicology, 6*(29), 20.

Borsboom, G. J., van Pelt, W., van Houwelingen, H. C., van Vianen, B. G., Schouten, J. P., & Quanjer, P. H. (1999). Diurnal variation in lung function in subgroups from two Dutch populations: consequences for longitudinal analysis. *American Journal of Respiratory and Critical Care Medicine, 159*(4), 1163-1171.

Brook, R. D., Brook, J. R., Urch, B., Vincent, R., Rajagopalan, S., & Silverman, F. (2002). Inhalation of fine particulate air pollution and ozone causes acute arterial vasoconstriction in healthy adults. *Circulation, 105*(13), 1534-1536.

Brook, R. D., Rajagopalan, S., Pope, C. A., Brook, J. R., Bhatnagar, A., Diez-Roux, A. V., . . . Mittleman, M. A. (2010). Particulate matter air pollution and cardiovascular disease an update to the scientific statement from the American Heart Association. *Circulation, 121*(21), 2331-2378.

Brook, R. D., Rajagopalan, S., Pope, C. A., III, Brook, J. R., Bhatnagar, A., Diez-Roux, A. V., . . . Council Nutr Phys Activity, M. (2010). Particulate Matter Air Pollution and Cardiovascular Disease An Update to the Scientific Statement From the American Heart Association. *Circulation, 121*(21), 2331-2378. doi:10.1161/CIR.0b013e3181dbece1

Burling, I., Yokelson, R. J., Griffith, D. W., Johnson, T. J., Veres, P., Roberts, J., . . . Weise, D. (2010). Laboratory measurements of trace gas emissions from biomass burning of fuel types from the southeastern and southwestern United States. *Atmospheric Chemistry and Physics, 10*(22), 11115-11130.

Cashdollar, K. L., Lee, C. K., & Singer, J. M. (1979). Three-wavelength light transmission technique to measure smoke particle size and concentration. *Applied Optics, 18*(11), 1763-1769.

Cesaroni, G., Badaloni, C., Gariazzo, C., Stafoggia, M., Sozzi, R., Davoli, M., & Forastiere, F. (2013). Long-term exposure to urban air pollution and mortality in a cohort of more than a million adults in rome. *Environmental Health Perspectives, 121*(3), 324-331. doi:10.1289/ehp.1205862

Chakrabarty, R. K., Moosmüller, H., Garro, M. A., Arnott, W. P., Walker, J., Susott, R. A., . . . Hao, W. M. (2006). Emissions from the laboratory combustion of wildland fuels: Particle morphology and size. *Journal of Geophysical Research: Atmospheres (1984–2012), 111*(D7).

Cherrie, J. W., Brosseau, L. M., Hay, A., & Donaldson, K. (2013). Low-toxicity dusts: current exposure guidelines are not sufficiently protective. *Annals of occupational hygiene, 57*(6), 685-691.

Coogan, P. F., White, L. F., Jerrett, M., Brook, R. D., Su, J. G., Seto, E., . . . Rosenberg, L. (2012). Air Pollution and Incidence of Hypertension and

Diabetes Mellitus in Black Women Living in Los Angeles. *Circulation, 125*(6), 767-772.

Daniels, R. D., Bertke, S., Dahm, M. M., Yiin, J. H., Kubale, T. L., Hales, T. R., . . . Pinkerton, L. E. (2015). Exposure-response relationships for select cancer and non-cancer health outcomes in a cohort of U.S. firefighters from San Francisco, Chicago and Philadelphia (1950-2009). *Occup Environ Med, 72*(10), 699-706. doi:10.1136/oemed-2014-102671

Daniels, R. D., Kubale, T. L., Yiin, J. H., Dahm, M. M., Hales, T. R., Baris, D., . . . Pinkerton, L. E. (2014). Mortality and cancer incidence in a pooled cohort of US firefighters from San Francisco, Chicago and Philadelphia (1950-2009). *Occup Environ Med, 71*(6), 388-397. doi:10.1136/oemed-2013-101662

Danielsen, P. H., Loft, S., Jacobsen, N. R., Jensen, K. A., Autrup, H., Ravanat, J.-L., . . . Møller, P. (2010). Oxidative stress, inflammation and DNA damage in rats after intratracheal instillation or oral exposure to ambient air and wood smoke particulate matter. *Toxicological sciences*, kfq290.

Delfino, R. J., Brummel, S., Wu, J., Stern, H., Ostro, B., Lipsett, M., . . . Tjoa, T. (2008). The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003. *Occupational and Environmental Medicine*.

Delfino, R. J., Staimer, N., Tjoa, T., Gillen, D. L., Polidori, A., Arhami, M., . . . Sioutas, C. (2009). Air Pollution Exposures and Circulating Biomarkers of Effect in a Susceptible Population: Clues to Potential Causal Component Mixtures and Mechanisms. *Environmental Health Perspectives, 117*(8), 1232-1238.

Dominici, F., Peng, R. D., Bell, M. L., Pham, L., McDermott, A., Zeger, S. L., & Samet, J. M. (2006). Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *Jama-Journal of the American Medical Association, 295*(10), 1127-1134. doi:10.1001/jama.295.10.1127

Dunn, K., Shulman, S., Stock, A., & Naeher, L. (2013). Personal carbon monoxide exposures among firefighters at prescribed forest burns in the southeastern United States. *Archives of environmental & occupational health, 68*(1), 55-59.

Dvonch, J. T., Kannan, S., Schulz, A. J., Keeler, G. J., Mentz, G., House, J., . . . Brook, R. D. (2009). Acute Effects of Ambient Particulate Matter on Blood

Pressure Differential Effects Across Urban Communities. *Hypertension, 53*(5), 853-859. doi:10.1161/hypertensionaha.108.123877

Ferek, R. J., Reid, J. S., Hobbs, P. V., Blake, D. R., & Liousse, C. (1998). Emission factors of hydrocarbons, halocarbons, trace gases and particles from biomass burning in Brazil. *Journal of Geophysical Research: Atmospheres (1984–2012), 103*(D24), 32107-32118.

Forchhammer, L., Moller, P., Riddervold, I. S., Bonlokke, J., Massling, A., Sigsgaard, T., & Loft, S. (2012). Controlled human wood smoke exposure: oxidative stress, inflammation and microvascular function. *Particle and Fibre Toxicology, 9*(7), 8977-8979.

Gaughan, D. M., Cox-Ganser, J. M., Enright, P. L., Castellan, R. M., Wagner, G. R., Hobbs, G. R., . . . Weissman, D. N. (2008). Acute upper and lower respiratory effects in wildland firefighters. *Journal of Occupational and Environmental Medicine, 50*(9), 1019-1028.

Gaughan, D. M., Siegel, P. D., Hughes, M. D., Chang, C. Y., Law, B. F., Campbell, C. R., . . . Kobzik, L. (2014b). Arterial stiffness, oxidative stress, and smoke exposure in wildland firefighters. *American journal of industrial medicine, 57*(7), 748-756.

Ghelfi, E., Rhoden, C. R., Wellenius, G. A., Lawrence, J., & Gonzalez-Flecha, B. (2008). Cardiac oxidative stress and electrophysiological changes in rats exposed to concentrated ambient particles are mediated by TRP-dependent pulmonary reflexes. *Toxicological sciences, 102*(2), 328-336.

Ghio, A. J., Soukup, J. M., Case, M., Dailey, L. A., Richards, J., Berntsen, J., . . . Rappold, A. (2012). Exposure to wood smoke particles produces inflammation in healthy volunteers. *Occupational and Environmental Medicine, 69*(3), 170-175.

Guzman, J. A. (2012). Carbon monoxide poisoning. *Critical care clinics, 28*(4), 537-548.

Heil, A., & Goldammer, J. (2001). Smoke-haze pollution: A review of the 1997 episode in Southeast Asia. *Regional Environmental Change, 2*(1), 24-37.

Hejl, A. M., Adetona, O., Diaz-Sanchez, D., Carter, J. D., Commodore, A. A., Rathbun, S. L., & Naeher, L. P. (2013). Inflammatory effects of woodsmoke exposure among wildland firefighters working at prescribed burns at the Savannah River Site, SC. *Journal of occupational and environmental hygiene, 10*(4), 173-180.

Henderson, S. B., Brauer, M., MacNab, Y. C., & Kennedy, S. M. (2011). Three measures of forest fire smoke exposure and their associations with respiratory and cardiovascular health outcomes in a population-based cohort. *Environmental Health Perspectives, 119*(9), 1266.

Hunter, A. L., Unosson, J., Bosson, J. A., Langrish, J. P., Pourazar, J., Raftis, J. B., . . . Nyström, R. (2014). Effect of wood smoke exposure on vascular function and thrombus formation in healthy fire fighters. *Particle and Fibre Toxicology, 11*(1), 62.

Ibald-Mulli, A., Stieber, J., Wichmann, H. E., Koenig, W., & Peters, A. (2001). Effects of air pollution on blood pressure: A population-based approach. *American Journal of Public Health, 91*(4), 571-577.

Iinuma, Y., Brüggemann, E., Gnauk, T., Müller, K., Andreae, M., Helas, G., . . . Herrmann, H. (2007). Source characterization of biomass burning particles: The combustion of selected European conifers, African hardwood, savanna grass, and German and Indonesian peat. *Journal of Geophysical Research: Atmospheres (1984–2012), 112*(D8).

Invernizzi, G., Boffi, R., Ruprecht, A., Barnes, P., Kharitonov, S., & Paredi, P. (2006). Real-time measurement of particulate matter deposition in the lung. *Biomarkers, 11*(3), 221-232.

Jacquin, L., Michelet, P., Brocq, F. X., Houel, J. G., Truchet, X., Auffray, J. P., . . . Jammes, Y. (2011). Short-term spirometric changes in wildland firefighters. *American journal of industrial medicine, 54*(11), 819-825.

Johnston, F. H., Webby, R. J., Pilotto, L. S., Bailie, R. S., Parry, D. L., & Halpin, S. J. (2006). Vegetation fires, particulate air pollution and asthma: a panel study in the Australian monsoon tropics. *International journal of environmental health research, 16*(6), 391-404.

Keywood, M. D., Ayers, G. P., Gras, J. L., Gillett, R. W., & Cohen, D. D. (2000). Size distribution and sources of aerosol in Launceston, Australia, during winter 1997. *Journal of the Air & Waste Management Association, 50*(3), 418-427.

Kido, T., Tamagawa, E., Bai, N., Suda, K., Yang, H.-H. C., Li, Y., . . . Sin, D. D. (2011). Particulate matter induces translocation of IL-6 from the lung to the systemic circulation. *American journal of respiratory cell and molecular biology, 44*(2), 197-204.

Kleeman, M. J., Schauer, J. J., & Cass, G. R. (1999). Size and composition distribution of fine particulate matter emitted from wood burning, meat charbroiling, and cigarettes. *Environmental Science & Technology,* *33*(20), 3516-3523.

Kristensson, A., Rissler, J., Löndahl, J., Johansson, C., & Swietlicki, E. (2013). Size-resolved respiratory tract deposition of sub-micrometer aerosol particles in a residential area with wintertime wood combustion. *Aerosol and Air Quality Research, 13*(1), 24-35.

Lee, S., Baumann, K., Schauer, J. J., Sheesley, R. J., Naeher, L. P., Meinardi, S., . . . Clements, M. (2005). Gaseous and particulate emissions from prescribed burning in Georgia. *Environmental Science & Technology, 39*(23), 9049-9056.

Lee, S., Kim, H. K., Yan, B., Cobb, C. E., Hennigan, C., Nichols, S., . . . Hu, Y. (2008). Diagnosis of aged prescribed burning plumes impacting an urban area. *Environmental Science & Technology, 42*(5), 1438-1444.

Leonard, S. S., Castranova, V., Chen, B. T., Schwegler-Berry, D., Hoover, M., Piacitelli, C., & Gaughan, D. M. (2007). Particle size-dependent radical generation from wildland fire smoke. *Toxicology, 236*(1), 103-113.

Leonard, S. S., Wang, S., Shi, X., Jordan, B. S., Castranova, V., & Dubick, M. A. (2000). Wood smoke particles generate free radicals and cause lipid peroxidation, DNA damage, NFκB activation and TNF-α release in macrophages. *Toxicology, 150*(1), 147-157.

Li, N., Kim, S., Wang, M., Froines, J., Sioutas, C., & Nel, A. (2002). Use of a stratified oxidative stress model to study the biological effects of ambient concentrated and diesel exhaust particulate matter. *Inhalation Toxicology, 14*(5), 459-486.

Li, N., Xia, T., & Nel, A. E. (2008). The role of oxidative stress in ambient particulate matter-induced lung diseases and its implications in the toxicity of engineered nanoparticles. *Free Radical Biology and Medicine, 44*(9), 1689-1699.

Lipsett, M. J., Ostro, B. D., Reynolds, P., Goldberg, D., Hertz, A., Jerrett, M., . . . Bernstein, L. (2011). Long-Term Exposure to Air Pollution and Cardiorespiratory Disease in the California Teachers Study Cohort. *American Journal of Respiratory and Critical Care Medicine, 184*(7), 828-835.

Liu, D., Tager, I. B., Balmes, J. R., & Harrison, R. J. (1992). The effect of smoke inhalation on lung function and airway responsiveness in wildland fire fighters. *American Review of Respiratory Disease, 146*, 1469-1469.

Ma, F., Fleming, L. E., Lee, D. J., Trapido, E., Gerace, T. A., Lai, H., & Lai, S. (2005).

Mortality in Florida professional firefighters, 1972 to 1999. *Am J Ind Med, 47*(6), 509-517. doi:10.1002/ajim.20160

McMeeking, G. R., Kreidenweis, S. M., Lunden, M., Carrillo, J., Carrico, C. M., Lee, T., . . . Collett, J. L. (2006). Smoke-impacted regional haze in California during the summer of 2002. *Agricultural and Forest Meteorology, 137*(1-2), 25-42.

Miller, K. A., Siscovick, D. S., Sheppard, L., Shepherd, K., Sullivan, J. H., Anderson, G. L., & Kaufman, J. D. (2007). Long-term exposure to air pollution and incidence of cardiovascular events in women. *New England Journal of Medicine, 356*(5), 447-458. doi:10.1056/NEJMoa054409

Mills, N. L., Donaldson, K., Hadoke, P. W., Boon, N. A., MacNee, W., Cassee, F. R., . . . Newby, D. E. (2009). Adverse cardiovascular effects of air pollution. *Nature Clinical Practice Cardiovascular Medicine, 6*(1), 36-44.

Miranda, A. I., Martins, V., Cascão, P., Amorim, J. H., Valente, J., Borrego, C., . . . Ottmar, R. (2012). Wildland smoke exposure values and exhaled breath indicators in firefighters. *Journal of Toxicology and Environmental Health, Part A, 75*(13-15), 831-843.

Naeher, L. P., Brauer, M., Lipsett, M., Zelikoff, J. T., Simpson, C. D., Koenig, J. Q., & Smith, K. R. (2007). Woodsmoke health effects: a review. *Inhalation Toxicology, 19*(1), 67-106.

Pope, C. A., 3rd, Burnett, R. T., Turner, M. C., Cohen, A., Krewski, D., Jerrett, M., . . . Thun, M. J. (2011). Lung cancer and cardiovascular disease mortality associated with ambient air pollution and cigarette smoke: shape of the exposure-response relationships. *Environmental Health Perspectives, 119*(11), 1616-1621. doi:10.1289/ehp.1103639

Pope, C. A., Burnett, R. T., Krewski, D., Jerrett, M., Shi, Y. L., Calle, E. E., & Thun, M. J. (2009). Cardiovascular Mortality and Exposure to Airborne Fine Particulate Matter and Cigarette Smoke Shape of the Exposure-Response Relationship. *Circulation, 120*(11), 941-948.

Pope, C. A., Burnett, R. T., Thurston, G. D., Thun, M. J., Calle, E. E., Krewski, D., & Godleski, J. J. (2004). Cardiovascular mortality and long-term exposure to particulate air pollution - Epidemiological evidence of general pathophysiological pathways of disease. *Circulation, 109*(1), 71-77.

Pope, C. A., Burnett, R. T., Turner, M. C., Cohen, A., Krewski, D., Jerrett, M., . . . Thun, M. J. (2011). Lung Cancer and Cardiovascular Disease Mortality

Associated with Ambient Air Pollution and Cigarette Smoke: Shape of the Exposure-Response Relationships. *Environmental Health Perspectives, 119*(11), 1616-1621.

Portin, H., Mielonen, T., Leskinen, A., Arola, A., Pärjälä, E., Romakkaniemi, S., . . . Komppula, M. (2012). Biomass burning aerosols observed in Eastern Finland during the Russian wildfires in summer 2010–Part 1: In-situ aerosol characterization. *Atmospheric Environment, 47*, 269-278.

Quinn, D. K., McGahee, S. M., Politte, L. C., Duncan, G. N., Cusin, C., Hopwood, C. J., & Stern, T. A. (2009). Complications of carbon monoxide poisoning: a case discussion and review of the literature. *Primary care companion to the Journal of clinical psychiatry, 11*(2), 74.

Raub, J. (1999). Carbon monoxide.

Raub, J. A., Mathieu-Nolf, M., Hampson, N. B., & Thom, S. R. (2000). Carbon monoxide poisoning—a public health perspective. *Toxicology, 145*(1), 1-14.

Reinhardt, T. E., & Ottmar, R. D. (2004). Baseline measurements of smoke exposure among wildland firefighters. *Journal of occupational and environmental hygiene, 1*(9), 593-606.

Reisen, F., & Brown, S. K. (2009). Australian firefighters' exposure to air toxics during bushfire burns of autumn 2005 and 2006. *Environment international, 35*(2), 342-352.

Schwarze, P., Øvrevik, J., Låg, M., Refsnes, M., Nafstad, P., Hetland, R., & Dybing, E. (2006). Particulate matter properties and health effects: consistency of epidemiological and toxicological studies. *Human & experimental toxicology, 25*(10), 559-579.

Serra, A., Mocci, F., & Randaccio, F. S. (1996). Pulmonary function in Sardinian fire fighters. *American journal of industrial medicine, 30*(1), 78-82.

Sillanpää, M., Saarikoski, S., Hillamo, R., Pennanen, A., Makkonen, U., Spolnik, Z., . . . Salonen, R. O. (2005). Chemical composition, mass size distribution and source analysis of long-range transported wildfire smokes in Helsinki. *Science of the total environment, 350*(1), 119-135.

Simkhovich, B. Z., Kleinman, M. T., & Kloner, R. A. (2008). Air pollution and cardiovascular injury epidemiology, toxicology, and mechanisms. *Journal of the American College of Cardiology, 52*(9), 719-726. doi:10.1016/j.jacc.2008.05.029

Slaughter, J. C., Koenig, J. Q., & Reinhardt, T. E. (2004). Association between lung function and exposure to smoke among firefighters at prescribed burns. *Journal of occupational and environmental hygiene, 1*(1), 45-49.

Sracic, M. K. (2016). Modeled regional airway deposition of inhaled particles in athletes at exertion. *Journal of Aerosol Science, 99*, 54-63.

Sutherland, E. R., Make, B. J., Vedal, S., Zhang, L., Dutton, S. J., Murphy, J. R., & Silkoff, P. E. (2005). Wildfire smoke and respiratory symptoms in patients with chronic obstructive pulmonary disease. *Journal of Allergy and Clinical Immunology, 115*(2), 420-422.

Swiston, J. R., Davidson, W., Attridge, S., Li, G. T., Brauer, M., & van Eeden, S. F. (2008). Wood smoke exposure induces a pulmonary and systemic inflammatory response in firefighters. *European Respiratory Journal, 32*(1), 129-138.

Tesfaigzi, Y., Singh, S. P., Foster, J. E., Kubatko, J., Barr, E. B., Fine, P. M., . . . Mauderly, J. L. (2002). Health effects of subchronic exposure to low levels of wood smoke in rats. *Toxicological sciences, 65*(1), 115-125.

Troyanov, S., Ghezzo, H., Cartier, A., & Malo, J.-L. (1994). Comparison of circadian variations using FEV1 and peak expiratory flow rates among normal and asthmatic subjects. *Thorax, 49*(8), 775-780.

Unosson, J., Blomberg, A., Sandström, T., Muala, A., Boman, C., Nyström, R., . . . Langrish, J. P. (2013). Exposure to wood smoke increases arterial stiffness and decreases heart rate variability in humans. *Particle and Fibre Toxicology, 10*(1), 20.

Urbanski, S. (2014). Wildland fire emissions, carbon, and climate: Emission factors. *Forest Ecology and Management, 317*, 51-60.

Verma, V., Polidori, A., Schauer, J. J., Shafer, M. M., Cassee, F. R., & Sioutas, C. (2009). Physicochemical and toxicological profiles of particulate matter in Los Angeles during the October 2007 southern California wildfires. *Environmental Science & Technology, 43*(3), 954-960.

Webb, H. E., McMinn, D. R., Garten, R. S., Beckman, J. L., Kamimori, G. H., & Acevedo, E. O. (2010). Cardiorespiratory responses of firefighters to a computerized fire strategies and tactics drill during physical activity. *Applied Ergonomics, 41*(3), 376-381.

Wegesser, T. C., Franzi, L. M., Mitloehner, F. M., Eiguren-Fernandez, A., & Last, J. A. (2010). Lung antioxidant and cytokine responses to coarse and fine particulate matter from the great California wildfires of 2008. *Inhalation Toxicology, 22*(7), 561-570.

Wolf, S. J., Lavonas, E. J., Sloan, E. P., & Jagoda, A. S. (2008). Clinical policy: critical issues in the management of adult patients presenting to the emergency department with acute carbon monoxide poisoning. *Journal of Emergency Nursing, 34*(2), e19-e32.

Xiao, G. G., Wang, M., Li, N., Loo, J. A., & Nel, A. E. (2003). Use of proteomics to demonstrate a hierarchical oxidative stress response to diesel exhaust particle chemicals in a macrophage cell line. *Journal of Biological Chemistry, 278*(50), 50781-50790.

Yokelson, R. J., Burling, I. R., Gilman, J., Warneke, C., Stockwell, C. E., Gouw, J. d., . . . Roberts, J. M. (2013). Coupling field and laboratory measurements to estimate the emission factors of identified and unidentified trace gases for prescribed fires. *Atmospheric Chemistry and Physics, 13*(1), 89-116.

Zanobetti, A., Canner, M. J., Stone, P. H., Schwartz, J., Sher, D., Eagan-Bengston, E., . . . Gold, D. R. (2004). Ambient pollution and blood pressure in cardiac rehabilitation patients. *Circulation, 110*(15), 2184-2189. doi:01.CIR.0000143831.33243.D8 [pii]

10.1161/01.CIR.0000143831.33243.D8

Zhang, Z. M., Whitsel, E. A., Quibrera, P. M., Smith, R. L., Liao, D. P., Anderson, G. L., & Prineas, R. J. (2009). Ambient Fine Particulate Matter Exposure and Myocardial Ischemia in the Environmental Epidemiology of Arrhythmogenesis in the Women's Health Initiative (EEAWHI) Study. *Environmental Health Perspectives, 117*(5), 751-756.

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