Taylor & Francis
Taylor & Francis Group

Inhalation Toxicology

International Forum for Respiratory Research

ISSN: 0895-8378 (Print) 1091-7691 (Online) Journal homepage: https://www.tandfonline.com/loi/iiht20

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

Olorunfemi Adetona, Timothy E. Reinhardt, Joe Domitrovich, George Broyles, Anna M. Adetona, Michael T. Kleinman, Roger D. Ottmar & Luke P. Naeher

To cite this article: Olorunfemi Adetona, Timothy E. Reinhardt, Joe Domitrovich, George Broyles, Anna M. Adetona, Michael T. Kleinman, Roger D. Ottmar & Luke P. Naeher (2016) Review of the health effects of wildland fire smoke on wildland firefighters and the public, Inhalation Toxicology, 28:3, 95-139, DOI: 10.3109/08958378.2016.1145771

To link to this article: https://doi.org/10.3109/08958378.2016.1145771

Published online: 26 Feb 2016.

Inhalation Toxicology

http://tandfonline.com/iiht
ISSN: 0895-8378 (print), 1091-7691 (electronic)

Inhal Toxicol, 2016; 28(3): 95–139
 DOI: 10.3109/08958378.2016.1145771

Taylor & Francis
Taylor & Francis Group

REVIEW ARTICLE

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

Olorunfemi Adetona[1,2], Timothy E. Reinhardt[3], Joe Domitrovich[4], George Broyles[5], Anna M. Adetona[1], Michael T. Kleinman[6], Roger D. Ottmar[7], and Luke P. Naeher[1]

[1]Department of Environmental Health Science, College of Public Health, University of Georgia, Athens, GA, USA, [2]Division of Environmental Health Sciences, College of Public Health, the Ohio State University, Columbus, OH, USA, [3]AMEC Foster Wheeler Environment & Infrastructure, Inc, Seattle, WA, USA, [4]USDA Forest Service, Missoula Technology and Development Center, Missoula, MT, USA, [5]SDA Forest Service, San Dimas Technology and Development Center, San Dimas, CA, USA, [6]Center for Occupational and Environmental Health, University of California, Irvine, CA, USA, and [7]USDA Forest Service, Pacific Northwest Research Station, Seattle, WA, USA

Abstract

Each year, the general public and wildland firefighters in the US are exposed to smoke from wildland fires. As part of an effort to characterize health risks of breathing this smoke, a review of the literature was conducted using five major databases, including PubMed and MEDLINE Web of Knowledge, to identify smoke components that present the highest hazard potential, the mechanisms of toxicity, review epidemiological studies for health effects and identify the current gap in knowledge on the health impacts of wildland fire smoke exposure. 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 community level exposure to wildland fire smoke. A few recent studies have also determined associations between acute wildland fire smoke exposure and cardiovascular health end-points. These cardiopulmonary effects were mostly observed in association with ambient air concentrations of fine particulate matter ($PM_{2.5}$). However, research on the health effects of this mixture is currently limited. 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 the 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.

Keywords

Cardiovascular effects, respiratory effects, toxicity mechanism, wildfire, wildland firefighter

History

Received 4 November 2015
Revised 15 January 2016
Accepted 20 January 2016
Published online 26 February 2016

Background

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. Due to climate change (Bedia et al., 2014; Johnston et al., 2012; Keywood et al., 2013; Liu et al., 2013), past forest management practices that have contributed to increased fuel loads in forests (Stephens & Ruth, 2005), large scale deforestation fires in developing countries (Silvestrine et al., 2011; Tosca et al., 2011) and increase in the number of people working and/or living in areas adjacent to forested areas which has resulted in the growth of the wildland–urban interface (Radeloff et al., 2005), the risk of exposures to wildfire smoke in both scenarios and their resulting adverse health effects may be expected to rise.

A majority of the investigation into the community level health effects of wildfire smoke exposure has been conducted

Address for correspondence: Luke P. Naeher, PhD, Department of Environmental Health Science, College of Public Health, University of Georgia, Environmental Health Science Building, Athens, GA 30602-2102, USA. Tel: +1 7065422454. E-mail: LNaeher@uga.edu

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).

In addition, 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. Small but measurable acute pulmonary effects have been observed in studies of occupational and community exposures. However, results from various studies including those related to wood smoke exposures from occupational or residential sources indicate possible systemic and long-term effects. Systemic inflammation, acute cardiovascular responses and reduction in birth weight (a delayed effect) are some of the other effects that have been reported.

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:

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. Although this is a pragmatic approach, it should be noted that the application of this set of criteria excludes many components of smoke that are known to be damaging to health, but are not currently regulated e.g. poly-aromatic hydrocarbons (PAHs). This discussion also highlights specific characteristics of wildland fire smoke derived particulate matter in terms of its chemical composition and size distribution.

Identify the components presenting the highest hazard ratios to wildland firefighters and the public based primarily on reported occupational exposure or ambient air concentrations.

Review the evidence for the adverse health impacts of wildfire smoke on wildland firefighters and the public.

Discussion of the possible mechanisms for wildland fire smoke toxicity.

Identification of research needs for determining the health effects of occupational and community level wildfire smoke exposure.

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 calculation of concentrations from emission factors is illustrated in Equation (1).

$$C_{\text{component}} = \left[N_{\text{component}} / N_{\text{CO or CO}_2}\right] \times C_{\text{CO or CO}_2} \quad (1)$$

$C_{\text{component}}$ is the concentration of a component of interest in wildland fire smoke; $N_{\text{component}}$ is the number of moles of the component based on its reported emission factor; $N_{\text{CO or CO}_2}$ is the reported emissions factor for carbon monoxide or carbon dioxide in the same study; $C_{\text{CO or CO}_2}$ is the maximum concentration of carbon monoxide or carbon dioxide as reported by 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 is 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 1. 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.

Wildland fire smoke composition

Smoke from wildland fires is a complex mixture containing hundreds of constituents/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).

Table 1. Literature search results.

Objective	Databases	Search terms	Years searched	Total number of papers	Number of papers selected for review	Number of papers excluded
Wildland fire smoke component and exposure	PubMed MEDLINE Web of Knowledge SportsDiscus Environmental Sciences and Pollution Management (ProQuest) ACS Symposium Series	Wildland fire smoke (or wildfire smoke, woodsmoke, peat fire smoke forest fire smoke, woodland fire smoke, vegetative fire smoke, vegetative fire smoke) and components (or emission, concentration)	1970–2014	219	107	112
Health effects (epidemiology)	PubMed MEDLINE Web of Knowledge SportsDiscus	Health effects (or cardiovascular, respiratory, reproductive, birth weight, preterm, perinatal, stillbirth, infant death) and wood smoke (or biomass smoke, biomass combustion, vegetation smoke, wildfire)	1970–2014	344	198[a]	146
Mechanism of toxicity	PubMed MEDLINE Web of Knowledge SportsDiscus	*In vitro* (or *in vivo*, human experiment, inflammation, oxidative stress, airway, cardiovascular) and wood smoke (or biomass smoke, biomass combustion, wildfire)	1970–2014	200	70[b]	130

[a]This total contains studies of the health effects related to vegetative smoke exposure situations other than wildland fire smoke exposure which were reviewed for supporting evidence. There were a total of 52 studies of health effects of occupational and general occupational exposure to wildland fire smoke.
[b]A total of 70 papers were reviewed with 67 included in the review for evidence directly relevant to the health effects observed in epidemiology studies.

These in turn are a function of fuel characteristics such as its chemistry, bulk density, arrangement and moisture content (Alves et al., 2010b; Burling et al., 2010; Urbanski, 2014). Such emission 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 et al., 1998; Heil & Goldammer, 2001; Urbanski, 2014; Yokelson et al., 2013).

Wildland fuels have 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). In addition, biomass burning is considered to be the second largest global atmospheric source of both total trace gases and gas-phase non-methane organic compounds (NMOC), and is the largest global atmospheric source of primary fine carbonaceous particles (Akagi et al., 2013; Yokelson et al., 2013). 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 US, accounting for 29% of total emissions compared to 9.2% from transportation sources (Aurell & 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 (Naeher et al., 2007). Wildland fire smoke could also contain exotic persistent organic compounds such as dioxins and furans (Black et al., 2011; Ward & Lincoln, 2006). 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 (Commodore et al., 2012; McMahon & Bush, 1992; Molto et al., 2010; Yoschenko et al., 2006). Since this review is health risk-driven, components with reported or estimable exposure levels and which are of concern based on comparisons with established exposure limits are the main focus of this section of the review. The exposure standards for these components are presented in Table 2, while their maximum reported study mean or individual time-weighted average (TWA) concentrations and hazard ratios based on the most stringent regulatory or recommended occupational or ambient air (acute or chronic) exposure limits are presented in Table 3. Comparisons with chronic exposure limits applicable to the general population are made with the consideration that episodic wildland fire smoke exposure is experienced rarely in most communities. In preparing Table 3, preference is given to components with fixed area ground or personal exposure measurement data. Components without such measurements but which may be of concern based on exposures estimated from emissions factor data are mentioned at the end of this section.

Based on the maximum reported mean or individual TWA fixed area ground or personal exposure measurements and relevant regulatory or recommended occupational or general

Table 2. Occupational and public health exposure limits for components of concern.

Components	Lowest occupational exposure limit[a]	Lowest short term occupational exposure limit[a]	Lowest general public daily exposure limit[a]	Lowest short term general public exposure limit[a]	Unit	Agency/organization issuing exposure limit (period or form of limit)[b,c]
Respirable particles ($PM_{3.5/4}$)	3000				$\mu g/m^3$	LOcEL – ACGIH
Fine particles ($PM_{2.5}$)			35		$\mu g/m^3$	LGPDEL – USEPA
Carbon monoxide	25	200	9	20.08	ppm	LOcEL – CalOSHA, ACGIH LSTOEL – CalOSHA, NIOSH (ceiling) LGPDEL – USEPA (8 h) LSTGPEL - CalOSHA (1 h)
Nitrogen dioxide	0.20	1.00		0.1	ppm	LOcEL – ACGIH LSTOEL – CalOSHA, NIOSH (STEL) LSTGPEL – USEPA (1 h)
Sulfur dioxide	2	0.25		0.075	ppm	LOcEL – CalOSHA, NIOSH LSTOEL – ACGIH (STEL) LSTGPEL – USEPA (1 h)
Ozone	0.1[d]	0.1	0.075	0.092	ppm	LOcEL – OSHA, CalOSHA LSTOEL – NIOSH ceiling LGPDEL – USEPA (8 h) LSTGPEL – CalEPA (1 h)
Acrolein	0.1	0.1	0.00015	0.001	ppm	LOcEL – OSHA, NIOSH LSTOEL – CalOSHA, ACGIH (ceiling) LGPDEL – USEPA (RfC – chronic inhalation) LSTGPEL – CalEPA (1-h)
Formaldehyde	0.016	0.1		0.045	ppm	LOcEL – NIOSH (as potential carcinogen) LSTOEL – NIOSH (ceiling) LSTGPEL – CalEPA (1 h)
Benzene	0.1	1	0.0028	0.0085	ppm	LOcEL – NIOSH (as potential carcinogen) LSTOEL – NIOSH (STEL) LGPDEL – CalEPA (reference exposure level) LSTGPEL – CalEPA (1 h)
Toluene	10	150	0.08	9.82	ppm	LOcEL – CalOSHA LSTOEL – CalOSHA NIOSH (STEL) LGPDEL – CalEPA (reference exposure level) LSTGPEL – CalEPA (1 h)
Xylene	100	150	0.16	5.07	Ppm	LOcEL – CalOSHA, NIOSH, ACGIH LSTOEL – CalOSHA, NIOSH, ACGIH (STEL) LGPDEL – CalEPA (reference exposure level) LSTGPEL – CalEPA (1 h)

[a]Both regulatory and recommended exposure limits are considered.
[b]LOcEL – lowest occupational exposure limit; LSTOEL – lowest short term occupational exposure limit; LGPDEL – lowest general public daily exposure limit; LSTGPEL – lowest short term general public exposure limit; OSHA – Occupational Safety and Health Administration; CalOSHA – California Occupational Safety and Health Administration; NIOSH – National Institute for Occupational Safety and Health; ACGIH – American Council of Governmental Industrial Hygienists; USEPA – United States Environmental Protection Agency; CalEPA – California Environmental Protection Agency; STEL – short term exposure limit (15 min); RfC – reference concentration.
[c]Limits are for 8-h and 24-h exposure for lowest occupational exposure limit (LOEL) and lowest general public daily exposure (LGPDEL) when periods are not specified.
[d]ACGIH OEL is as low as 0.05 and as high as 0.20 depending on workload and time.

population exposure limits for acute and chronic exposures, the components of most concern are respirable or fine particulate matter, acrolein, carbon monoxide, nitrogen dioxide, benzene and formaldehyde.

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 et al., 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). In addition, greater increases in concentrations of particles in the accumulation mode have been observed in studies of ambient air

Table 3. Hazard indices for components of concern based on occupational and general public relevant exposure limits.

Components	Type of study + description[a]	Maximum occupational TWA value reported	Maximum occupational short-term or instantaneous exposure reported	Unit	Hazard ratio (daily occupational 8 h)	Hazard ratio (occupational short-term)[b]
Occupational hazard indices based on occupational exposures or fireline measurements						
Respirable particles ($PM_{3.5/4}$) (Reinhardt & Ottmar, 2004)	Personal exposure at fireline	10 500		$\mu g/m^3$	3.50	
Carbon monoxide (Reinhardt & Ottmar, 2004; Reisen et al., 2011)	Personal measurements	58	1085[c]	ppm	2.32	5.43[d]
Nitrogen dioxide (Miranda et al., 2012)	Personal exposure	2.5	7.00[c]	ppm	12.5	1.40[d]
Formaldehyde (Reinhardt & Ottmar, 2004)	Personal measurements	0.6	1.46	ppm	38	14.6
Acrolein (De Vos et al., 2006; Reinhardt & Ottmar, 2004)	Personal exposure; measurement inside experimental firefighter mask	0.153	0.129	ppm	1.53	1.29[e]
Benzene (Reinhardt & Ottmar, 2004; Barboni et al., 2010)	Personal measurements	0.384	16.9[f]	ppm	3.84	16.9
Hazard indices based on exposures measured in areas remote from the fireline						
Components	Type of study + description[a]	Maximum general public TWA value reported	Maximum general public short-term or instantaneous exposure reported	Unit	Hazard ratio (Public daily)	Hazard ratio (public short-term)[g]
Fine particles ($PM_{2.5}$) (Wu et al., 2006)	Area measurements	90		$\mu g/m^3$	2.57	
Carbon monoxide (Tan et al., 2000)	Area measurements	17.6		ppm	1.95[h]	
Ozone (Smith et al., 1996; Tham et al., 2009)	Area measurements	0.09	0.12[i]	ppm	1.20[h]	

[a]Personal measurements are reported where available. Area measurements are given only when personal measurements are not available.
[b]Comparison is with STEL or ceiling values.
[c]Instantaneous peak measurement.
[d]Comparison is between instantaneous measurements and ceiling value.
[e]Comparison of the short-term exposure with lowest ceiling value; note that TWA is also higher than the short-term exposure.
[f]15-min averages.
[g]Comparison with limits for exposure for 1-hour period or less.
[h]Comparison is with USEPA 8-h exposure standard.
[i]Hourly averages.

during periods of wildland fire compared to periods without such events (Alonso-Blanco et al., 2012; Cashdollar et al., 1979; Portin et al., 2012; Sillanpää et al., 2005; Verma et al., 2009). Particle formation during combustion of vegetative biomass usually starts with the nucleation mode (aerodynamic diameter $< 0.1\,\mu m$) with condensation nuclei consisting of compounds such as PAHs or low volatility organic compounds (LVOCs) depending on fuel characteristics and combustion conditions (Chakrabarty et al., 2006). Submicrometer 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 et al., 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 et al., 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. 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 level of exposure (Cherrie et al., 2013). However, wildland fire smoke-derived particles contain water soluble components, and redox reactive metals and polar organic compounds (Alves et al., 2011; Balachandran et al., 2013; Lee et al., 2005b, 2008a; Leonard et al., 2000, 2007; Wegesser et al., 2010). It may also induce measurable acute pulmonary and systemic responses at lower exposure levels (Naeher et al., 2007).

Particulate matter emitted from the combustion of vegetative biomass is mostly carbonaceous and is typically composed of at least 50% organic carbon by weight

(Alves et al., 2010a,b; Chen et al., 2007a; Fine et al., 2001, 2002a,b, 2004a,b; Robinson et al., 2011; Schmidl et al., 2008). Elemental (the inorganic form of) carbon may constitute less than 10% of the particulate matter, but could sometimes be more substantial depending on the specie or type of vegetation (Alves et al., 2010a,b; Chen et al., 2007a; Fine et al., 2001, 2002a,b, 2004a,b; Robinson et al., 2011; Schmidl et al., 2008). Wildland fire smoke contains black carbon which is the strong light absorbing component of elemental carbon and is a climate forcing agent (Chen et al., 2007a; Ramanathan & Carmichael, 2008). Exposure to black carbon has also been associated with effects on cardiovascular and respiratory health (Jansen et al., 2005; Nichols et al., 2013).

Levoglucosan, which is a sugar anhydride and a pyrolytic product of cellulose, is the most abundant organic compound in wildland fire associated smoke particulate matter (Lee et al., 2005b). Other sugar anhydrides, aliphatic and oxygenated aliphatic hydrocarbons, sterols, methoxyphenols, which are pyrolytic products of lignin, PAHs and oxygenated PAHs, are also present (Fine et al., 2001, 2002ab, 2004a,b).

Although, the currently existing occupational standard for particulate matter may be inadequate for particles in wildland fire smoke as previously stated, ambient air concentration in the immediate vicinity of fires (12.5 mg/m^3) (Alves et al., 2010a,b) and personal wildland firefighter exposure (10.5 mg/m^3) (Reinhardt & Ottmar, 2004) that exceed the lowest occupational exposure limit (3 mg/m^3) recommended by the American Conference of Governmental Industrial Hygienists (ACGIH) have been reported. These levels also exceed the Occupational Safety and Health Administration's (OSHA) regulatory standard with a higher permissible exposure limit of 5 mg/m^3. These levels are of course well above the current 24-h National Ambient Air Quality Standard (NAAQS) for ambient air (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 (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 et al., 2013a; Dunn et al., 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 regulatory standard (permissible exposure limit – 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 due to the dilution of carbon monoxide in air during transport from the fire to public receptor locations.

The toxicity of carbon monoxide is partly due to its ability to bind hemoglobin more strongly than oxygen (~240 times) causing the formation of carboxyhemoglobin (COHb) (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 (Raub, 1999). Higher COHb concentrations could result in headache, dizziness, weakness, disorientation and impair decision making (Raub, 1999; Raub et al., 2000). The elimination half-life of COHb is 4–5 h without any intervention, and treatment of carbon monoxide poisoning involves speeding up the elimination rate (Annane et al., 2011; Guzman, 2012; Quinn et al., 2009; Wolf et al., 2008).

Although COHb levels measured in wildland firefighters are mostly below 5% (Dunn et al., 2009; Gaskill et al., 2010; Miranda et al., 2012), working in heavy smoke or for longer periods could contribute towards elevated COHb concentrations due to its potential to accumulate in the blood (Gaskill et al., 2010). Consequently, wildland firefighters and other persons potentially could experience elevated COHb levels when they are in close proximity to wildland fires. However, it should be noted that other sources of carbon monoxide such as pumps, generators and gasoline trucks could significantly contribute to the exposures of firefighters working at wildland fires (Gaskill et al., 2010).

Respiratory irritants: acrolein and formaldehyde

Both acrolein and formaldehyde are respiratory irritants at low concentrations. Exposure to these pollutants could result in respiratory symptoms, and nasal and respiratory tract irritation (Bein & Leikauf, 2011; Lang et al., 2008). Acrolein is a more potent irritant (Roemer et al., 1993), and exposure at higher concentrations could result in lung injury (Bein & Leikauf, 2011). Formaldehyde is also classified as a probable human carcinogen by the USEPA.

Olfactory detection of formaldehyde occurs between 0.04 and 0.40 ppm (Lang et al., 2008). Most of the published average occupational TWA exposures are below this range, and all maximum occupational TWA exposures reported in identified studies are below the OSHA PEL of 0.75 ppm (De Vos et al., 2009; Reinhardt & Ottmar, 2004; Reisen & Brown, 2009; Reisen et al., 2011). However, some of the average occupational TWA exposures reported for wildland firefighters in the US and Australia in these studies exceed the lowest OEL of 0.016 ppm (National Institute for Occupational Safety and Health [NIOSH] recommended exposure limit) which is based on the carcinogenic effect of formaldehyde. Exceedance of this OEL was by up to 3700% for the highest reported average TWA. The maximum short-term exposure in the literature (1.46 ppm) was reported among wildland firefighters at prescribed burns in the US (Reinhardt &

Ottmar, 2004). This is an order of magnitude higher than the NIOSH recommended ceiling of 0.1 ppm.

Both average and maximum occupational TWAs reported for acrolein in the literature were all below the lowest OEL of 0.10 ppm (OSHA) and the recommended ceiling value of 0.1 ppm (California OSHA and ACGIH) except for a maximum TWA of 0.15 ppm measured in the respirator of a wildland firefighter working at a bushfire in Australia (De Vos et al., 2006). This TWA was also higher than the maximum reported short-term exposure of 0.129 ppm observed among wildland firefighters conducting prescribed burns in the US (Reinhardt & Ottmar, 2004). Additive effects from multiple irritants should be considered, and risk assessment of occupational wildland firefighters to wildland fire smoke indicates that their concurrent exposures to particulate matter, acrolein and formaldehyde at wildland fires may be of concern (Reinhardt & Ottmar, 2004). It is possible that cancer risk from formaldehyde exposure may be slightly increased above the acceptable 10^{-5} level for occupational exposure when the average duration of exposure at wildland fires, the frequency of exposure and career length of the wildland firefighter are considered (Booze et al., 2004).

Benzene

Five studies of the assessment of benzene exposure due to wildland fire from ground measurements or personal monitoring were identified (Barboni et al., 2010; Barboni & Chiaramonti, 2010; Evtyugina et al., 2013; Reinhardt & Ottmar, 2004; Reisen & Brown, 2009). Although repeated exposure to low levels of benzene may result in adverse non-cancer hematological, neurological and immunological effects (Galbraith et al., 2010; Gist & Burg, 1997), the average TWA concentrations reported are well below estimated or measured levels for which these adverse effects were observed in various studies. However, a maximum individual TWA personal exposure of 0.384 ppm observed in one study was 3.84 times the NIOSH recommended OEL which is based on carcinogenic effects (Reinhardt & Ottmar, 2004). In addition, a maximum 15-min fixed area measurement of 16.9 ppm reported in a study in France was 16.9 times the NIOSH recommended short-term exposure limit (Barboni et al., 2010). However, it is worth noting that the 15-min measurements in the France study may not be representative of typical occupational exposures since the measurements were conducted by firefighting personnel in very close proximity (1–10 m) to the fire line. It is possible that cancer risk from benzene exposure may contribute to a total risk above the acceptable 10^{-5} level for occupational exposure when the average duration of exposure at wildland fires, the frequency of exposure and career length of the wildland firefighter are considered (Booze et al., 2004).

Nitrogen dioxide

Nitrogen dioxide (NO_2) induces various pulmonary responses including decrement in lung function, airway hyper-responsiveness and bronchoconstriction (WHO, 2006). In addition, ambient air concentration of NO_2 has been associated with respiratory and cardiovascular events as indicated by increases in mortality and physician or emergency room visits due to morbidity (Poloniecki et al., 1997; Samoli et al., 2006). In general, susceptible individuals with pre-existing diseases such as asthma and chronic obstructive disease are more vulnerable to exposure to NO_2 (WHO, 2006). These adverse responses seem to be solely dependent on concentration more than the duration or total dose of exposure (WHO, 2006). Therefore, the short-term exposure may be the more relevant metric for NO_2 during wildland fires. The maximum personal TWA exposure and the maximum peak area measurement reported for nitrogen dioxide suggest that it may be of concern during wildland fires (Miranda et al., 2012). The maximum personal TWA exposure reported for nitrogen dioxide (2.5 ppm) also exceeded the ACGIH and California OSHA recommended ceiling of 1 ppm for the pollutant.

Ozone and others

Ozone is a secondary air pollutant formed through a series of reactions involving the interaction of light and other air pollutants including nitrogen dioxides and volatile organic compounds. In addition to being present in background ambient air, some of these primary air pollutants, as stated earlier in this section, are emitted in wildland fire smoke (Evtyugina et al., 2013; Simpson et al., 2011). Consequently, ozone could be a pollution problem in areas downwind from wildland fires. Eight studies reporting on the concentration of ozone in ambient air impacted by wildland fire smoke were identified (Evans et al., 1977; Hu et al., 2008; Phuleria et al., 2005; Portin et al., 2012; Smith et al., 1996; Tan et al., 2000; Tham et al., 2009; Wang et al., 2012). The maximum mean fixed area 24-h (90 ppb) and 1-h (120 ppb) ambient air concentrations of ozone under such conditions exceeded the USEPA 8-h NAAQS of 75 ppb by 20 and 60%, respectively (Smith et al., 1996; Tham et al., 2009). The recommended NIOSH ceiling of 0.1 ppm was also exceeded by the maximum mean 1-h concentration by 20% (Smith et al., 1996). Elevated concentration of ozone in ambient air is associated with acute effects including decline in lung function, enhancement of airway responsiveness, autonomic cardiovascular effects and morbidity and mortality related especially to respiratory illnesses (WHO, 2006). Some of these effects are observed in association with ambient air concentrations below the maximum concentrations referenced above (WHO, 2006).

Concentrations of 1,3-butadiene and hydrogen cyanide estimated from available emissions factor data and compared to USEPA reference concentrations for chronic inhalation exposure suggest that both could be pollutants of concern for the general public if exposure is experienced a few times a year (Burling et al., 2010; Urbanski, 2014; Yokelson et al., 2013).

The health impact 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 studies of related ambient or household air pollution are also discussed.

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. It should be noted that the burning of agricultural residues or fields was the source of exposure in eight of the studies that were identified.

Acute cardiovascular and/or respiratory impacts with lagged effects mostly restricted to within 6 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 4.

Respiratory effects of wildland fire smoke in the general public

Naeher et al. (2007), in a major comprehensive review of the health effects of vegetative biomass smoke, concluded that exposure to smoke from wildland fires or burning of agricultural fields/residues resulted in respiratory symptoms and illnesses. They noted that the results were consistent across studies in different locations except for those that were conducted in Australia. Studies that have been published since the Naeher et al. comprehensive review in 2007, including six that were conducted in Australia, have reported results positive for the respiratory effects of wildland fire smoke exposure in the general public (Analitis et al., 2011; Crabbe, 2012; Delfino et al., 2008; Epton et al., 2008; Hanigan et al., 2008; Henderson et al., 2011; Johnston et al., 2007; Martin et al., 2013; Mirabelli et al., 2009; Morgan et al., 2010; Rappold et al., 2011; Tham et al., 2009).

Occurrence of wildfires (forest fires) in Athens, Greece, was associated with increases in mortality due to respiratory illnesses (Analitis et al., 2011). An apparent dose–response relationship was observed with more deaths occurring with increasing size of the forest area burned. While small fires (defined as fires burning 10 000–1 000 000 m^2) were not associated with increases in respiratory mortality, medium (defined as fires burning 1 000 000–30 000 000 m^2) and large fires (defined as fires burning >30 000 000 m^2) were associated with 16.2% (95% CLs: 1.3, 33.4%) and 92.0% (47.5, 150.0%) increases in respiratory mortality respectively. Sastry (2002) had similarly observed a significant doubling of respiratory mortality in Kuching, Malaysia, in association with reduced visibility (<0.91 km), which was used as a surrogate of ambient air pollution, during the 1997 Southeast Asia forest fire-related haze episode. An insignificant increase was observed in Kuala Lumpur, Malaysia during the same period (Sastry, 2002). Increase in respiratory mortality was not observed in association with ambient air PM_{10} concentrations on high pollution days that were heavily influenced by bushfires in Sydney, Australia (Morgan et al., 2010).

The adverse health effects of wildland fire smoke exposure in the general public have been most consistently observed as increases in hospital admissions, physician or emergency room visits due to respiratory illnesses. Effects of forest fires were observed among a cohort of registered individuals on the Medical Service Plan in the province of British Columbia, Canada (Henderson et al., 2011). While no relationship was observed between either physician visits or hospital admissions due to all respiratory diseases and the occurrence of fire on the same day, significant increases were observed for both effects (odds ratio [OR]: 1.02 and 1.05, respectively) in association with an increase of 10 $\mu g/m^3$ in the same-day 24-h average ambient concentration of PM_{10} recorded at fixed monitoring station during the forest fire season. Significant increases in hospital admissions due to all respiratory diseases (OR: 1.11) were also observed in association with an increase of 60 $\mu g/m^3$ in the same-day 24-h average ambient concentration of PM_{10} derived from the integration of satellite data with the CALPUFF smoke dispersion model in the same study (Henderson et al., 2011).

In a study of the effects of the 2003 southern California wildfires, the 10-day wildfire period seemed to be protective against hospital admissions for all respiratory diseases (relative risk [RR]: 0.903) when compared to the 3-week period before the wildfires in a model adjusting for the concentration of ambient air $PM_{2.5}$ (Delfino et al., 2008). Conversely, the risk for hospital admissions increased during the 2-week period immediately following the wildfires (RR: 1.173) indicating some delayed effects, although, the authors could not rule out a seasonal effect for the post-wildfire results (Delfino et al., 2008). It should be noted that results were in similar directions when the same comparisons were made in a model without adjustment for $PM_{2.5}$, but the difference in the risk of hospital admission for all respiratory diseases between the wildfire and pre-wildfire periods was not significant in this model. In addition to testing the effect of the period (relative to the wildfire) on hospital admissions, the authors tested the effect of the interaction term between the period and ambient air $PM_{2.5}$ concentration. Although, the positive exposure ($PM_{2.5}$)–response relationship was not statistically different between the periods, it was stronger

Table 4. Summary of review of epidemiological studies of the general public.

Citation (Author.Year.Journal)	Location	Biomass type	Study design	Subject description	Sample size	Exposure measure	Health end-point(s)	Major confounders (apart from adjustment for trend and seasonal components in trend analyses)	Result summary
Holstius et al. (2012). Environ Health Perspect	Southern California, USA	Forest fire	Cohort	Singleton births	886 034 (747 590 births from pregnancy before or after wildfire events, 60 270 births from pregnancy with wildfire occurs during the first trimester, 39 435 – second trimester, 38 739 – third trimester)	Exposure versus non-exposure periods	Birth weight	Age, educational attainment, parity, race/ethnicity, infant's gender, gestational age	Mean birth weight was 7.0 g lower [95% CI: −11.8, −2.2] when the wildfire occurred during the third trimester, 9.7 g lower when it occurred during the second trimester (95% CI: −14.5, −4.8), and 3.3 g lower when it occurred during the first trimester (95% CI: −7.2, 0.6) compared with pregnancies before and after the wildfires
Torigoe et al. (2000). Pediatr Int	Niigata, Japan	Agricultural fire – rice straw	Cohort	Boys and girls with the average age of 7.3 years	438 boys and 262 girls	Burn season versus non-burn season (PM_{10} also measured but not used for associations with health end-points)	Asthma attack	The weather and pressure patterns, temperature, wind direction and velocity, daylight hours and precipitation	There was a significant increase in the number of children with asthma attack visiting emergency room among the exposed group ($p < 0.001$)
Jalaludin et al. (2000). Aust NZ J Public Health	Sydney, Australia	Bush fire	Cohort	Children with a history of wheeze	32	PM_{10} (O_3, NO_2 also measured but not used for associations with health end points)	Lung function (peak expiratory flow rate)	Asthma medications, time trend, mean temperature, mean humidity, number of hours spent outdoors, total pollen and alternaria counts	Overall, no significant association was observed between mean PM_{10} and peak expiratory flow rate ($\rho \not\leftarrow = -0.009$, $p = \not\leftarrow 0.86$). However, significant negative association between peak expiratory flow rate and PM_{10} $\rho \not\leftarrow = \not\leftarrow -0.1029$, $p = 0.03$) was observed among children without bronchial hyperreactivity. Bush fire period was not significantly associated with peak expiratory flow rate
Henderson et al. (2011). Environ Health Perspec	British Columbia, Canada	Forest fire	Cohort	Residents in the study area whose addresses were precisely geocoded	281,711	PM_{10}	Respiratory and cardiovascular (physician visit and hospital admission)	Effect modification by age, sex, and SES were examined	There were increases in odds for a 30 $\mu g/m^3$ increase in TEOM-based PM_{10} were for all respiratory physician visits – 1.05 (95% CI, 1.03–1.06); for asthma-specific visits – 1.16 (95% CI, 1.09–1.23); and for

(*continued*)

Table 4. Continued

Citation (Author.Year.Journal)	Location	Biomass type	Study design	Subject description	Sample size	Exposure measure	Health end-point(s)	Major confounders (apart from adjustment for trend and seasonal components in trend analyses)	Result summary
									respiratory hospital admissions – 1.15 (95% CI, 1.00–1.29). There were no associations with cardiovascular outcomes among the whole population in the study
Mott et al. (2002). West J Med	Hoopa, California, USA	Forest fire	Retrospective cohort	American Indians (92 had pre-existing cardiopulmonary conditions)	289	Exposure versus non-exposure periods	Respiratory illnesses (medical visits)	None listed or considered in analysis	Increase in medical visits for respiratory illnesses from 417 to 634 visits (52%) was observed during the weeks of the forest fire compared to the previous year, and those with preexisting cardiopulmonary conditions reported more symptoms before, during and after the smoke episode
Johnston et al. (2006). Int J Environ Health Res	Darwin, Australia	Bush fire	Cohort (panel study)	Adult and children	251	PM_{10} and $PM_{2.5}$	Acute respiratory symptoms on individuals with asthma	Minimum daily air temperature, relative humidity, pollen and spore counts, the weekly rate of consultations to general practitioners for influenza-like illness, temporal autocorrelation of outcomes, weekends and holiday periods	A rise of 10 $\mu g/m^3$ in PM10 ranged was significantly associated with onset of asthma symptoms (OR =←1.240, CI 1.106–1.39), commencing oral steroid medication (OR =← 1.540, CI 1.01–2.34), the mean daily symptom count (OR =← 1.020, CI 1.001–1.031) and the mean daily dose of reliever medication (OR =← 1.020, CI 1.00–1.030) and similar results were observed for $PM_{2.5}$.More severe outcomes of asthma attacks, increased health care attendances or missed school/work days, were not associated with exposure measures.

Sutherland et al. (2005). J Allerg Clin Immunol	Denver, Colorado, USA	Forest fire	Cohort (panel study)	Denver residents with COPD	21	Spike days indicated by elevated concentrations of $PM_{2.5}$ (PM_{10} and CO were also measured)	Respiratory symptoms in adult subjects with COPD	None listed or considered in analysis	Symptom scores were significantly elevated on spike days versus non-spike days ($p =$ .0002). The symptom score on spike days was 21.5 (IQR = 3.0), and on non-spike days was 20.0 (IQR = 4.0)
Kunzli et al. (2006). Am J Respir Crit Care Med	Southern California, USA	Forest fire	Cross-sectional	High-school students	6424 ($n =$ 873; 17–18 years old and $n =$ 5551; age, 6–7 years old elementary-school children)	Number of days of fire smoke smell indoors	Respiratory symptoms	Sex, ethnicity, educational level of parents, asthma status before the fire (physician-diagnosed asthma) and cohort (high-school versus elementary-school cohort)	Various symptoms were 2–5.5 times more likely to occur among those with 6 or more days of fire smell indoors: general, sleep-disturbing and speech-limiting wheezing occurred 3.5, 4.9 and 5.5 times more often respectively among those with 6 or more days of fire smell indoors compared with those reporting no day of fire smoke smell indoors. Asthma attacks also significantly increased 63% among this group. The trend across the different levels of fire smell duration (0, 1–5 and >5 days) was highly significant for all outcomes except for asthma attacks ($p =$ 0.12). There was significant difference between the communities with the highest and lowest PM_{10} levels (210 and 30 µg/m^3, respectively) for dry coughs at various times during the day, irritated eyes, watery/itchy eyes and sneezing/runny/blocked nose.
Long et al. (1998). Chest	Winnipeg, Canada	Agricultural residue	Cross-sectional	Adults (35 to 64 years old)	428	Exposure versus non-exposure periods (PM_{10}, CO, NO_2, and VOC were used but not measured for associations with health endpoints)	Respiratory symptoms	Age, gender, smoking status	Although 37% of subjects reported that they were not bothered by smoke at all, 42% of subjects reported that at least one symptom (cough, wheezing, chest tightness, shortness of breath) developed or became

(*continued*)

Table 4. Continued

Citation (Author.Year.Journal)	Location	Biomass type	Study design	Subject description	Sample size	Exposure measure	Health end-point(s)	Major confounders (apart from adjustment for trend and seasonal components in trend analyses)	Result summary
									worse due to the air pollution episode and 20% reported that they had breathing trouble: females compared to males and ex-smokers compared to smokers were more likely to be affected and subjects with asthma and chronic bronchitis were also more likely to be affected
Analitis et al. (2011). Occup Environ Med	Athens, Greece	Forest fire	Ecological time series	General population	Population size was not mentioned	Exposure versus non-exposure and size of burns	Non-accidental mortality including those due to respiratory and cardiovascular illnesses	Time trend and meteorological variables	No mortality effects were observed for small fires (10 000–1 000 000 m^2). Medium sized fires (1 000 000–30 000 000 m^2) were associated with an increase of 4.9% (95% CI 0.3–9.6%) in the daily mortality, 6.0% (95% CI 0.3–12.6%) in cardiovascular mortality and 16.2% (95% CI 1.3–33.4%) in respiratory mortality. Cardiovascular effects are larger in those aged <75 years, while respiratory effects are larger in older people. Large fires (>30 000 000 m^2) were associated with: 49.7% (95% CI 37.2–63.4%) increase in daily mortality, 60.6% (95% CI 43.1–80.3%) in cardiovascular mortality and 92.0% (95% CI 47.5–150.0%) in respiratory mortality. Respiratory and not cardiovascular mortality were more pronounced in persons >75 years old.

Jacobs et al. (1997). Environ Health Perspec	Butte County, California, USA	Agricultural residue – rice straw	Ecological time series	General population	Population size was not mentioned	Burn acreage	Asthma hospitalization	Maximum daily temperature, seasonal factors, and yearly population	Burn acreage showed a small but statistically significant elevation of risk for hospitalization per acre of rice burned (RR = 1.0001; 95% CI, 1.00004-1.0002), after adjusting for confounders. An apparent dose-response was observed with days with the greatest acreage burned (>499 acre) having the largest risk of hospitalization (RR = 11.23; CI, 1.09-1.39), while days with moderate burning (between 100 and 499 acre) had a slightly lower risk of admission (RR = 1.2; CI, 1.05-1.37).
Johnston et al. (2002). Med J Aust	Darwin, Australia	Bush fire	Ecological time series	General population	Population size was not mentioned	PM_{10}	Asthma presentations (ER visits)	Weekly rates of influenza, day of week (weekend or weekday)	A 10 μg/m^3 increase in PM_{10} was associated with increase in asthma presentations even after adjusting for confounders (adjusted rate ratio, 1.20; 95% CI, 1.09–1.34; $p < 0.001$). The strongest effect was seen on days when the PM_{10} was above 40 μg/m^3 (adjusted rate ratio, 2.39; 95% CI, 1.46–3.90), compared with days when PM_{10} levels were less than 10 μg/m^3
Moore et al. (2006). Can J Public Health	Kelowna and Kamloops, British Columbia, Canada	Forest fire	Ecological time series	General population	Kelowna – average of 146 199 and Kamloops – average of 100 548 during study period	Exposure period versus non-exposure period ($PM_{2.5}$ and PM_{10} were also measured but not used for associations with health end-point)	Respiratory and cardiovascular (physician visit)	None listed and not evident that any were considered in analysis	In the Kelowna region PM level was more elevated, increases in physician visits due to respiratory illnesses of between 46 and 78% above 10-year mean rates were observed for three weeks during the forest fire period but similar effects were not observed in Kamloops. Effects on visits for cardiovascular diseases or mental disorders were not seen in either of the two communities.

(*continued*)

Table 4. Continued

Citation (Author.Year.Journal)	Location	Biomass type	Study design	Subject description	Sample size	Exposure measure	Health end-point(s)	Major confounders (apart from adjustment for trend and seasonal components in trend analyses)	Result summary
Rappold et al. (2011). Environ Health Perspect	Eastern North Carolina, USA	Peat forest fire	Ecological time series	General population	Population size was not mentioned	Exposed versus unexposed counties (aerial optical depth as a proxy for smoke pollution was used to determine exposed counties)	Respiratory and cardiovascular (ER visits)	None listed and not evident that any were considered in analysis	Significant increases in cumulative RR for ER visits for asthma [1.65 (95% confidence interval, 1.25–2.1)], chronic obstructive pulmonary disease [1.73 (1.06–2.83)] and pneumonia and acute bronchitis [1.59 (1.07–2.34)] were observed. ER visits associated with cardiopulmonary symptoms [1.23 (1.06–1.43)] and heart failure [1.37 (1.01–1.85)] were also significantly increased in exposed counties.
Vedal & Dutton (2006). Environ Res	Denver, Colorado, USA	Forest fire	Ecological time series	General population	>2 000 000	Exposure versus no exposure periods (PM_{10} and $PM_{2.5}$ were measured but not used for associations with health end-points)	Mortality from respiratory and cardiovascular illnesses	None listed or considered in analysis	Abrupt PM peaks associated with fires were not associated with differential increase in cardiorespiratory mortality between exposed and control counties.
Hanigan et al. (2008). Environ Health	Darwin, Australia	Bush fire	Ecological time series	General population	Population size was not mentioned	PM_{10}	Respiratory and cardiovascular illnesses (hospital admission)	Daily temperature and humidity	An increase of 10 $\mu g/m^3$ in same-day estimated ambient PM_{10} was associated with a non-significant 4.81% (95%CI: −1.04%, 11.01%) increase in total respiratory admissions, but a statistically different association was found between PM_{10} and admissions three days later for respiratory infections of indigenous people (15.02%; 95%CI: 3.73%, 27.54%) than for non-indigenous people (0.67%; 95%CI: −7.55%, 9.61%) (3). Null associations were observed for cardiovascular effects with ambient PM_{10} having positive (non-

									significant) associations with cardiovascular admissions of indigenous people two and three days later.
Morgan et al. (2010). Epidemiology	Sydney, Australia	Bush fire	Ecological time series	General population	Population size was not mentioned	PM_{10}	Respiratory and cardiovascular illnesses (hospital admission and mortality)	Temperature, humidity, day of week, flu epidemic	A 10 μg/m^3 increase in bushfire PM_{10} was associated with same day increase of 1.24% (95% CI: 0.22–2.27%) in all respiratory disease admissions, a 3.80% (1.40–6.26%) increase in chronic obstructive pulmonary disease admissions 2 days later, and a same day 5.02% (1.77–8.37%) increase in adult asthma admissions. Bushfire PM_{10} was associated with a large increase in same day admissions for adult asthma (5.02, 1.77–8.37) and a moderate decrease in childhood asthma admissions 3 days later (3.10, 6.18–0.07) . Bushfire PM_{10} was not associated with all cardiovascular disease admissions, cardiac admissions or ischemic heart disease admissions.
Tham et al. (2009). Respirology	Victoria, Australia	Bush fire	Ecological time series	General population	Population size was not mentioned	PM_{10} and O_3	Respiratory illnesses (ER visits)	Day-of-the-week and trend effects (meteorological data)	After adjusting for confounders, the strongest associations were observed between PM_{10} and daily respiratory emergency department attendances in Melbourne (RR =← 1.018, 95% CI: 1.004–1.033, p =← 0.01) (3). Non-significant positive associations were observed for all respiratory outcomes with API and ozone after adjustment.

(*continued*)

Table 4. Continued

Citation (Author.Year.Journal)	Location	Biomass type	Study design	Subject description	Sample size	Exposure measure	Health end-point(s)	Major confounders (apart from adjustment for trend and seasonal components in trend analyses)	Result summary
Chen et al. (2006). Int J Environ Health Res	Brisbane, Australia	Bush fire	Ecological time series	General population	Population size was not mentioned	PM_{10}	Respiratory illnesses (hospital admissions)	Daily average maximum and minimum temperatures, relative humidity (at 9 am), rainfall and wind direction (at 3 pm), seasonality, day of the week, holidays, long-term trends and influenza	The relative risk for respiratory hospital admissions increased by 9 and 11% for the medium level of ambient PM_{10} concentration (15–20 μg/m^3) and by 19 and 13% for higher level of ambient PM_{10} concentration (>20 μg/m^3) during bushfire and non-bushfire periods, respectively compared with admissions when there was lower level of PM_{10} (<15 μg/m^3).
Mott et al. (2005). Int J Hyg Environ Health	Kuching, Malaysia	Forest fire	Ecological time series	General population	Population size was not mentioned	Exposure versus no exposure	Respiratory and cardiovascular illnesses (hospital admission)	None listed or considered in analysis	Statistically significant fire-related increases were observed for respiratory hospitalizations across all ages (more so for 40–64 year old adults), specifically related to COPD and asthma; no associations were observed for cardio-respiratory or circulatory diseases. The survival analyses indicated that persons over age 65 years with previous hospital admissions for any cause, any cardiorespiratory disease, any respiratory disease or COPD, were significantly more likely to be rehospitalized during the follow-up period in 1997 than during the follow-up periods in the pre-fire years of 1995 or 1996
Arbex et al. (2010). J Epidemiol Community Health	Araraquara, Brazil	Agricultural burn – sugar cane plantations	Ecological time series	General population	200 000 inhabitants	TSP	Hypertension (hospital admission)	Temperature, day of the week, humidity	Increase in TSP concentration was associated with a low but statistically significant increase in the number of

									hypertension-related hospital admissions (0.233, $p < 0.001$) with association being significant only during burn period. Increase in hypertension hospital admission associated with a 10 μg/m^3 rise in 3-day average TSP lagged 1 day was 30% higher during burn periods compared to non-burn periods
Arbex et al. (2007). J Epidemiol Community Health	Araraquara, Brazil	Agricultural burn – sugar cane plantations	Ecological time series	General population	192 000 inhabitants	TSP	Asthma (hospital admission)	Temperature, day of the week, humidity	A 10 μg/m^3 increase in 5-day moving average TSP was associated with an increase of 11.6% (95% CI 5.4–17.7) in asthma hospital admission. No threshold effect seemed apparent.
Crabbe. (2012). Environ Geochem Health	Darwin, Australia	Bush fire	Ecological time series	General population	Population size was not mentioned	PM10, coarse particulate matter, fine particulate matter, carbon black	Respiratory and cardiovascular illnesses (hospital admission)	Meteorological data, holiday, day of week, periods of influenza epidemics	Ambient fine particulate matter, black carbon and PM_{10} concentrations lagged 1 day were associated with cardiovascular hospitalization, while ambient fine particulate matter, black carbon lagged 1 day were associated with respiratory hospitalization.
Johnston et al. (2007). BMC Public Health	Darwin, Australia	Bush fire	Case-crossover	General population	110 000 total residents; 11 500 indigenous persons	PM_{10}	Respiratory and cardiovascular illnesses (hospital admission)	Weekly influenza rate, days with rainfall >5 mm, same day mean temperature and humidity, the mean temperature and humidity of the previous 3 days and public holidays	There was a positive relationship between PM_{10} and admissions for all respiratory conditions (OR 1.08 95%CI 0.98–1.18) with a larger magnitude in the Indigenous subpopulation (OR1.17 95% CI 0.98–1.40). There was a positive association with ischemic heart disease in Indigenous people, greatest at a lag of 3 days (OR 1.71 95%CI 1.14–2.55), but no relationship between PM10 and

(*continued*)

Table 4. Continued

Citation (Author.Year.Journal)	Location	Biomass type	Study design	Subject description	Sample size	Exposure measure	Health end-point(s)	Major confounders (apart from adjustment for trend and seasonal components in trend analyses)	Result summary
									cardiovascular admissions overall.
Martin et al. (2013). Aust NZ J Public Health	Sydney, Newcastle and Wollongong, Australia	Bush fire	Ecological time series	General population	Sydney (3 862 000 residents), Newcastle (406 000 residents); Wollongong (278 000 residents)	Exposure versus no exposure periods (PM_{10}, $PM_{2.5}$ were measured but not used for associations with health end-points	Respiratory and cardiovascular illnesses (hospital admission)	None listed or considered in analysis	Bush fire events in Sydney were associated with a 6% (OR =↓1.06, 95%CI =← 1.02–1.09) same day increase in respiratory hospital admissions, same day increase of 13% (OR =↓1.13, 95%CI =← 1.05–1.22) in chronic obstructive pulmonary disease admissions and 12% (OR =← 1.12, 95%CI =↓1.05–1.19) in asthma admissions. Events were also associated with increased admissions for respiratory conditions in Newcastle and Wollongong. There were no associations with cardiovascular illnesses.
Sastry (2002). Demography	Malaysia	Forest fire	Ecological time series	General population	Population size was not mentioned	Exposure versus non exposure periods (determined by dichotomized variables - PM_{10} and visibility)	Mortality from respiratory and cardiovascular illnesses	weather factors and seasonal terms	In Kuala Lumpur, null associations were observed for all types of mortality; but significant relative risks of 1.218, 1.225 and 1.208 for total, nontraumatic and other mortality were observed with respect to visibility measure, while association was significant for total, nontraumatic and cardiovascular mortality among 65-74 year olds; In Kuching: significant relative risk for of 2.049 was observed only for respiratory mortality in overall population, but significant associations with total, nontraumatic and cardiovascular mortality was

									observed among >75 year olds
Churches & Corbett. (1991). NSW Public Health Bull	Australia	Bush fire	Ecological time series	General population	Population size was not mentioned	Exposure versus non-exposure periods (PM, CO and other pollutants measured but not used in analyses)	Asthma (hospital admission)	None listed or considered in analysis	There were no differences between asthma hospital attendances between days when there were bushfires and on days when there were none
Duclos et al. (1990). Arch Environ Health	USA	Forest fire	Ecological time series	General population	Population size was not mentioned	Exposure versus non-exposure periods	Respiratory illnesses (ER visits)	None listed or considered in analysis	Wildfire event was associated with observed/expected ratio of 1.4 and 1.3 for emergency room visits for asthma and COPD, respectively. There was also an increase in emergency room visit for laryngitis and sinusitis while no association was observed for pneumonia, pharyngitis and coronary problems
Emmanuel (2000). Respirology	Singapore	Forest fire	Ecological time series	General population	Population size was not mentioned	PM_{10} (other measures recorded including CO, NO_2, O_3, SO_2 and pollutant standard index but not used)	Respiratory illnesses (Hospital admissions and ER visit)	Weather factors	An increase in PM concentration from 50 to 150 $\mu g/m^3$ was associated with a 12, 19 and 26% increase in upper respiratory tract illnesses, asthma and rhinitis. There was no increase in hospital admissions and mortality.
Smith et al. (1996). Int J Epidemiol	Australia	Bush fire	Ecological time series	General population	Population size was not mentioned	PM_{10}, NO_2, O_3	Asthma (ER visit)	Weather factors	There was no relationship between PM_{10}, NO_2 and O_3 concentrations and asthma emergency room visits.
Delfino et al. (2008). Occup Environ Med	USA	Forest fire	Ecological time series	General population	Population size was not mentioned	$PM_{2.5}$	Respiratory and cardiovascular illnesses (hospital admissions)	Weather factors, fungal spores	There were positive associations between 10 $\mu g/m^3$ rise in $PM_{2.5}$ and hospital admission for respiratory illnesses (all, asthma, pneumonia, COPD) during the wildfire event. The increase per 10 $\mu g/m^3$ rise in $PM_{2.5}$ was stronger during wildfire event compared to before except for pneumonia with the difference for asthma being marginally significant. There were

(*continued*)

Table 4. Continued

Citation (Author.Year.Journal)	Location	Biomass type	Study design	Subject description	Sample size	Exposure measure	Health end-point(s)	Major confounders (apart from adjustment for trend and seasonal components in trend analyses)	Result summary
									increases in admissions for various cardiovascular illnesses in association for the same rise in PM_{10} (all, ischemic heart disease, congestive heart failure, cerebrovascular disease + stroke) during the wildfire event but none of these was significant.
Golshan et al. (2002). Int J Environ Health Res	Iran	Agricultural burn – rice straw	Cohort study	1–80 years old male and female	994 in first phase (questionnaire) – dropped to 134 in clinical phase	Exposure versus no exposure period	Lung function (spirometry measures)	Previous respiratory illness history, confounding exposure (smoking)	Percent predicted FEV_1, FEV1/FVC, PEFR, $FEF_{25–75}$ decreased after exposure to rice burning smoke while recent asthma attacks, using asthma medication, sleep disturbed by dyspnea, cough and exercise induced cough increased.
van Eeden et al. (2001). Am J Respir Crit Care Med	Singapore	Forest fire	Cohort (panel study)	Healthy young male	30	Exposure versus no exposure period	Acute systemic inflammation	None listed or considered in analysis	Concentrations of circulating cytokines (IL-1β, IL-6, GM-CSF) reduced from during the wildfire event to the period after the wildfire event.
Tan et al. (2000). Am J Respir Crit Care Med	Singapore	Forest fire	Cohort (panel study)	Healthy young male	30	Exposure versus no exposure period ($PM_{2.5}$, SO_2 measurements were collected but not used for association with health end-points)	Acute systemic inflammation	None listed or considered in analysis	Percentage of white blood cells that was polymorphonuclear leukocytes reduced from during the wildfire event to the period after the wildfire event and was associated with PM10 with no lag days and SO_2 with 3 lag days. There was no change in lung function as measured by FEV_1 and FVC or in the number of different white blood cell types.
Golshan et al. (2002). Int J Environ Health Res	Iran	Rice (agricultural burning) – whole vegetation	Cohort study	1–80 years old male and female	994 in first phase (questionnaire) – dropped to 134 in clinical phase	Rice straw burning smoke and PM_{10}	Lung function measured as spirometry; respiratory symptoms	Previous respiratory illness history, confounding exposure (smoking)	Percent predicted FEV_1, FEV_1/FVC, PEFR, $FEF_{25–75}$ decreased after exposure to rice burning smoke while

									recent asthma attacks, using asthma medication, sleep disturbed by dyspnea, cough and exercise induced cough increased.
Hanninen et al. (2009). J Exp Sci Environ Epidemiol	Finland	Wildfire – whole vegetation (long range transport)	Ecological time series	General population	3.4 million	$PM_{2.5}$	Daily all-cause mortality	Temperature, weekday	Non-significant 0.8–2.1% increase in daily all-cause mortality for exposure lagged 0–4 days and 5-day average exposure for every 10 $\mu g/m^3$ increase in $PM_{2.5}$
Cancado et al. (2006). Environ Health Perspect	Brazil	Sugar cane field burn	Ecological time series	General population	320 000	$PM_{2.5}/PM_{10}$	Respiratory hospital admissions	Relative humidity, temperature, weekday	Increases of 10.2 $\mu g/m^3$ in $PM_{2.5}$ and 42.9 $\mu g/m^3$ in PM_{10} were associated with 21.4% (4.3, 38.5%) and 31.03% (1.25, 60.21%) increases in child and elderly respiratory hospital admissions respectively. Associations were more positive on sugar cane burn days.

during the wildfire period. An increase of 2.8% in hospital admissions for all respiratory diseases was observed in association with an increase of 10 $\mu g/m^3$ in the 2-day average ambient air $PM_{2.5}$ concentrations during the wildfire period (Delfino et al., 2008). While the authors acknowledged the inconsistencies in the results obtained from the different models without providing an explanation of probable causes for them, the significant increase in hospital admission in the period immediately after the wildfire event, and the positive relationship between ambient air $PM_{2.5}$ concentration and hospital admissions during the wildfire period suggest a respiratory effect of wildfire smoke exposure.

Similarly, an increased risk for emergency room visits for all respiratory diseases (RR: 1.66) was observed in North Carolina counties that were determined to have been exposed to smoke from peat forest fires between 1 June 2008 and 14 July 2008 based on a satellite platform derived aerial optical density measure (Rappold et al., 2011). In addition, significant associations, which were stronger during the sugar cane field burning season, were observed between ambient air PM_{10} concentrations and hospital admissions for both children (>13 years) and the elderly (>64 years) in a city in Brazil (Cancado et al., 2006). The results referenced above for all respiratory diseases are supported by those reported in prior studies (those included in the Naeher et al. review) conducted in countries other than Australia (Chen et al., 2006; Moore et al., 2006; Mott et al., 2002, 2005). They are also supported by results reported in other more recent studies (published after the 2007 review by Naeher et al.) that were conducted in Australia with regards to bushfire smoke exposure (Crabbe, 2012; Hanigan et al., 2008; Johnston et al., 2007; Morgan et al., 2010; Tham et al., 2009). Although non-significant overall increases in risk were observed in two of the more recent Australian studies (a case-crossover study and an ecological time-series study) (Hanigan et al., 2008; Johnston et al., 2007), a significant increase was observed for indigenous people in one of these studies (Hanigan et al., 2008).

These two Australian studies also did not observe increases in hospital admissions for asthma in association with bushfire related air pollution (Hanigan et al., 2008; Johnston et al., 2007). However, significant increase in the odds (same day OR: 1.12) of having hospital admission for asthma was associated with the occurrence of bushfire related high pollution in Sydney, Australia (Martin et al., 2013). No association was found for asthma hospital admissions in two other Australian cities, Newcastle and Wollongong, in the same study. Associations of asthma hospital admissions or emergency room visits with various measures of particulate matter air pollution were observed with respect to peat forest fires in North Carolina (65% increase in emergency room visits in exposed counties) (Rappold et al., 2011), bushfire in Australia (5.02% increase in hospital admissions per same day 10 $\mu g/m^3$ rise in PM_{10} in 15–64 year old) (Morgan et al., 2010), forest fires in British Columbia (16% increase in odds of hospital admissions per 30 $\mu g/m^3$ rise in same-day PM_{10} during the forest fire season) (Henderson et al., 2011) and forest fires in California (4.8% increase in hospital admission per 10 $\mu g/m^3$ rise in 2-day average $PM_{2.5}$ during wildfire period) (Delfino et al., 2008). Results for asthma hospital

admissions from prior studies (those included in the Naeher et al. review) are more inconsistent. Significant positive associations were observed in three of the prior studies, including two which focused on burning of agricultural fields or residues (Arbex et al., 2007; Jacobs et al., 1997; Mott et al., 2005), while null results were reported in two studies (Churches & Corbett, 1991; Duclos et al., 1990). Weather factors, which are commonly controlled for in ecological time series studies were not adjusted for in these two prior studies that reported the null findings for asthma hospital admissions. One prior study reported null findings for asthma related physician visits (Johnston et al., 2006). Three prior studies reported significant positive associations for asthma emergency room visits (Duclos et al., 1990; Emmanuel, 2000; Johnston et al., 2002), while one prior study reported a null finding (Smith et al., 1996). Two prior studies (one, cohort and the other, a cross-sectional study) also observed associations with various asthma-related symptoms and wellness measures in relation to wildfire events (Johnston et al., 2006; Kunzli et al., 2006).

Wildland fire related air pollution exposure has also been associated with hospital admissions for COPD among the general public in two more recent ecological time-series studies (published after the 2007 review by Naeher et al.) (Delfino et al., 2008; Morgan et al., 2010). A bushfire related $10\,\mu g/m^3$ rise in ambient air PM_{10} was associated with an increase of 3.29% in hospital admissions for COPD among persons older than 65 years in Sydney, Australia. This was six times higher than the insignificant 0.57% increase in hospital admissions for COPD that was observed for a $10\,\mu g/m^3$ rise in PM_{10} in background ambient air in the same study (Morgan et al., 2010). Delfino et al. (2008) also reported a 3.8% increase in COPD hospital admissions among persons between 20 and 99 years old for every $10\,\mu g/m^3$ rise in $PM_{2.5}$ during the 2003 southern California wildfires. This association was stronger than those observed in the periods immediately preceding or following the wildfires. In a case-crossover study, the odds of subjects being admitted into the hospital for COPD during the bushfire season in Darwin, Australia increased by 21% for every $10\,\mu g/m^3$ rise in PM_{10} (Johnston et al., 2007). In another ecological time-series study conducted in Australia, significant increases in odds for COPD hospital admissions were observed in association for same day (12%) and previous day (10%) bushfire related severe pollution event in Sydney, Australia (Martin et al., 2013). However, null results were observed for Newcastle and Wollongong. No associations were also observed in Sydney in a different ecological time-series study (Hanigan et al., 2008). Significant positive associations were reported in studies included in the Naeher et al. (2007) review for various COPD outcomes: incidences of symptoms in Denver, CO (Sutherland et al., 2005), emergency room visits in California (Duclos et al., 1990) and hospital admissions in Malaysia (Mott et al., 2005).

Adverse effects of wildland fires for other respiratory outcomes among the general population have been reported. These include hospital admission for acute bronchitis and bronchiolitis (Delfino et al., 2008), and pneumonia (Delfino et al., 2008; Morgan et al., 2010). Significant effects were also observed for emergency room visits for pneumonia and acute bronchitis (Rappold et al., 2011), and upper respiratory tract illness including laryngitis, sinusitis and rhinitis (Duclos et al., 1990; Emmanuel, 2000). Although, null results were reported in one study for hospital admissions for pneumonia and acute bronchitis in Sydney and Wollongong in association with bushfire associated elevated pollution, significant positive associations were observed in Newcastle in the same study on the day immediately following or two days after the pollution event (Hanigan et al., 2008). Null results were also reported for emergency room visits for pneumonia and pharyngitis in association with forest fires in California (Duclos et al., 1990), and unspecified upper respiratory tract infection in association with forest fires in California and peat forest fires in North Carolina (Duclos et al., 1990; Rappold et al., 2011).

Cardiovascular effects of wildland fire smoke in the general public

In all, 13 peer-reviewed papers reporting on the possible cardiovascular effects of wildland fires 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), and a case-crossover study conducted in Australia (Johnston et al., 2007). 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. In addition, 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 old (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,

Table 5. Summary of review of epidemiological studies of wildland firefighters.

Citation (Author.Year.Journal)	Location	Type of fire activity/burn	Study design	Subject description	Sample size	Exposure measures	Health end-point(s)	Major confounders accounted for	Result summary
Betchley et al. (1997). Am J Ind Med	Salem, Oregon, USA	Forest fire	Cohort (repeated measures)	Wildland firefighters	76 firefighters for cross-shift and 53 firefighters for cross-seasonal	Participation at work-shift and burn season with exposure to wildland fire smoke	FVC and FEV_1 by spirometry, and respiratory symptoms including cough, phlegm production, sore throat, chest tightness, chest pain and wheezing by questionnaire	Smoke exposure prior to preseason, smoking within 2 h, of pulmonary function tests, chest cold within the preceding 4 weeks, medications, allergies, wood use	The change of respiratory symptoms cross-shift and cross-seasonal were not statistically significant (2). There were significant mean individual cross-shift declines in FVC, FEV_1 and $FEF_{25–75}$, and significant cross-seasonal declines in FEV_1 and $FEF_{25–75}$ decreases which continued after nearly 2.5 months. Recovery in all spirometry measures was observed across the winter months (171–380 days) when firefighting activities are minimal.
Jacquin et al. (2011). Am J Ind Med	Corsica, France	Forest fire	Cohort (repeated measures)	Wildland firefighters	108 (59 were smokers and 49 were non-smokers without any acute or chronic pulmonary disease)	Participation at work-shift and burn season with exposure to wildland fire smoke	FVC, FEV_1 and PEFR were measured by spirometer. Exhaled CO was also measured. Symptoms relating to related to smoke exposure or CO intoxication were recorded.	Smoking	FEV_1, FVC and PEF declined immediately after the end of exposure and greater declines was seen after 24 h (−0.53 l, −0.59 l and −531 min^{-1}, respectively). Declines in spirometric parameters persisted through 3 months after the fire season in comparison with baseline values (FEV_1 −0.28 l; FVC −0.34 l; PEF −45 l min^{-1}, $p < 0.05$ for each). No difference was observed in declines between smoking and non-smoking groups.
Gaughan et al. (2008). J Occup Environ Med	Rocky Mountain National Park, Yosemite National Park, Glacier National Park, USA	Forest fire	Cohort (repeated measures)	Wildland firefighters from two Interagency Hotshot Crews	58	Participation at work-shift and burn season with exposure to wildland fire smoke	Respiratory symptoms collected using a questionnaire, lung functions were assessed by spirometry, intracellular and airway inflammation (extracellular myeloperoxidase and eosinophilic cationic protein were measured through induced sputum and nasal lavage analyses)	Age, gender, height and race/ethnicity	Mean FEV_1, mean upper and lower respiratory symptom scores were higher immediately after exposure compared to preseason. FEV_1 recovered by post-season after subjects had spent time away from fighting fires. After adjusting for a significant association between an

(*continued*)

Table 5. Continued

Citation (Author.Year.Journal)	Location	Type of fire activity/burn	Study design	Subject description	Sample size	Exposure measures	Health end-point(s)	Major confounders accounted for	Result summary
									individual's preseason FEV_1 and subsequent FEV_1 values, lower FEV_1 values were associated with greater upper respiratory symptom scores, with higher sputum eosinophilic cationic protein concentration and with higher sputum myeloperoxidase concentrations. Individual increases in sputum and nasal eosinophilic cationic protein and myeloperoxidase from preseason to post-fire were all significantly associated with post-fire respiratory symptom scores.
Hejl et al. (2013). J Occup Environ Hyg	Savannah River Site, SC, USA	Prescribed burns	Cohort (repeated measures)	U.S. Forest Service wildland firefighters	12	Participation at work-shift and burn season with exposure to wildland fire smoke, $PM_{2.5}$ and CO	Inflammation (IL-1β, IL-6, IL-8, TNF-α and CRP, SAA, ICAM-1, VCAM-1 in dried blood spot samples)	Various confounders including task, residential use of wood, exposure to secondhand smoke collected but not controlled for in a multi-factor model	IL-8 concentration in dried blood spot samples significantly increased across the work shift as indicated by a post-/pre-work shift ratio of 1.70 (95% CL: 1.35, 2.13), while concentrations of IL-8, CRP, and ICAM-1 increased in 91.7, 75 and 67% of subject-days across work shift. Firefighters who lighted fires as opposed to other work tasks had the largest cross-work shift increase in IL-8 concentrations.
Adetona et al. (2011). Inhal Toxicol	Savannah River Site, SC, USA	Prescribed burns	Cohort (repeated measures)	Wildland firefighters	24	Participation at work-shift and burn season with exposure to wildland fire smoke, $PM_{2.5}$ and CO	Lung function measured by spirometry	Gender, race, work-time, cumulative burn days, smoking and allergies	Declines across work-shift on burn days were observed for mean individual FVC and FEV_1, but not for $FEF_{25–75}$, PEF and FEV_1/FVC, but changes on burn days were not significantly different from those on non-burn days. However, each additional day of working at a prescribed burn,

									at any given point during the burn season, was associated with declines of 24 ml in pre-shift FVC and 24 ml in pre-shift FEV_1 in non-allergic firefighters, and 8 ml in FVC and 4 ml in FEV_1 in allergic firefighters.
Rothman et al. (1991). J Occup Environ Med	Northern California, USA	Forest fire	Cohort (repeated measures)	Wildland firefighters	52	Participation at work-shift and burn season with exposure to wildland fire smoke	Lung functions measured by spirometry	Age, race, gender, passive exposure to cigaret smoke, exposure to diesel exhaust, smoking status, history of asthma, history of hay fever, previous number of years as a firefighter, hours of fire-fighting occurring before baseline measurements and use of a bandanna for respiratory protection	The mean cross-seasonal change in FEV_1 and FVC were −1.2% (95% CI −0.5%, −2.0%) and −0.3% (95% CI 0.4%, −1.0%) respectively. Decreases in FEV_1 and FVC were most strongly associated with hours of recent fire-fighting activity. Firefighters in the high activity category (mean ± SE, 73 ± 7 h of fire-fighting in previous week) had a −2.9% (130 ml) change in FEV_1 and a −1.9% (102 ml) change in FVC. Most respiratory symptoms evaluated increased significantly across the fire season and several symptoms (eye irritation, nose irritation and wheezing) were associated with recent fire-fighting.
Slaughter et al. (2004). J Occup Environ Hyg	Western United States	Forest fire	Cohort (repeated measures)	Wildland firefighters	65	$PM_{3.5}$, acrolein, formaldehyde, and CO	Lung functions measured by spirometry	Gender, age, and current smoking status	FEV_1 declined significantly by −0.1251 from preshift to postshift. A 1000 μg/m^3 increase in PM3.5 was associated with a −0.0301 change in the cross-shift FEV_1 (95% CI [−0.087, 0.026]). Acrolein, formaldehyde, and carbon monoxide exposure were also not significantly associated with changes in FEV_1, FVC, or FEF_{25-75}

(*continued*)

Table 5. Continued

Citation (Author.Year.Journal)	Location	Type of fire activity/burn	Study design	Subject description	Sample size	Exposure measures	Health end-point(s)	Major confounders accounted for	Result summary
Swiston et al. (2008). Eur Respir J	British Columbia, Canada	Forest fire	Cohort (repeated measures)	Seasonal forest firefighters	52	Participation at work-shift and burn season with exposure to wildland fire smoke, CO	Pulmonary symptoms was assessed by question-naires; spirometry; local airway and systemic inflammation measured as cell counts and cyto-kine concentrations	Information collected and controlled for at the subject selection stage (smoking, co-mor-bidities). Study design eliminated the others.	The number of circulat-ing white blood cells, band cells and per-centage sputum gran-ulocytes increased significantly follow-ing fire-fighting shifts. Serum inter-leukin (IL)-6, IL-8 and monocyte chemotactic protein-1 levels also signifi-cantly increased fol-lowing fire-fighting, while there were no changes in band cells, IL-6 and IL-8 fol-lowing strenuous physical exertion without fire-fighting.
Gaughan et al. (2014). J Occup Environ Hyg	Rocky Mountain National Park, Colorado, USA	Forest fire	Cohort (repeated measures)	Wildland firefighters of the Alpine Interagency Hotshot Crew	17	Participation at work-shift and burn season with exposure to wildland fire smoke, CO, levoglucosan, respirable crystalline silica, respirable par-ticulate matter, exhaled breath CO	Lung functions measured by spirometry	Age, job and crew operation	Cross-shift declines in FEV_1 were associated with exposure to higher concentrations of respirable levoglu-cosan. FEV_1 declined across the work shift by -0.051 ($p = 0.08$), with participating wildland firefighters in the high levoglu-cosan exposure group having a mean FEV_1 decline of 0.231 compared to a mean decline of 0.021 in the low levoglucosan exposure group. Larger mean values of pre-shift exhaled breath CO were asso-ciated with larger declines in FEV_1.
Miranda et al. (2012). J Toxicol Environ Health	Leiria, Coimbra and Aveiro, Portugal	Forest fire	Cohort (repeated measures)	Wildland firefighters	40	CO, VOC and NO_2	Exhaled nitric oxide was measured; lung function measured by spirometry	None listed	FEV_1, F_{25}, F_{50} and MEF) significantly declined after exposure at work shifts. Exhaled nitric oxide concen-tration significantly decreased after exposure to smoke.
Adetona et al. (2013b). Sci Total Environ	USA	Prescribed burn – whole vegetation	Cohort (repeated measures)	Wildland firefighters	17	$PM_{2.5}$ and carbon monoxide	Urinary oxidative stress biomarkers	Age, length of fire-fighting career	There was no cross-shift increase in mean malondialdehyde concentrations. However, 8OHdG significantly increased across

									workshift for only subjects who had been firefighters for 2 years or less, while decreases were observed for firefighters who had been firefighters for longer periods with a significant decrease observed for those working as firefighters for 10 years or more. This effect of career length was not evident when age which correlated with career length was added into the model.
Gaughan et al. (2014). Am J Ind Med	USA	Wildfire – whole vegetation	Cross-sectional	Wildland firefighters	38	Levoglucosan (particle)	Urinary oxidative stress biomarkers, inflammatory biomarkers, arterial stiffness and lung function was measured using spirometry	Smoking, historical occupational exposures, allergies, pre-existing asthma	Aortic augmentation index increased by 10.5% (2.5, 18.5%) with every unit increase in oxidative stress score (average of the *z*-scores of 8-OHdG and 8-isoprostane), while the oxidative stress score was positively associated with levoglucosan concentration.
Liu et al. (1992). Am Rev Respir Dis	USA	Wildfire – whole vegetation	Cohort (repeated measures)	Wildland firefighters	63	Wildland fire smoke	Lung function	Gender, smoking history, allergies/asthma, years as a firefighter, symptoms	FVC, FEV_1 and $FEF_{25–75}$ declined by 0.09 l, 0.151 and 0.44 l/s across the wildfire season while airway responsiveness increased across the season.
Serra et al. (1996). Am J Ind Med	Italy	Wildfire – whole vegetation	Cross-sectional	Wildland firefighters as exposed group and policemen as control	92 wildland firefighters versus 51 policemen	Wildland fire smoke	Lung function	Age, height, smoking, previous work experience	Wildland firefighters had significantly ($p < 0.05$) lower spirometry measures compared to the policemen – FEV_1 (3.90 versus 4.04 l); FEF_{75} (8.37 versus 8.38 l/s); FEV_1/FVC (80.07 versus 83.89%); FEF_{25} (1.58 versus 1.99 l/s) and FEF_{50} (4.73 versus 5.54 l/s).

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 visit due to heart failure in relation to exposure to peat forest fire smoke in North Carolina (Rappold et al., 2011), and hospital admission 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 US 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). In addition, four other studies – one from Malaysia in relation to the 1997 forest fire haze episode in Southeast Asia, one from the US in relation to forest fires, and two from Australia in relation to bushfires – report null findings for hospital admission 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 US (Delfino et al., 2008; Rappold et al., 2011), myocardial infarction in one study from the US (Rappold et al., 2011), and stroke from two studies, one from the US and the other from Australia (Delfino et al., 2008; Morgan et al., 2010).

Other health effects of wildland fire smoke in the general public

Several studies have investigated the association between wildland fire smoke exposure and all-cause, non-traumatic or ill-defined mortality among the general public (Analitis et al., 2011; Emmanuel, 2000; Hanninen et al., 2009; Morgan et al., 2010; Sastry, 2002; Vedal & Dutton, 2006). Hanninen et al. (2009) reported non-significant increases of 0.8–2.1% in daily mortality in provinces in Southern Finland per additional $10\,\mu g/m^3$ of $PM_{2.5}$ with varying lag periods (lag 0–3, 0–4 day average) during a 2-week period in September 2002 when air quality in the provinces was impacted by smoke from wildfires in Eastern Europe. Significant increases in non-traumatic and ill-defined mortality were observed among different age groups in association with dichotomous measures of air pollution (visibility < 0.91 km and $PM_{10} > 210\,\mu g/m^3$) in two cities in Malaysia during the 1997 Southeast Asia forest fire related haze episode (Sastry, 2002). However, results were inconsistent among the age groups across the two cities. Also, null findings for all-cause mortality among the general public in association with forest fires were reported in one study in Singapore and another in Denver, CO (Emmanuel, 2000; Vedal & Dutton, 2006). However, Analitis et al. (2011) reported increases in all natural deaths among the general public in Athens, Greece in association with forest fires in areas adjacent to the city. Medium fires were associated with a 4.9% (0.3, 9.6%) increase in all natural deaths while large fires were associated with a 49.7% (37.2, 63.4%) increase.

The effect of *in utero* acute exposure to wildland fire smoke on birth outcomes has been investigated in one study (Holstius et al., 2012). Compared to babies born from pregnancies occurring entirely during periods before or after the forest fires, birth weights of newborns were 7.0 g (2.2, 11.8 g), 9.7 g (4.8, 14.5 g) and 3.3 g (−0.6, 7.2 g) lower when the wildfires occurred in the first, second and third trimester of pregnancy respectively. Finally, increases in circulating immature polymorphonuclear (band cells) leukocytes and serum pro-inflammatory cytokine concentrations were increased in healthy male volunteers in Singapore during the 1997 Southeast Asia forest fire haze compared to the period immediately after the episode (Tan et al., 2000; Van Eeden et al., 2001). These results indicated that exposure to smoke from the forest fires caused systemic inflammation. However, the authors reported the lack of measurements prior to the forest fire related exposure as a limitation of their study.

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, including one non-peer reviewed paper. 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 work shift have been observed in a few studies. Betchley et al. (1997) reported declines of 65 ml, 150 ml and 497 ml/s in forced vital capacity (FVC), forced expiratory volume in 1 s (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 et al., 2004). However, Gaughan et al. (2014) 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 due to the large variability that is associated with lung function measures

(Borsboom et al., 1999; Troyanov et al., 1994), and the probable confounding effect of physical exertion that accompanies working at wildfires or prescribed burns. Moreover, Adetona et al. (2011) did not detect any differences in cross-shift changes in lung function measures between days when a crew of wildland firefighters in Southeastern US 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. (2011) 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 Southeastern US (Adetona et al., 2011). Declines in lung function measures have also been observed across periods encompassing one or two burn seasons (Betchley et al., 1997; Gaughan et al., 2008; Liu et al., 1992; Miranda et al., 2012). Significant cross-season declines of 90 ml, 150 ml and 440 ml/s in FVC, FEV_1 and $FEF_{25–75}$, respectively, were 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/s (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. In addition, 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., 2011), 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 et al., 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 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 percentage granulocytes, mostly neutrophils, in induced sputum among wildland firefighters in British Columbia. On the other hand, exhaled nitric oxide decreased across firefighting work-shifts in a group of firefighters in another study that was conducted in Portugal (Miranda et al., 2012). Although, the investigators were not expecting this result since reduction in exhaled nitric oxide indicates reduction in airway inflammation, they noted that their observation was similar to results observed in smokers. They noted that cigaret 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 cigaret 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 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 US (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., 2000, 2007) only two studies of oxidative stress among wildland firefighters were identified (Adetona et al., 2013b; Gaughan et al., 2014). 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 2 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. (2014) 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 study of wildland firefighters that were identified investigated the effect of occupational wildland fire

smoke exposure over the longer term. Consequently, very little 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 5.

Evidence from the health effects of ambient air pollution indirectly linked to combustion of wood or vegetation

In all, 11 papers that reported on the adverse health effects of ambient air pollution indirectly linked to the combustion of wood or the vegetation were identified. Health outcomes were limited to cardiovascular and/or respiratory health end-points in most of the studies. Null results were reported for all-cause mortality in the whole population in a study involving a city-wide program to replace wood heaters as the primary source of residential heating in Launceston, Australia (Johnston et al., 2013). However, a significant reduction in all-cause mortality was reported for males in the population after the implementation of the stove replacement program. No association was observed between wood/vegetative smoke-associated particulate matter and all-cause mortality in Phoenix, Arizona or Washington, DC in two source apportionment studies (Ito et al., 2006; Mar et al., 2006). These two studies also reported no associations for cardiovascular or respiratory mortality (Ito et al., 2006; Mar et al., 2006). Although, no reduction in all-cause, cardiovascular and respiratory mortality among the whole population was observed in association with a stove intervention program in Launceston, Australia, significant reductions of 11.4, 17.9 and 22.8%, respectively, were observed for males in the city (Johnston et al., 2013). No reductions were observed during the period under study for Hobart, Australia, the control city where no specific air quality intervention had occurred. Sanhueza et al. (2009) reported an increase of 12.5 and 5.5% in respiratory and cardiovascular mortality respectively for every $100\,\mu g/m^3$ rise in PM_{10} in Temuco, Chile. The association between respiratory mortality and ambient particulate matter pollution was stronger during the winter with a rise of 15.7% in mortality for every $100\,\mu g/m^3$ rise in PM_{10}. Almost 70% of the population in Temuco is reported to use wood for cooking or heating during the winter, and 87% of the winter PM_{10} is estimated to originate from residential wood combustion (Díaz-Robles et al., 2014).

In a source apportionment method, Sarnat et al. (2008) did not find any association between admission for all respiratory diseases combined and wood smoke associated $PM_{2.5}$ determined using chemical balance, factor analysis or tracer technique in Atlanta, Georgia. However, a 2.3% rise was observed for every inter-quartile increase in total carbon, the tracer of vegetative burning in Spokane, Washington in another source apportionment study (Schreuder et al., 2006). Associations were also reported between ambient particulate matter concentrations and respiratory admission in Temuco, Chile and Christchurch, New Zealand (Mcgowan et al., 2002; Sanhueza et al., 2009) and outpatient visits for all respiratory illnesses combined in Temuco Chile (Díaz-Robles et al., 2014). Positive relationships were also observed between ambient particulate matter concentrations and respiratory infections including pneumonia and influenza in Christchurch, New Zealand where 90% of the particulate air pollution is estimated to originate from wood burners (Mcgowan et al., 2002). In addition, reduced ambient $PM_{2.5}$ was associated with decrease in the incidences of bronchitis, influenza and throat infection in a cohort study conducted among children in Libby, Montana after a wood stove replacement program (Noonan et al., 2012). Reductions in the incidences of wheeze and colds were also observed in the same study. On the other hand, the odds of experiencing respiratory symptoms within the previous twelve months were not different among study participants living in two cities with substantially different rates of wood stove use and levels of ambient wood smoke exposure in Australia (Bennett et al., 2010). Associations have been reported between residential wood combustion derived ambient air pollution and hospital admissions for asthma and COPD in Christchurch, New Zealand (Mcgowan et al., 2002), and clinical encounters (inpatient and outpatient) for infant bronchiolitis in British Columbia (Karr et al., 2009).

McGowan et al. (2002) did not find any association between residential wood combustion derived ambient particulate matter pollution and hospital admission for cardiac dysrhythmia, ischemic heart disease or heart failure in Christchurch, New Zealand. Conversely, a significant 1.26% increase in hospital admission for all cardiovascular diseases combined for every $14.8\,\mu g/m^3$ increase in PM_{10} was observed in the same study. In addition, Sarnat et al. (2008) reported a positive association between wood smoke derived ambient particulate matter pollution as determined by chemical balance, factor analysis or tracer technique and emergency room visits for all cardiovascular diseases combined in Atlanta, while a 5.8% increase in hospital admissions for all cardiovascular diseases combined in association with every $100\,\mu g/m^3$ increase in ambient air PM_{10} was observed in Temuco, Chile during the cold season (Sanhueza et al., 2009). Schreuder et al. (2006) did not observe an association between concentrations of total carbon, used as a tracer of vegetative burning and emergency room visits for all cardiovascular diseases combined.

Evidence from the health effects of household air pollution related to the combustion of wood or other vegetative materials

As with other exposure scenarios, most of the studies of the health effects of household air pollution associated with the combustion of wood and other vegetative materials investigate respiratory health end-points. There is strong evidence that continuous long-term exposures to smoke related to residential wood combustion is linked to the development of COPD and chronic bronchitis. A recent meta-analyses showed that the odds of having doctor diagnosed or lung function defined COPD increased more than four folds (OR: 4.29 [1.35, 13.70]) in populations using wood burners in their residences compared to those using cleaner fuels (Kurmi et al., 2010). The corresponding odds ratio for bronchitis in

the same study is 2.64 (2.12, 3.29). Similar results are reported by two other recent meta-analyses of studies investigating residential combustion of all solid biomass fuel types including wood (Kurmi et al., 2012; Po et al., 2011). Similarities have been shown in the pathology of wood and tobacco smoke associated COPD (González-García et al., 2012; Guzmán-Grenfell et al., 2011; Montano et al., 2004; Moran-Mendoza et al., 2008; Sandoval et al., 1993). However, some of the indicators of the disease have been observed to be more severe in wood smoke associated COPD (González-García et al., 2012; Guzmán-Grenfell et al., 2011; Montano et al., 2004; Sandoval et al., 1993). Higher metalloproteinase activity was observed in patients with wood smoke associated COPD (Montano et al., 2004), while they also had more severe pulmonary arterial hypertension and bronchial hyper-responsiveness (González-García et al., 2012; Sandoval et al., 1993).

It is not clear that there is an association between exposure to smoke from residential combustion of wood and asthma. Ostro et al. (1994) reported an association (OR: 1.59 [1.28, 1.97]) between the use of wood stove or fireplace and nocturnal asthma and other asthma related symptoms among adult patients (18–70 years) diagnosed with asthma in Denver, CO (Ostro et al., 1994). Significant association (OR: 5.64 [1.1, 27.9]) was reported for asthma together with other ''respiratory problems'' among children less than 12 years in one study in Mexico (Graham et al., 2005). Lack of separation prevented firm conclusion specific to asthma to be drawn from the results of the study. No association was observed between wood stove use and acute asthma in young children (1 month to 5 years) in a hospital-based study that was conducted in Malaysia (Azizi et al., 1995). Eisner et al. (2002) reported no association between wood stove use and asthma health outcomes in Northern California. In a survey study, an insignificant increase in odds for the prevalence of doctor-diagnosed asthma was observed among children (4–6 years) living in homes in three rural communities in Guatemala where wood for cooking was combusted exclusively in open fires relative to those living in homes where improved stoves were used (Schei et al., 2004). On the other hand, significant positive associations were observed for asthma-related symptoms in the same study. Similar results have been reported for asthma-related symptoms including wheezing and shortness of breath among children and adults (Da Silva et al., 2012; Ingale et al., 2013; Mengersen et al., 2011; Romieu et al., 2009; Smith-Sivertsen et al., 2009). Residential wood combustion has also been linked with other respiratory symptoms and decreased lung function (Da Silva et al., 2012; Diaz et al., 2007; Fullerton et al., 2011; Guggisberg et al., 2003; Guneser et al., 1994; Ingale et al., 2013; Köksal et al., 2013; Mengersen et al., 2011; Rinne et al., 2006; Riojas-Rodríguez et al., 2001; Romieu et al., 2009; Saha et al., 2005; Smith-Sivertsen et al., 2009; Triche et al., 2002, 2005).

Results were inconsistent between two meta-analyses of studies of the relationship between asthma and household air pollution due to the combustion of solid biomass fuel without any specification of type (Kurmi et al., 2012; Po et al., 2011). While Kurmi et al. (2012) reported a doubling of the risk of developing asthma (OR: 1.96 [1.29, 2.99]) in children exposed to biomass smoke in the indoor environment, Po et al. (2011) reported non-significant reduction in risk among children (OR: 0.50 [0.12, 1.98]) and a non-significant increase in risk among women (OR: 1.34 [0.93, 1.93]). There was an overlap of only two studies out of a total of 12 that were considered by both meta-analyses. Kurmi et al. (2012) reported that the positive result from their meta-analysis be interpreted with caution because the methodology was imperfect in all the five papers they reviewed.

Acute lower respiratory disease and pneumonia, especially among children, is perhaps the most studied health end-point in association with household air pollution due to the combustion of solid biomass fuel including wood. Positive associations between exposure to smoke from residential combustion of wood and acute respiratory infections among children were reported in all seven papers that were identified (Collings et al., 1990; Etiler et al., 2002; Johnson & Aderele, 1991; Johnson et al., 2008; Mahalanabis et al., 2002; Smith et al., 2011; Taylor & Nakai, 2012). Association was positive but insignificant in the only study that reported results for adults (women) (Taylor & Nakai, 2012). In a randomized control trial involving replacement of open fires with chimney stoves, an intention-to-treat analysis showed that a 50% reduction in personal carbon monoxide exposure related to the intervention was associated with reduced odds of being diagnosed by the physician with pneumonia (OR: 0.82 [0.70, 0.98]) or hypoxemic pneumonia (OR: 0.72 [0.59, 0.92]) (Smith et al., 2011). Cooking with wood also increased the risk of mortality among children admitted into the hospital for acute lower respiratory infection in Nigeria (Johnson & Aderele, 1991; Johnson et al., 2008).

Residential wood combustion was linked to tuberculosis in two studies from Mexico. The odds of having active tuberculosis increased 1.5 (1.0, 2.4) times with past or present use of wood among subjects in Mexico City (Perez-Padilla et al., 2001), while cooking with wood for more than 20 years increased the odds of being diagnosed with tuberculosis among subjects living in rural areas in Southern Mexico (García-Sancho et al., 2009). These results are supported by the findings of two meta-analyses of studies that investigated the association between tuberculosis and household air pollution due to the combustion of solid biomass fuel without specification of type (Kurmi et al., 2014; Sumpter & Chandramohan, 2013). However, the authors of a third meta-analysis reported that the association is uncertain (Lin et al., 2007). Association in three of the five studies they included in the meta-analysis was significantly positive, while it was insignificantly negative in the other two. It should be noted that the findings that wood smoke exposure is a risk factor for tuberculosis are similar to those reported for tobacco smoke (Kurmi et al., 2012).

The authors of a pooled analysis of the International Lung Cancer Consortium of data from seven studies across Asia and North America reported an increased odds of 1.21 (1.06, 1.38) for having lung cancer among all subjects that combusted wood for cooking or heating in their homes (Hosgood et al., 2010). The odds (OR: 1.43 [0.97, 2.11]) were higher but not significant for lifetime wood users alone. Similar results were reported by Kurmi et al. (2012) in another meta-analysis of studies investigating the association between lung cancer and household air pollution due to

residential combustion of solid biomass fuel (all types with and without coal). A higher effect estimate (odds ratio) was reported for females (1.81) compared to males (1.16) for residential combustion of all types of solid biomass fuel combined. This is probably because women experience higher exposure levels compared to men in many of the study areas since they are usually the primary cooks and spend more time in the home.

Household air pollution due to residential combustion of wood has also been linked to adverse birth outcome. Most of the studies have focused on birth weight. Newborns were 14–243 g lighter when they were born to mothers living in homes where wood was used for cooking and/or heating compared to mothers living in homes that used cleaner fuels (Abusalah et al., 2012; Amegah et al., 2012; Boy et al., 2002; Kadam et al., 2013; Siddiqui et al., 2008; Wylie et al., 2014). The results were marginally significant in the study recording the smallest difference (14 g) (Wylie et al., 2014), and another reporting a difference of 82 g (Siddiqui et al., 2008). The smallest difference reported in studies with significant results was 63 g (Boy et al., 2002). Children who were born to women who continued to use open fire to cook were 89 (range: 27–204) grams lighter than those who switched to chimney stoves in a randomized control trial in Guatemala (Thompson et al., 2011). Although the results were not always significant, the incidence of low birth weight (birth weight <2500 g) also tended to be higher among newborns of mothers cooking with wood compared to those cooking with cleaner fuels, and among newborns of mothers cooking with open fire compared to those cooking with chimney stoves. The risk of stillbirth was observed to be higher (PR: 1.24 [1.08, 1.41]) among births to mothers cooking with firewood compared to those cooking with liquefied petroleum gas or electricity in a national survey in India (Lakshmi et al., 2013). Stillbirth was also observed to be more common (4% versus 0%) among women cooking with wood in another Indian study (Wylie et al., 2014). The positive findings for reduced birth weight and stillbirth are supported by results of a meta-analysis of studies investigating the associations between adverse birth outcomes and exposure to household air pollution due to the combustion of solid biomass fuel without specification of type (Pope et al., 2010).

Very few studies have investigated the cardiovascular health effects of chronic exposure to household air pollution due to combustion of wood. Reduced blood pressure has been observed in association with the replacement of open fire with chimney stoves in Guatemala and Nicaragua (Clark et al., 2013; Mccracken et al., 2007). Probable cardiovascular effects of residential exposure to wood smoke have also been demonstrated by observed increase in reactive hyperemia index (a measure of endothelial function) and reduced incidence of non-specific ST-segment depression (a measure of ventricular repolarization) in association with exposure reduction interventions (Allen et al., 2011; Mccracken et al., 2011). The possibility that wood smoke could be a risk factor for adverse cardiovascular outcomes is also supported by results of studies of residential combustion of non-specified solid biomass fuel including wood. A recent study found a higher prevalence of atherosclerotic plaque and an increased mean carotid artery intima-media thickness, an indicator of the progression of atherosclerosis, in association with household solid biomass fuel use (Painschab et al., 2013). Higher blood pressure (Baumgartner et al., 2011), higher prevalence of arterial hypertension (Dutta et al., 2012) and worse measures of markers of pulmonary hypertension in association with residential biomass fuel use have also been reported in recent studies (Emiroglu et al., 2010). Schematics of the pathways involved in pulmonary and systemic effects of wildland fire smoke exposure are presented in Figures 1 and 2.

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 gavage 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, 2008; Xiao et al., 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.

Oxidative stress and inflammation in the airways

Wood smoke particles contain and possess the potential to generate ROS including the hydroxyl radical, superoxide anions and hydrogen peroxide in cells (Danielsen et al., 2011; Lee et al., 2008b; Leonard et al., 2000, 2007; Liu et al., 2005). No increase in ROS generation was observed in one study (Forchhammer et al., 2012a). However, measurement of ROS in this study was done in human umbilical endothelial cells, unlike in the other studies in which measurements were conducted in airway cell lines. Due to its ability to cause cellular ROS generation, wood smoke exposure can clearly result in oxidative stress. This is measured as the induction of antioxidant enzymes, changes in antioxidant capacity or as changes in concentrations of products of oxidative degradation of macromolecules (lipid peroxidation or oxidative DNA damage). Results are consistent for oxidative stress in *in vitro* studies conducted with monocytes, macrophages, epithelial and endothelial cells. Upregulation of antioxidant enzymes such as heme oxygenase (HO-1) and superoxide dismutase (Cu/Zn SOD), depletion of endogenous antioxidant capacity such as reduction in glutathione (GSH) and increases

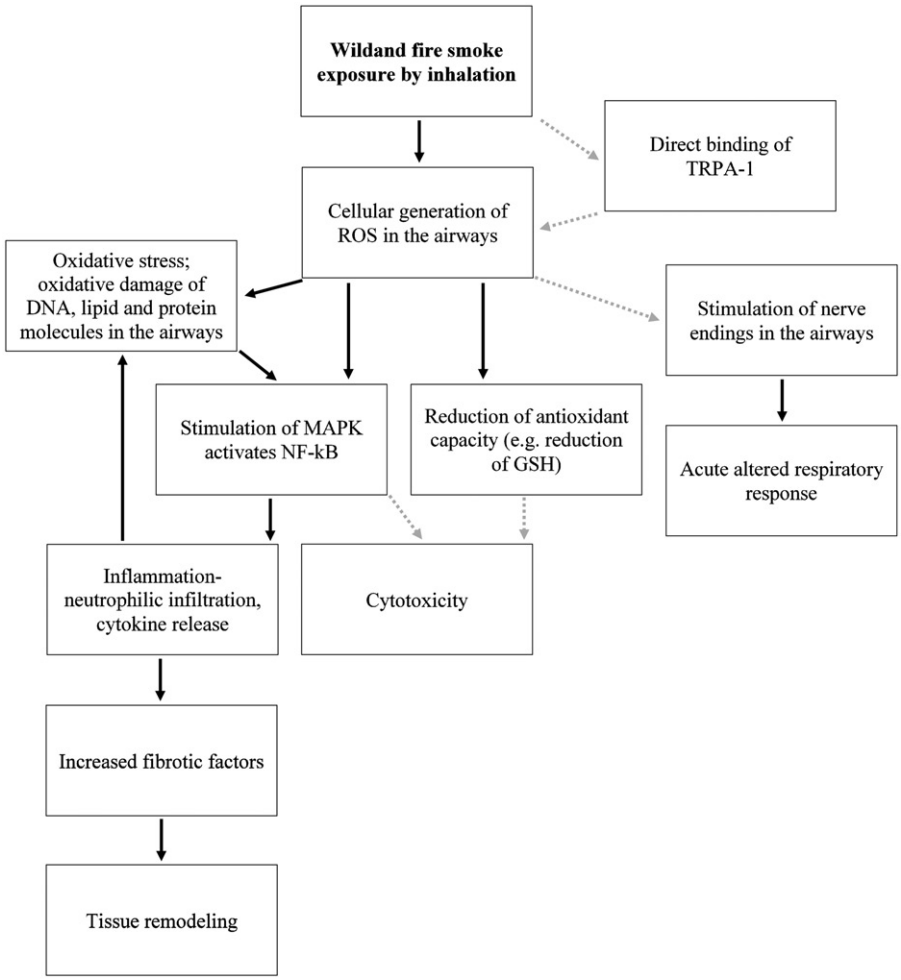


Figure 1. Schematic representation of the pathways for the effects of wildland fire smoke exposure by inhalation on the respiratory system. The solid black arrows represent pathways known with higher level of certainty, while the broken gray arrows represent pathways known with less certainty. *Abbreviations*: TRPA-1: transient receptor potential ankyrin-1; MAPK: mitogen-activated protein kinases; NF-kB: nuclear factor-κB; GSH: glutathione.

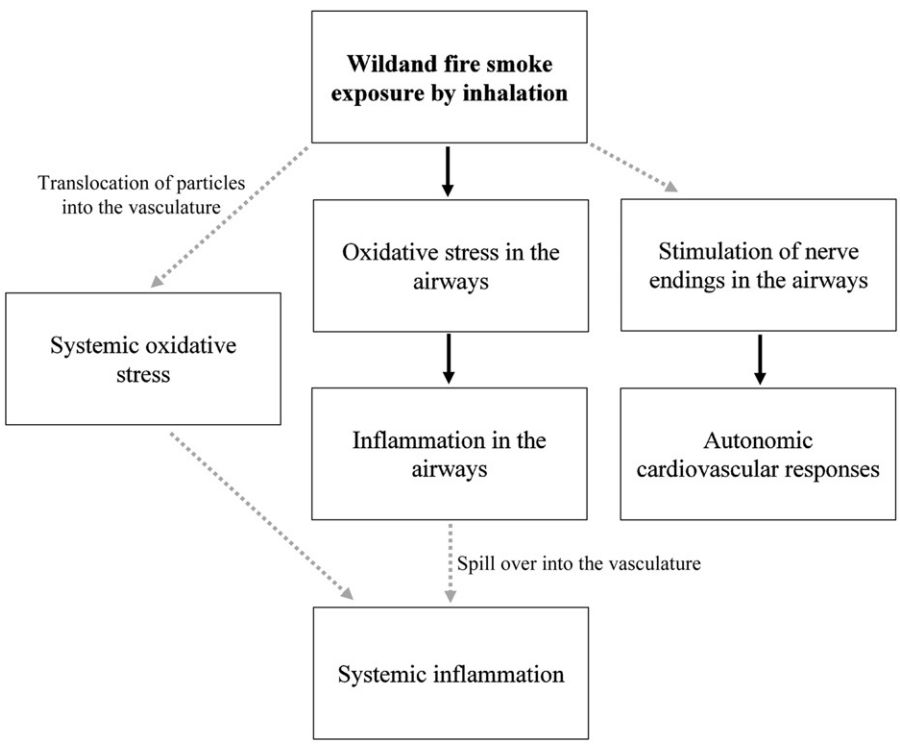


Figure 2. Schematic representation of the pathways for the systemic effect of wildland fire smoke exposure by inhalation. The solid black arrows represent pathways known with higher level of certainty, while the broken gray arrows represent pathways known with less certainty.

in products of oxidative damage of macromolecules such as DNA strand breaks, oxidized guanines and lipid peroxides are observed across these airway cell lines (Corsini et al., 2013; Danielsen et al., 2009,2011; Forchhammer et al., 2012a; Karlsson et al., 2006; Kubátová et al., 2006; Lee et al., 2008b; Leonard et al., 2007; Liu et al., 2005). Dose-dependent increases in the formation of strand breaks and formamidopyrimidine DNA glycosylase (FPG) sites were observed in human A549 lung epithelial and THP-1 monocytic cell lines in a pair of studies (Danielsen et al., 2009, 2011). The induction of oxidative stress response by wood smoke is dependent on its composition. Non-polar and mid-polar fractions of wood smoke particle extracts caused more GSH depletion than the polar fraction in murine 264.7 macrophages (Kubátová et al., 2006). In addition, the organic extract of wood smoke particulate matter generated more strand breaks in human A549 epithelial cells (Danielsen et al., 2009). Oxidative responses including oxidative lipid damage and reduction in antioxidant capacity have also been observed in the airways in *in vivo* models (Park et al., 2004; Ramos et al., 2013; Wegesser et al., 2009; Williams et al., 2013). However, results about oxidative responses in the airways from human experimental studies are not as consistent as those reported for *in vitro* and *in vivo* studies (Barregard et al., 2008; Sehlstedt et al., 2010; Stockfelt et al., 2012). Although, Barregard et al. (2008) reported an increase in exhaled malondialdehyde, Stockfelt et al. (2012) observed no difference after experimental exposure of healthy adults to wood smoke. Possible reasons for this inconsistency include heterogeneity in the measured oxidative stress marker, timing of measurements, dose of exposure, the type of wood and exposure protocol as it relates to the combustion conditions.

Airway inflammation characterized by an increase in cytokine release by various airway cells (Bølling et al., 2012; Corsini et al., 2013; Danielsen et al., 2011; Forchhammer et al., 2012a; Karlsson et al., 2006; Kocbach et al., 2008a,b; Myatt et al., 2011), and infiltration of immune cells, especially neutrophils, in various *in vivo* models is also induced by exposure to wood smoke particles (Bhattacharyya et al., 2004, 1998; Danielsen et al., 2010; Karlsson et al., 2006; Park et al., 2004; Samuelsen et al., 2009; Wegesser et al., 2009, 2010; Williams et al., 2013; Zhu et al., 2012). There is evidence that ROS generated by wood smoke particles stimulate mitogen-activated protein kinases (MAPKs) such as Jun-N-terminal kinases (JNK). This in turn activates nuclear factor-κB (NF-κB) and other pro-inflammatory transcription factors, thus causing an up-regulation of cytokines. This pathway is similar to what has been described for pulmonary inflammation induced by ambient air particulate matter (Brook et al., 2010). Inhibition of endogenous generation of ROS by wood smoke particle extract and each of the following steps in the theorized pathway resulted in the reduction of the activities of subsequent steps and eventual expression of IL-8 in human bronchial epithelial cells (Perng et al., 2013). In addition, IL-8 release induced by extracts of beech wood smoke generated particles was completely blocked by a specific inhibitor of p38 MAPK in both A549 and THP-1 cells (Corsini et al., 2013). However, specific inhibition of NF-κB resulted in significant inhibition of smoke extract induced IL-8 in only the A549 cells in the same study. Activation of MAPKs by ROS may also result in the up-regulation of anti-oxidant genes. The induction of HO-1 in alveolar epithelial cell II by wood smoke particle extract was completely abolished by pre-treatment with a combination of MAPKs (JNK, p38 and ERK) inhibitors (Lee et al., 2008b).

While endotoxins could adhere to particles and cause inflammation, their inactivation results in only partial attenuation of the pro-inflammatory effects of wood smoke particles (Kocbach et al., 2008a,b). Rather there is evidence that organic components of the particles (which could be metabolized to produce ROS) are largely responsible (Bølling et al., 2012; Kocbach et al., 2008a,b; Wegesser et al., 2009, 2010; Wong et al., 2011). Inflammatory response to wood smoke exposure may also be dependent on combustion conditions which are a determining factor for the physico-chemical properties of the resulting particles (Bølling et al., 2012; Danielsen et al., 2010, 2011). This may account for the negative findings that have been observed with respect to inflammation in some human chamber experiment studies in contrast with actual exposure situations for wildland fire-fighters (Gaughan et al., 2008; Hejl et al., 2013; Riddervold et al., 2012; Sehlstedt et al., 2010; Stockfelt et al., 2012; Swiston et al., 2008). Furthermore, neutrophilic infiltration in the lungs, increased exhaled nitric oxide and serum/urine Clara cell protein (CC16) observed in some human experiment studies suggest a pulmonary pro-inflammatory effect of wood smoke in humans (Barregard et al., 2008; Ghio et al., 2012; Stockfelt et al., 2012).

Alternative mechanistic pathways may contribute to the oxidative stress and inflammatory potentials of wood smoke particles in the airways. The binding of the electrophile binding site of Transient Receptor Potential Ankyrin-1 (TRPA-1) has been postulated as a potential pathway (Shapiro et al., 2013). Although TRPA-1 is mainly expressed in C-fibers that innervate the airways, they are also expressed in non-neuronal airway cells including fibroblasts and small airway epithelial cells. It is hypothesized that the binding of TRPA-1 in these non-neuronal cells could result in their release of pro-inflammatory mediators. It is possible that other alternative pathways that have been described for ambient air particulate matter may also contribute to the pro-inflammatory effects of wood smoke particles (Brook et al., 2010).

Cytotoxicity in airway cells

Cytotoxicity measured as increase in the release of lactate dehydrogenase (LDH) due to membrane damage or reduction in the number of viable cells is induced *in vitro* by exposure to suspension of wood smoke particles (Bølling et al., 2012; Danielsen et al., 2009; Forchhammer et al., 2012a; Kocbach et al., 2008a; Kubátová et al., 2006). Increase in LDH (Samuelsen et al., 2009), increase in the number of dead macrophages and reduction in the number of viable macrophages in bronchoalveolar lavage have also been observed in *in vivo* models (Wegesser et al., 2009; Williams et al., 2013). It appears that macrophages are especially sensitive to toxicity from acute wood smoke particle (Franzi et al., 2011; Kubátová et al., 2006; Wegesser et al., 2009). Activation of

NF-κB consequent upon phagocytosis of particles has been suggested as a possible pathway for wood smoke particles induced toxicity (Franzi et al., 2011; Williams et al., 2013), and diminution of antioxidant capacity (GSH depletion) may also contribute (Kubátová et al., 2006). Less efficient combustion conditions and higher organic content may also contribute towards more potent induction of cytotoxicity in airway cells (Bølling et al., 2012; Kocbach et al., 2008a; Kubátová et al., 2006).

Systemic oxidative stress and inflammation

It is unclear how wood smoke inhalation could cause systemic inflammation and uncertain how pulmonary oxidative stress and inflammation may spill over into the circulation. However, it has been demonstrated in a rat model that exposure to wood smoke by oral gavage causes increases in markers of oxidative stress and inflammation in the liver (Danielsen et al., 2010). Moreover, increases in MCP-1, a chemotactic cytokine, and HO-1 and 8-oxoguanine glycosylase (OGG1) increased in the liver 24 h after intratracheal instillation of wood smoke particles. These suggest that both inflammatory responses and the induction of antioxidant enzymes in the liver are caused by inhalation exposure to wood smoke.

Increases in the concentrations of markers of lipid peroxidation in circulation have been observed in other *in vivo* models consequent upon acute exposures to elevated levels of wood smoke (Park et al., 2004; Ramos et al., 2013). Inconsistent results have been observed in human experiments (Barregard et al., 2006, 2008; Danielsen et al., 2008; Forchhammer et al., 2012b; Sehlstedt et al., 2010; Stockfelt et al., 2012, 2013). Nonetheless, increases in mRNA levels of OGG1 in peripheral blood mononuclear cells (Danielsen et al., 2008), urinary concentration of 8-isoprostane (Barregard et al., 2006) and serum concentrations of inflammation markers have been observed in human subjects after experimental exposure to wood smoke compared to filtered air (Barregard et al., 2006, 2008; Stockfelt et al., 2012).

Exposure to elevated levels of wood smoke may cause neurological effects, and induce inflammatory responses and oxidative stress responses including lipid peroxidation and genotoxic DNA damage in brain tissues (Chen et al., 2007b; Gorgun et al., 2014; Lee et al., 2005a, 2010, 2011). These toxic effects have been linked to the ability of wood smoke to inhibit mitochondrial complexes and consequent augmentation of ROS generation, possibly from increased supply of nicotinamide adenine dinucleotide (NADH) from the glycolytic pathway (Gorgun et al., 2014; Lee et al., 2010). Glycolytic metabolism may serve as a compensatory mechanism for impaired mitochondrial respiration induced by wood smoke exposure (Gorgun et al., 2014; Lee et al., 2010). Overexpression of neuroglobin in mice has been shown to attenuate the inhibition of mitochondrial complexes, ameliorate the shift towards glycolytic metabolism and reduce oxidative DNA damage in brain tissue after exposure to wood smoke (Gorgun et al., 2014; Lee et al., 2011).

Inflammation and thrombosis are closely linked and it has been hypothesized that lower airway inflammation could induce production of coagulation in the liver (Barregard et al., 2006; Stockfelt et al., 2013). However, inconsistent results have been observed for the effects of acute wood smoke exposure on thrombosis in human exposure studies Both Barregard et al. (2006) and Stockfelt et al. (2013) observed a larger increase in plasma concentration of coagulation factor VIII after wood smoke exposure relative to filtered air exposure. However, Stockfelt et al. (2013) reported larger decreases in plasma concentration of fibrinogen and in platelet counts after exposure to wood smoke relative to exposure to filtered air. In addition, Hunter et al. (2014) reported no difference in platelet count, platelet activation and thrombus formation following experimental exposure to wood smoke relative to exposure to filtered air.

Immune suppression

Although initial activation of immune cells could result in a temporary bactericidal effect, immune suppression may be the long-term effect (Samuelsen et al., 2009). Whereas neutrophilic infiltration may result in some initial clearance of infection, the sensitivity of macrophages to cytotoxicity and the impairment by wood smoke exposure of their ability to phagocytize and kill bacteria may suppress immune response in the longer term (Samuelsen et al., 2009). Instillation of wood smoke particles reduced *in vivo* clearance of *Staphylococcus aureus* and Fc-receptor mediated phagocytosis (Zelikoff et al., 2002). In one study, macrophages from wood smoke exposed rabbits were less adherent, had reduced phagocytic ability, and a lower maximum number of associated bacteria (Fick et al., 1984). Although there was no change in macrophage phagocytic ability 24 h after exposure, there was an increase in bacterial load in lung tissue of wood smoke exposed mice inoculated with *Streptococcus pneumoniae* in another study (Migliaccio et al., 2013). Reduction in T-cell activation was also observed 2 h and up to 7 days after wood smoke exposure in macrophages co-cultured with $CD4^+$ cells accompanied by a reduction in the production of interferon-gamma (IFN-γ). Activation of the non-canonical NF-κB, RelB, with the possible involvement of the aryl-hydrocarbon receptor (AhR) activation by PAHs has been hypothesized as a possible pathway for suppression of macrophages (Migliaccio et al., 2013).

Tissue remodeling

Chronic wood smoke exposure has been identified as a cause of COPD in patients with the disease (González-García et al., 2012; Guzmán-Grenfell et al., 2011; Montano et al., 2004; Moran-Mendoza et al., 2008; Sandoval et al., 1993). *In vivo* experiments have recently been carried out to study the linkage (Ramos et al., 2009; Zou et al., 2014). Features that are characteristic of the pathogenesis of COPD were observed in the experiments. Increase in the expression and activities of metalloproteinases, which are involved in the degradation of the extracellular matrix, was observed in the chronic exposure (1–7 months) animal models (Ramos et al., 2009; Zou et al., 2014). Macrophage number in bronchoalveolar lavage increased after 1–4 months of exposure, while neutrophils

increased after 4–7 months of exposure (Ramos et al., 2009). Both cell types possess the secretor phenotype of metalloproteinases. Metalloproteinases can activate transforming growth factor beta (TGF-β) resulting in the proliferation of fibroblasts (Zou et al., 2014). In addition, the serum concentration of tissue inhibitor of metalloproteinase (TIMP-1) in rats increased after 4 or 7 months of exposure (Zou et al., 2014). This indicates a protease–antiprotease imbalance which is a hallmark of the disease.

The transition of epithelial cells to fibroblast phenotype or epithelial–mesenchymal transition in small airways has been proposed as a potential mechanism contributing to airway fibrosis in COPD. Evidence of this transition was observed *in vitro*. Increase in the expression of vimentin and type I collagen (mesenchymal markers) and a decrease in E-cadherin (epithelial markers) were observed in rat tracheal epithelial cells, while both types of markers were in the airway sub-epithelium *in vivo* indicating that bronchial fibroblasts may directly originate from epithelial cells in wood smoke exposed rats (Zou et al., 2014). The number of fibroblasts in the small airways also increased after wood smoke exposure.

Finally, emphysematous lesions also increased in the rats and guinea pig models after 7 months of exposure (Ramos et al., 2009; Zou et al., 2014). Collagen deposition was observed in the rat but not in the guinea pig model after 7 months of exposure. Twenty-eight days of exposure increased deposition of collagen protein, hydroxyproline, collagen I and III, in lung tissue in another rat model indicating that chronic wood exposure could cause pulmonary fibrosis (Zhu et al., 2012).

Interaction with the autonomic nervous system in the airways

Wood smoke interacts with vagal pulmonary afferent nerves. Rapidly adapting myelinated irritant receptors and nerve endings of the unmyelinated bronchopulmonary C-fibers can both be stimulated by wood smoke exposure (Lai & Kou, 1998a,b). Apparently, two distinct types of immediate ventilator responses are caused by the stimulation of these sensory receptors. The stimulation of the C-fibers causes inhibitory response observed as slow respiration after spontaneous wood smoke (6 ml) inhalation by tracheostomy in Sprague–Dawley rats, while excitatory augmented inspiration resulted from the stimulation of irritant receptors (Kou et al., 1995). Pre-treatment with capsaicin which selectively blocks conduction by C-fibers abolished slow respiration in exposed animals, while cooling both cervical vagi at the higher temperature required for blocking conduction by only the myelinated fibers abolished augmented inspiration. Filtration of particles did not affect slow respiration while it prevented augmented inspiration in some of the exposed animals (Kou et al., 1995). Consequently, it can be concluded that the stimulation of C-fibers by the gas phase of wood smoke resulted in the inhibitory slow respiration response, and this effect is for example similar to that observed for acrolein (Faroon et al., 2008a,b). Gas and/or particulate phase components of wood smoke induced the excitatory augmented inspiration via the irritant receptors. Pre-treatment of animals with a hydroxyl radical scavenger or an iron chelator to prevent the formation of the radical abolished or attenuated both effects in most of the exposed animals (Ho & Kou, 2000; Kou et al., 1997).

Wood smoke induces bronchoconstriction as indicated by increased lung resistance and reduced dynamic lung compliance and hyper-responsiveness to itself or other bronchoconstrictors (Ho & Kou, 2002; Hsu & Kou, 2001; Hsu et al., 1998a,b, 2000). These effects are also mediated through the interaction of wood smoke with bronchopulmonary C-fibers. The involvement of both cholinergic mechanisms and tachykinins such as substance P and neurokinin A released due to the stimulation of C-fiber nerve endings has been determined (Ho & Kou, 2002; Hsu et al., 1998a,b). Pre-treatment of experimental animals with inhibitors of tachykinin and acetylcholine receptors attenuated bronchoconstriction effects. The endogenous production of free radicals subsequent to wood smoke exposure is also involved, as pre-treatment with a hydroxyl radical scavenger attenuated wood smoke induced airway hyper-responsiveness (Hsu et al., 2000). Tachykininergic and endogenous production of hydroxyl radicals have also been implicated in wood smoke-induced lung injury characterized by increased extravascular water, atelectasis and pulmonary parenchymal injury (Lin & Kou, 2000; Lin et al., 2001).

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 1 h after exposure to wood smoke with an average PM_1 concentration of 300 $\mu g/m^3$ for 3 h compared to filtered air exposure (Unosson et al., 2013). There were no changes immediately or 20 h after wood smoke exposure in both the time domain and repolarization variables of the electrocardiogram in another human experimental study (2 h 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-h period following experimental 1-h exposure of firefighters to wood smoke with an average PM_1 concentration of 1115 $\mu g/m^3$ (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 h 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, 6 or 20 h following 3 h exposures to average $PM_{2.5}$ concentrations of 200 and 354 $\mu g/m^3$ (Forchhammer et al., 2012b). 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).

Table 6. Evidence summary.

Class of effects	Confidence level	Basis for rating
Public		
Acute respiratory	Strong evidence	Consistent results across epidemiological studies of different designs and across different study regions; dose-response with ambient air PM concentration; plausibility indicated by results of epidemiological studies of ambient PM and experimental studies of wood smoke.
Acute cardiovascular	Weak evidence	Inconsistent results with few positive findings from small number of ecological epidemiological studies; few relevant experimental studies with inconsistent results with some positive findings; plausibility indicated by results of ambient PM studies.
Birth outcome	Not enough evidence to conclude	Positive results from one ecological study; applicability of evidence from related exposure situation limited by various factors (lack of specificity in exposure, difference in exposure pattern, undefined temporality); no experimental evidence.
Wildland firefighter		
Acute respiratory	Weak evidence	Some effects observed in a few wildland firefighter studies; strong evidence from related exposure situation in humans; plausibility of effects supported by experimental studies; although lung function decline across the work-shift was observed in most studies, controls were not used to account for changes that may have otherwise occurred; no difference was observed in lung function decline between exposure and no exposure days in the only study that included controls.
Acute cardiovascular	Not enough evidence to conclude	Virtual lack of study among wildland firefighters; applicability of evidence from related exposure situation limited by various factors (lack of specificity in exposure, difference between public and healthy wildland firefighter; undefined temporality); few relevant experimental studies with inconsistent results.
Chronic respiratory	Not enough evidence to conclude	Virtual lack of study among wildland firefighters; applicability of evidence from related exposure situation limited by various factors (lack of specificity in exposure, difference in exposure pattern, undefined temporality); very few relevant experimental studies.
Chronic cardiovascular	Not enough evidence to conclude	Virtual lack of study among wildland firefighters; weak but limited evidence from related exposure situation; lack of relevant experimental studies.

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 from the lungs into the vasculature, 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, 2010; Ghelfi et al., 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.

Summary of evidence

The summary of evidence for the hazard associated with wildland fire smoke is presented in Table 6. 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 et al., 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.

No study of the effect of wood smoke on an experimental model with pre-existing airway disease was identified. However, as noted earlier, there is a preponderance of evidence from *in vivo* studies that wood smoke exposure could result in neutrophilic inflammation in the lungs and bronchoconstriction induced via tachykinin receptors in the airways. These responses could contribute to the exacerbation of COPD and asthma, respectively (Ling & Van Eeden, 2009; Papi et al., 2006; Ramalho et al., 2011). Mechanistic studies also reveal that wood smoke exposure could result in immune suppression subsequent to the initial pro-inflammatory response. This could plausibly explain the increases in medical visits for respiratory infections in association with wildland fire smoke exposure. Effects of wood smoke exposure on airway inflammation were not observed in most human experiment studies. The contrasting results with other types of studies could have been partly due to the use of healthy subjects in the human experiment studies, differences in exposure conditions and possibly differences in the physicochemistry of the emissions contributing towards the exposure. We thus conclude with high level of confidence that wildland fire smoke exposure is a respiratory hazard to the general public.

Ambient PM concentration is associated with cardiovascular morbidity and mortality (Brook et al., 2010). However, results for the cardiovascular effects of PM exposure specific to wildland fire smoke among the general public is less unequivocal. Most of the studies focus on acute outcomes and reported null findings. Significant positive findings were reported for associations between measures of particulate matter exposure and hospital admissions for hypertension with respect to agricultural burns and emergency room visits for heart failure during a peat fire event (Arbex et al., 2010; Rappold et al., 2011). While two of the papers reported non-significant protective effects in association with wildland fire associated PM (Hanigan et al., 2008; Johnston et al., 2007), five others reported non-significant positive effects for various cardiovascular health end-points (Crabbe, 2012; Delfino et al., 2008; Duclos et al., 1990; Morgan et al., 2010; Rappold et al., 2011). However, all but (one cohort and one case-crossover) of the 13 studies identified for cardiovascular health end-points were based on the ecological time-series study design. These studies could therefore have been limited by probable misclassification of exposure (including avoidance by those with preexisting condition) and the lack of power to detect small differences on a population level. Consequently, we conclude that there is currently weak evidence that wildland fire smoke exposure is a cardiovascular hazard to the general public. No effect of wood smoke exposure on systemic inflammation, which contributes towards the precipitation of cardiovascular events, was observed in most of the human experiment studies that have been conducted. Yet, the use of healthy subjects limits the generalization of the results to the general population, and acute systemic inflammation in response to occupational wood smoke exposure has been observed among wildland firefighters.

Although there is evidence that cumulative exposure results in progressive lung function decline during the burn season among wildland firefighters, it is presently unclear whether this decline persists across off-seasons or whether this decline is larger than what would be expected for an average individual. Respiratory symptoms and biomarkers of airway and systemic inflammation have also been observed to increase in association with occupational wildland fire smoke exposure in a few studies. However, results in many of the studies are limited by small sample sizes and the determination of outcomes through self-reporting. Acute airway and systemic oxidative stress and inflammation, and effects on the autonomic nervous system were observed consequent upon wood smoke exposure in *in vivo* studies, but findings were mostly not positive in human experiments involving healthy subjects (firefighters in one study). The sample sizes in the human experiments were small by design and exposure was for limited periods. Emissions were also generated under combustion conditions very different from what is typical for the wildland firefighters. Given the significant differences in emissions of smoke constituents among combustion phases and different fuel/vegetation types, this is a critical deficiency in broadly applying results to the wildland firefighter population and the general public.

No study of long-term effects of continuing occupational smoke exposure among wildland firefighters was identified. Nevertheless, chronic exposure to household air pollution due to the residential combustion of wood is associated with COPD and chronic bronchitis. Such exposures have also been linked to acute lower respiratory infections, and possibly asthma, tuberculosis and lung cancer. However, the differences in combustion conditions and emissions, exposure patterns, susceptibility status and population characteristics make extrapolation of results of household air pollution studies to wildland firefighters difficult. Exposures are typically more frequent and occur over longer durations for household air pollution, while firefighters are more likely to be male and a healthier working population. Wildland firefighters are also more likely to be exposed to emissions generated under better oxygenated combustion conditions with a higher heat release rate. It is also unclear how immune suppression and fibrotic/emphysema-like effects observed in relation to wood smoke exposure *in vivo* studies may be interpreted with respect to wildland firefighter occupational exposure since exposure patterns in these studies are not reflective of what the wildland firefighter experiences. Taken together, we conclude that there is weak evidence for acute respiratory and systemic effects of occupational wildland fire smoke among wildland firefighters. However, it is unclear what these acute pulmonary and systemic physiological responses translate to in terms of the occurrence of acute and chronic diseases among wildland firefighters. Furthermore, the current lack of studies of health end-points of known clinical significance among this working population leads us to conclude that there is not enough information to determine the long-term cardiovascular and respiratory hazard of cumulative occupational wildland fire smoke exposure among wildland firefighters. A recent study demonstrated that organic constituents of combustion-generated ambient aerosols can aggravate and promote atherosclerosis and cardiovascular disease in a cumulative fashion (Keebaugh et al., 2015). The freshly emitted smoke from woodland fires may have greater percentage of toxic organic compounds than ambient particles, and thus might represent a greater potential health risk to firefighters over the course of their careers.

There is a need for studies of clinically significant health end-points including the incidences of diseases in relation to occupational wildland fire smoke among this population. Wildland firefighters would be expected to be healthier than the average population, and they have a very different wildland fire smoke exposure pattern compared to the exposure of the general public which is generally at a lower concentration and less frequent, or to exposure of individuals to smoke due to residential combustion of wood which typically is more continuous. In addition, the tasks in wildland firefighting can lead to greater exposures to particulates as the job is physically demanding and require elevated ventilation rates, which can result in substantially increased doses of smoke to the respiratory tract (Danielsen et al., 2008). Rothman et al. (1991) demonstrated that recent cumulative exposures were more strongly associated with greater changes in lung function, and it would be important to note if such was the case regarding cardiovascular function. The cumulative exposure effect previously mentioned would be particularly important for those who are at most risk occupationally as it is

unknown whether cessation of exposure among wildland firefighters during the off-season may allow for recovery and reversibility of effects (Danielsen et al., 2008). Consequently, it is hard to extrapolate results from other populations to wildland firefighters.

Evidence for effects of wildland fire smoke exposure on birth outcomes is currently very limited. Only one study has so far been conducted with results of a small effect on birth weight. Extrapolation of the results from household air pollution studies is difficult for some of the same reasons that were discussed above for cardiovascular health end-points among the general public and health effects among wildland firefighter.

Conclusion

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 would reduce their exposure, and result in misclassification of their exposure and the bias of 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 6 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). In addition, 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.

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 for wildland firefighters 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. In addition, 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.

Acknowledgements

The authors would like to acknowledge Guannan Huang and Nicole Nation of the Department of Environmental Science, University of Georgia for their literature search assistance.

Declaration of interest

The authors acknowledge funding from the Joint Fire Science Program (Project # 13-1-02-14) with additional support from the Pacific Northwest Research station of the United States Forest Service. The manuscript reflects solely the opinion of the authors and not of the funding source. The authors declare that there are no conflicts of interest.

References

Abusalah A, Gavana M, Haidich A-B, et al. (2012). Low birth weight and prenatal exposure to indoor pollution from tobacco smoke and wood fuel smoke: a matched case-control study in Gaza Strip. Matern Child Health J 16:1718–27.

Academy of Nutrition and Dietetics 2012. Evidence analysis manual. Chicago, IL: Academy of Nutrition and Dietetics.

Adetona O, Hall DB, Naeher LP. (2011). Lung function changes in wildland firefighters working at prescribed burns. Inhal Toxicol 23: 835–41.

Adetona O, Simpson CD, Onstad G, Naeher LP. (2013a). Exposure of wildland firefighters to carbon monoxide, fine particles, and levoglucosan. Ann Occup Hyg 57:979–91.

Adetona O, Zhang JJ, Hall DB, et al. (2013b). Occupational exposure to woodsmoke and oxidative stress in wildland firefighters. Sci Total Environ 449:269–75.

Akagi S, Yokelson RJ, Burling IR, et al. (2013). Measurements of reactive trace gases and variable o 3 formation rates in some South Carolina biomass burning plumes. Atmosph Chem Phys 13:1141–65.

Alföldy B, Giechaskiel B, Hofmann W, Drossinos Y. (2009). Size-distribution dependent lung deposition of diesel exhaust particles. J Aerosol Sci 40:652–63.

Allen RW, Carlsten C, Karlen B, et al. (2011). An air filter intervention study of endothelial function among healthy adults in a woodsmoke-impacted community. Am J Respir Crit Care Med 183:1222–30.

Alonso-Blanco E, Calvo A, Fraile R, Castro A. (2012). The influence of wildfires on aerosol size distributions in rural areas. Scientific World J 2012:735697.

Alves C, Gonçalves C, Evtyugina M, et al. (2010a). Particulate organic compounds emitted from experimental wildland fires in a Mediterranean ecosystem. Atmos Environ 44:2750–9.

Alves C, Gonçalves C, Pio C, et al. (2010b). Smoke emissions from biomass burning in a Mediterranean shrubland. Atmos Environ 44: 3024–33.

Alves C, Vicente A, Nunes T, et al. (2011). Summer 2009 wildfires in Portugal: emission of trace gases and aerosol composition. Atmos Environ 45:641–9.

Amegah AK, Jaakkola J, Quansah R, et al. (2012). Cooking fuel choices and garbage burning practices as determinants of birth weight: a cross-sectional study in Accra, Ghana. Environ Health 11:78.

Analitis A, Georgiadis I, Katsouyanni K. (2011). Forest fires are associated with elevated mortality in a dense urban setting. Occup Environ Med 69:158–62.

Annane D, Chadda K, Gajdos P, et al. (2011). Hyperbaric oxygen therapy for acute domestic carbon monoxide poisoning: two randomized controlled trials. Intensive Care Med 37:486–92.

Anttila P, Makkonen U, Hellén H, et al. (2008). Impact of the open biomass fires in spring and summer of 2006 on the chemical composition of background air in south-eastern Finland. Atmos Environ 42:6472–86.

Araujo JA, Nel AE. (2009). Particulate matter and atherosclerosis: Role of particle size, composition and oxidative stress. Part Fibre Toxicol 6:24.

Arbex MA, Martins LC, De Oliveira RC, et al. (2007). Air pollution from biomass burning and asthma hospital admissions in a sugar cane plantation area in Brazil. J Epidemiol Community Health 61: 395–400.

Arbex MA, Saldiva PHN, Pereira LA, Braga ALF. (2010). Impact of outdoor biomass air pollution on hypertension hospital admissions. J Epidemiol Community Health 64:573–9.

Aurell J, Gullett BK. (2013). Emission factors from aerial and ground measurements of field and laboratory forest burns in the southeastern US: $PM_{2.5}$, black and brown carbon, voc, and pcdd/pcdf. Environ Sci Technol 47:8443–52.

Azizi B, Zulkifli H, Kasim M. (1995). Indoor air pollution and asthma in hospitalized children in a tropical environment. J Asthma 32:413–18.

Balachandran S, Pachon JE, Lee S, et al. (2013). Particulate and gas sampling of prescribed fires in south Georgia, USA. Atmos Environ 81:125–35.

Barboni T, Cannac M, Pasqualini V, et al. (2010). Volatile and semi-volatile organic compounds in smoke exposure of firefighters during prescribed burning in the Mediterranean region. Int J Wildland Fire 19:606–12.

Barboni T, Chiaramonti N. (2010). Btex emissions during prescribed burning in function of combustion stage and distance from flame front. Combust Sci Technol 182:1193–200.

Barregard L, Sällsten G, Andersson L, et al. (2008). Experimental exposure to wood smoke: effects on airway inflammation and oxidative stress. Occup Environ Med 65:319–24.

Barregard L, Sällsten G, Gustafson P, et al. (2006). Experimental exposure to wood-smoke particles in healthy humans: effects on markers of inflammation, coagulation, and lipid peroxidation. Inhal Toxicol 18:845–53.

Baumgartner J, Schauer JJ, Ezzati M, et al. (2011). Indoor air pollution and blood pressure in adult women living in rural China. Environ Health Perspect 119:1390–5.

Bein K, Leikauf GD. (2011). Acrolein – a pulmonary hazard. Mol Nutr Food Res 55:1342–60.

Bedia J, Herrera S, Camia A, et al. (2014). Forest fire danger projections in the Mediterranean using ENSEMBLES regional climate change scenarios. Clim Change 122:185–99.

Bennett C, Dharmage S, Matheson M, et al. (2010). Ambient wood smoke exposure and respiratory symptoms in Tasmania, Australia. Sci Total Environ 409:294–9.

Betchley C, Koenig JQ, Van Belle G, et al. (1997). Pulmonary function and respiratory symptoms in forest firefighters. Am J Ind Med 31: 503–9.

Bhattacharyya SN, Dubick MA, Yantis LD, et al. (2004). In vivo effect of wood smoke on the expression of two mucin genes in rat airways. Inflammation 28:67–76.

Bhattacharyya SN, Manna B, Smiley R, et al. (1998). Smoke-induced inhalation injury: effects of retinoic acid and antisense oligodeoxynucleotide on stability and differentiated state of the mucociliary epithelium. Inflammation 22:203–14.

Black RR, Meyer CP, Touati A, et al. (2011). Emissions of PCDD and PCDF from combustion of forest fuels and sugarcane: a comparison between field measurements and simulations in a laboratory burn facility. Chemosphere 83:1331–8.

Bølling AK, Pagels J, Yttri KE, et al. (2009). Health effects of residential wood smoke particles: the importance of combustion conditions and physicochemical particle properties. Part Fibre Toxicol 6:20.

Bølling AK, Totlandsdal AI, Sallsten G, et al. (2012). Wood smoke particles from different combustion phases induce similar pro-inflammatory effects in a co-culture of monocyte and pneumocyte cell lines. Part Fibre Toxicol 9:45.

Booze TF, Reinhardt TE, Quiring SJ, Ottmar RD. (2004). A screening-level assessment of the health risks of chronic smoke exposure for wildland firefighters. J Occup Environ Hyg 1:296–305.

Borsboom GJ, Van Pelt W, Van Houwelingen HC, et al. (1999). Diurnal variation in lung function in subgroups from two Dutch populations: consequences for longitudinal analysis. Am J Respir Crit Care Med 159:1163–71.

Boy E, Bruce N, Delgado H. (2002). Birth weight and exposure to kitchen wood smoke during pregnancy in rural Guatemala. Environ Health Perspect 110:109–14.

Brook RD, Brook JR, Urch B, et al. (2002). Inhalation of fine particulate air pollution and ozone causes acute arterial vasoconstriction in healthy adults. Circulation 105:1534–6.

Brook RD, Rajagopalan S, Pope CA, et al. (2010). Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. Circulation 121: 2331–78.

Burling I, Yokelson RJ, Griffith DW, et al. (2010). Laboratory measurements of trace gas emissions from biomass burning of fuel types from the southeastern and southwestern United States. Atmosph Chem Phys 10:11115–30.

Cancado JE, Saldiva PH, Pereira LA, et al. (2006). The impact of sugar cane-burning emissions on the respiratory system of children and the elderly. Environ Health Perspect 114:725–9.

Cashdollar KL, Lee CK, Singer JM. (1979). Three-wavelength light transmission technique to measure smoke particle size and concentration. Appl Opt 18:1763–9.

Chakrabarty RK, Moosmüller H, Garro MA, et al. (2006). Emissions from the laboratory combustion of wildland fuels: particle morphology and size. J Geophys Res: Atmospheres 111: D07204.

Chen L-WA, Moosmüller H, Arnott WP, et al. (2007a). Emissions from laboratory combustion of wildland fuels: emission factors and source profiles. Environ Sci Technol 41:4317–25.

Chen L, Lee HM, Greeley GH, Englander EW. (2007b). Accumulation of oxidatively generated DNA damage in the brain: a mechanism of neurotoxicity. Free Radic Biol Med 42:385–93.

Chen L, Verrall K, Tong S. (2006). Air particulate pollution due to bushfires and respiratory hospital admissions in Brisbane, Australia. Int J Environ Health Res 16:181–91.

Cherrie JW, Brosseau LM, Hay A, Donaldson K. (2013). Low-toxicity dusts: current exposure guidelines are not sufficiently protective. Ann Occup Hyg 57:685–91.

Churches T, Corbett S. (1991). Asthma and air pollution in Sydney. New South Wales Public Health Bull 2:72–4.

Clark ML, Bachand AM, Heiderscheidt JM, et al. (2013). Impact of a cleaner-burning cookstove intervention on blood pressure in Nicaraguan women. Indoor Air 23:105–14.

Collings D, Sithole S, Martin K. (1990). Indoor woodsmoke pollution causing lower respiratory disease in children. Trop Doct 20: 151–5.

Commodore AA, Jannik GT, Eddy TP, et al. (2012). Radioactivity in smoke particulates from prescribed burns at the Savannah River Site and at selected southeastern United States forests. Atmos Environ 54: 643–56.

Corsini E, Budello S, Marabini L, et al. (2013). Comparison of wood smoke $PM_{2.5}$ obtained from the combustion of fir and beech pellets on inflammation and DNA damage in A549 and THP-1 human cell lines. Arch Toxicol 87:2187–99.

Crabbe H. (2012). Risk of respiratory and cardiovascular hospitalisation with exposure to bushfire particulates: new evidence from Darwin, Australia. Environ Geochem Health 34:697–709.

Da Silva LFF, Saldiva SRDM, Saldiva PHN, et al. (2012). Impaired lung function in individuals chronically exposed to biomass combustion. Environ Res 112:111–17.

Danielsen PH, Bräuner EV, Barregard L, et al. (2008). Oxidatively damaged DNA and its repair after experimental exposure to wood smoke in healthy humans. Mutat Res 642:37–42.

Danielsen PH, Loft S, Jacobsen NR, et al. (2010). Oxidative stress, inflammation and DNA damage in rats after intratracheal instillation or oral exposure to ambient air and wood smoke particulate matter. Toxicol Sci 118:574–85.

Danielsen PH, Loft S, Kocbach A, et al. (2009). Oxidative damage to DNA and repair induced by Norwegian wood smoke particles in human A549 and THP-1 cell lines. Mutat Res 674:116–22.

Danielsen PH, Møller P, Jensen KA, et al. (2011). Oxidative stress, DNA damage, and inflammation induced by ambient air and wood smoke particulate matter in human A549 and THP-1 cell lines. Chem Res Toxicol 24:168–84.

De Vos AJ, Cook A, Devine B, et al. (2006). Effect of protective filters on fire fighter respiratory health during simulated bushfire smoke exposure. Am J Ind Med 49:740–50.

De Vos AJ, Reisen F, Cook A, et al. (2009). Respiratory irritants in australian bushfire smoke: air toxics sampling in a smoke chamber and during prescribed burns. Arch Environ Contam Toxicol 56:380–8.

Delfino RJ, Brummel S, Wu J, et al. (2008). The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003. Occup Environ Med 66:189–97.

Díaz-Robles LA, Fu JS, Vergara-Fernández A, et al. (2014). Health risks caused by short term exposure to ultrafine particles generated by residential wood combustion: a case study of Temuco, Chile. Environ Int 66:174–81.

Diaz E, Bruce N, Pope D, et al. (2007). Lung function and symptoms among indigenous Mayan women exposed to high levels of indoor air pollution. Int J Tuberc Lung Dis 11:1372–9.

Duclos P, Sanderson LM, Lipsett M. (1990). The 1987 forest fire disaster in California: assessment of emergency room visits. Arch Environ Health 45:53–8.

Dunn K, Shulman S, Stock A, Naeher L. (2013). Personal carbon monoxide exposures among firefighters at prescribed forest burns in the southeastern United States. Arch Environ Occup Health 68: 55–9.

Dunn KH, Devaux I, Stock A, Naeher LP. (2009). Application of end-exhaled breath monitoring to assess carbon monoxide exposures of wildland firefighters at prescribed burns. Inhal Toxicol 21:55–61.

Dutta A, Ray MR, Banerjee A. (2012). Systemic inflammatory changes and increased oxidative stress in rural Indian women cooking with biomass fuels. Toxicol Appl Pharmacol 261:255–62.

Eisner MD, Yelin EH, Katz PP, et al. (2002). Exposure to indoor combustion and adult asthma outcomes: environmental tobacco smoke, gas stoves, and woodsmoke. Thorax 57:973–8.

Emiroglu Y, Kargin R, Kargin F, et al. (2010). Bnp levels in patients with long-term exposure to biomass fuel and its relation to right ventricular function. Pulm Pharmacol Ther 23:420–4.

Emmanuel SC. (2000). Impact to lung health of haze from forest fires: the Singapore experience. Respirology 5:175–82.

Epton MJ, Dawson RD, Brooks WM, et al. (2008). The effect of ambient air pollution on respiratory health of school children: a panel study. Environ Health 7:23–33.

Etiler N, Velipasaoglu S, Aktekin M. (2002). Incidence of acute respiratory infections and the relationship with some factors in infancy in Antalya, Turkey. Pediatr Int 44:64–9.

Evans LF, Weeks IA, Eccleston AJ, Packham DR. (1977). Photochemical ozone in smoke from prescribed burning of forests. Environ Sci Technol 11:896–900.

Evtyugina M, Calvo AI, Nunes T, et al. (2013). VOC emissions of smouldering combustion from Mediterranean wildfires in central Portugal. Atmos Environ 64:339–48.

Faroon O, Roney N, Taylor J, et al. (2008a). Acrolein health effects. Toxicol Ind Health 24:447–90.

Faroon O, Roney N, Taylor J, et al. (2008b). Acrolein environmental levels and potential for human exposure. Toxicol Ind Health 24: 543–64.

Ferek RJ, Reid JS, Hobbs PV, et al. (1998). Emission factors of hydrocarbons, halocarbons, trace gases and particles from biomass burning in Brazil. J Geophys Res 103:32107–18.

Fick Jr R, Paul E, Merrill W, et al. (1984). Alterations in the antibacterial properties of rabbit pulmonary macrophages exposed to wood smoke. Am Rev Respir Dis 129:76–81.

Fine PM, Cass GR, Simoneit BR. (2001). Chemical characterization of fine particle emissions from fireplace combustion of woods grown in the northeastern United States. Environ Sci Technol 35:2665–75.

Fine PM, Cass GR, Simoneit BR. (2002a). Chemical characterization of fine particle emissions from the fireplace combustion of woods grown in the southern United States. Environ Sci Technol 36:1442–51.

Fine PM, Cass GR, Simoneit BR. (2002b). Organic compounds in biomass smoke from residential wood combustion: emissions characterization at a continental scale. J Geophys Res 107:ICC 11-1–9.

Fine PM, Cass GR, Simoneit BR. (2004a). Chemical characterization of fine particle emissions from the fireplace combustion of wood types grown in the midwestern and western United States. Environ Eng Sci 21:387–409.

Fine PM, Cass GR, Simoneit BR. (2004b). Chemical characterization of fine particle emissions from the wood stove combustion of prevalent United States tree species. Environ Eng Sci 21:705–21.

Forchhammer L, Loft S, Roursgaard M, et al. (2012a). Expression of adhesion molecules, monocyte interactions and oxidative stress in human endothelial cells exposed to wood smoke and diesel exhaust particulate matter. Toxicol Lett 209:121–8.

Forchhammer L, Moller P, Riddervold IS, et al. (2012b). Controlled human wood smoke exposure: oxidative stress, inflammation and microvascular function. Part Fibre Toxicol 9:7.

Franzi LM, Bratt JM, Williams KM, Last JA. (2011). Why is particulate matter produced by wildfires toxic to lung macrophages? Toxicol Appl Pharmacol 257:182–8.

Fullerton D, Suseno A, Semple S, et al. (2011). Wood smoke exposure, poverty and impaired lung function in Malawian adults. Int J Tuberc Lung Dis 15:391–8.

Galbraith D, Gross SA, Paustenbach D. (2010). Benzene and human health: a historical review and appraisal of associations with various diseases. Crit Rev Toxicol 40:1–46.

García-Sancho MC, García-García L, Báez-Saldaña R, et al. (2009). Indoor pollution as an occupational risk factor for tuberculosis among women: a population-based, gender oriented, case-control study in southern Mexico. Rev Invest Clin 61:392–8.

Gaskill SE, Fascm PC, Spear A, et al. (2010) Carboxyhemoglobin values in wildland firefighters. Med Sci Sports Exerc 52:769.

Gaughan DM, Cox-Ganser JM, Enright PL, et al. (2008). Acute upper and lower respiratory effects in wildland firefighters. J Occup Environ Med 50:1019–28.

Gaughan DM, Siegel PD, Hughes MD, et al. (2014). Arterial stiffness, oxidative stress, and smoke exposure in wildland firefighters. Am J Ind Med 57:748–56.

Ghelfi E, Rhoden CR, Wellenius GA, et al. (2008). Cardiac oxidative stress and electrophysiological changes in rats exposed to concentrated ambient particles are mediated by TRP-dependent pulmonary reflexes. Toxicol Sci 102:328–36.

Ghio AJ, Soukup JM, Case M, et al. (2012). Exposure to wood smoke particles produces inflammation in healthy volunteers. Occup Environ Med 69:170–5.

Gist GL, Burg JR. (1997). Benzene – a review of the literature from a health effects perspective. Toxicol Ind Health 13:661–714.

Golshan M, Faghihi M, Roushan-Zamir T, et al. (2002). Early effects of burning rice farm residues on respiratory symptoms of villagers in suburbs of Isfahan, Iran. Int J Environ Health Res 12:125–31.

González-García M, Torres-Duque CA, Bustos A, et al. (2012). Bronchial hyperresponsiveness in women with chronic obstructive pulmonary disease related to wood smoke. Int J Chron Obstruct Pulmon Dis 7:367–73.

Gorgun FM, Zhuo M, Singh S, Englander EW. (2014). Neuroglobin mitigates mitochondrial impairments induced by acute inhalation of combustion smoke in the mouse brain. Inhal Toxicol 26:361–9.

Graham JP, Corella-Barud V, Avitia-Diaz R, Gurian P. (2005). The in-home environment and household health: a cross-sectional study of informal urban settlements in northern México. Int J Environ Res Public Health 2:394–402.

Guggisberg M, Hessel PA, Michaelchuk D, Ahmed I. (2003). Respiratory symptoms and exposure to wood smoke in an isolated northern community. Can J Public Health 94:372–6.

Guneser S, Atici A, Alparslan N, Cinaz P. (1994). Effects of indoor environmental factors on respiratory systems of children. J Trop Pediatr 40:114–16.

Guzmán-Grenfell A, Nieto-Velázquez N, Torres-Ramos Y, et al. (2011). Increased platelet and erythrocyte arginase activity in chronic obstructive pulmonary disease associated with tobacco or wood smoke exposure. J Investig Med 59:587–92.

Guzman JA. (2012). Carbon monoxide poisoning. Crit Care Clin 28: 537–48.

Hanigan IC, Johnston FH, Morgan GG. (2008). Vegetation fire smoke, indigenous status and cardio-respiratory hospital admissions in Darwin, Australia, 1996-2005: a time-series study. Environ Health 7:42.

Hanninen OO, Salonen RO, Koistinen K, et al. (2009). Population exposure to fine particles and estimated excess mortality in Finland from an east European wildfire episode. J Expo Sci Environ Epidemiol 19:414–22.

Heil A, Goldammer J. (2001). Smoke-haze pollution: a review of the 1997 episode in southeast Asia. Reg Environ Change 2:24–37.

Hejl AM, Adetona O, Diaz-Sanchez D, et al. (2013). Inflammatory effects of woodsmoke exposure among wildland firefighters working at prescribed burns at the Savannah River Site, SC. J Occup Environ Hyg 10:173–80.

Henderson SB, Brauer M, Macnab YC, Kennedy SM. (2011). Three measures of forest fire smoke exposure and their associations with respiratory and cardiovascular health outcomes in a population-based cohort. Environ Health Perspect 119:1266–71.

Ho C-Y, Kou Y. (2000). Protective and defensive airway reflexes evoked by nasal exposure to wood smoke in anesthetized rats. J Appl Physiol 88:863–70.

Ho C-Y, Kou YR. (2002). Mechanisms of wood smoke-induced increases in nasal airway resistance and reactivity in rats. Eur J Pharmacol 436: 127–34.

Holstius DM, Reid CE, Jesdale BM, Morello-Frosch R. (2012). Birth weight following pregnancy during the 2003 southern California wildfires. Environ Health Perspect 120:1340–5.

Hosgood H, Boffetta P, Greenland S, et al. (2010). In-home coal and wood use and lung cancer risk: a pooled analysis of the international lung cancer consortium. Environ Health Perspect 118: 1743–7.

Hsu TH, Kou YR. (2001). Airway hyperresponsiveness to bronchoconstrictor challenge after wood smoke exposure in guinea pigs. Life Sci 68:2945–56.

Hsu TH, Lai Y-L, Kou YR. (1998a). Acetylcholine and tachykinin receptor antagonists attenuate wood smoke-induced bronchoconstriction in guinea pigs. Eur J Pharmacol 360:175–83.

Hsu TH, Lai Y-L, Kou YR. (1998b). Smoke-induced airway hyperresponsiveness to inhaled wood smoke in guinea pigs: tachykininergic and cholinergic mechanisms. Life Sci 63:1513–24.

Hsu TH, Lai Y-L, Kou YR. (2000). Wood smoke-induced airway hyperreactivity in guinea pigs: time course, and role of leukotrienes and hydroxyl radical. Life Sci 66:971–80.

Hu Y, Odman MT, Chang ME, et al. (2008). Simulation of air quality impacts from prescribed fires on an urban area. Environ Sci Technol 42:3676–82.

Hunter AL, Unosson J, Bosson JA, et al. (2014). Effect of wood smoke exposure on vascular function and thrombus formation in healthy fire fighters. Part Fibre Toxicol 11:62.

Iinuma Y, Brüggemann E, Gnauk T, et al. (2007). Source characterization of biomass burning particles: the combustion of selected European conifers, African hardwood, Savanna grass, and German and Indonesian peat. J Geophys Res 112:D8.

Ingale LT, Dube KJ, Sarode DB, et al. (2013). Monitoring and respiratory health assessment of the population exposed to cooking fuel emissions in a rural area of Jalgaon district, India. Asia Pac J Public Health 25:463–75.

Invernizzi G, Boffi R, Ruprecht A, et al. (2006). Real-time measurement of particulate matter deposition in the lung. Biomarkers 11:221–32.

Ito K, Christensen WF, Eatough DJ, et al. (2006). Pm source apportionment and health effects: 2. An investigation of intermethod variability in associations between source-apportioned fine particle mass and daily mortality in Washington, DC. J Exp Sci Environ Epidemiol 16:300–10.

Jacobs J, Kreutzer R, Smith D. (1997). Rice burning and asthma hospitalizations, Butte County, California, 1983-1992. Environ Health Perspect 105:980–5.

Jacquin L, Michelet P, Brocq FX, et al. (2011). Short-term spirometric changes in wildland firefighters. Am J Ind Med 54:819–25.

Jalaludin B, Smith M, O'Toole B, et al. (2000). Acute effects of bushfires on peak expiratory flow rates in children with wheeze: a time series analysis. Aust NZ J Public Health 24:174–7.

Jansen KL, Larson TV, Koenig JQ, et al. (2005). Associations between health effects and particulate matter and black carbon in subjects with respiratory disease. Environ Health Perspect 113:1741–6.

Johnson A-WB, Osinusi K, Aderele WI, et al. (2008). Etiologic agents and outcome determinants of community-acquired pneumonia in urban children: a hospital-based study. J Natl Med Assoc 100: 370–85.

Johnson A, Aderele W. (1991). The association of household pollutants and socio-economic risk factors with the short-term outcome of acute lower respiratory infections in hospitalized pre-school Nigerian children. Ann Trop Paediatr 12:421–32.

Johnston FH, Bailie RS, Pilotto LS, Hanigan IC. (2007). Ambient biomass smoke and cardio-respiratory hospital admissions in Darwin, Australia. BMC Public Health 7:240.

Johnston FH, Hanigan IC, Henderson SB, Morgan GG. (2013). Evaluation of interventions to reduce air pollution from biomass smoke on mortality in Launceston, Australia: retrospective analysis of daily mortality, 1994–2007. BMJ 346:e8446.

Johnston FH, Kavanagh AM, Bowman D, Scott RK. (2002). Exposure to bushfire smoke and asthma: an ecological study. Med J Austr 176: 535–8.

Johnston FH, Webby RJ, Pilotto LS, et al. (2006). Vegetation fires, particulate air pollution and asthma: a panel study in the Australian monsoon tropics. Int J Environ Health Res 16:391–404.

Johnston FH, Henderson SB, Chen Y, et al. (2012). Estimated global mortality attributable to smoke from landscape fires. Environ Health Perspect 120:695–701.

Kadam YR, Mimansa A, Chavan PV, Gore AD. (2013). Effect of prenatal exposure to kitchen fuel on birth weight. Indian J Community Med 38:212–16.

Karlsson HL, Ljungman AG, Lindbom J, Möller L. (2006). Comparison of genotoxic and inflammatory effects of particles generated by wood combustion, a road simulator and collected from street and subway. Toxicol Lett 165:203–11.

Karr CJ, Demers PA, Koehoorn MW, et al. (2009). Influence of ambient air pollutant sources on clinical encounters for infant bronchiolitis. Am J Respir Crit Care Med 180:995–1001.

Keebaugh AJ, Sioustas C, Pakbin P, et al. (2015). Is atherosclerotic disease associated with organic components of ambient fine particles? Sci Total Environ 533:69–75.

Keywood MD, Ayers GP, Gras JL, et al. (2000). Size distribution and sources of aerosol in Launceston, Australia, during winter 1997. J Air Waste Manag Assoc 50:418–27.

Keywood M, Kanakidou M, Stohl A, et al. (2013). Fire in the air: biomass burning impacts in a changing climate. Crit Rev Environ Sci Technol 43:40–83.

Kido T, Tamagawa E, Bai N, et al. (2011). Particulate matter induces translocation of IL-6 from the lung to the systemic circulation. Am J Respir Cell Mol Biol 44:197–204.

Kocbach A, Herseth JI, Låg M, et al. (2008a). Particles from wood smoke and traffic induce differential pro-inflammatory response patterns in co-cultures. Toxicol Appl Pharmacol 232:317–26.

Kocbach A, Namork E, Schwarze PE. (2008b). Pro-inflammatory potential of wood smoke and traffic-derived particles in a monocytic cell line. Toxicology 247:123–32.

Köksal H, Saygı A, Sarıman N, et al. (2013). Evaluation of clinical and functional parameters in female subjects with biomass smoke exposure. Respir Care 58:424–30.

Kou Y, Lai C, Hsu T, Lin Y. (1997). Involvement of hydroxyl radical in the immediate ventilatory responses to inhaled wood smoke in rats. Respir Physiol 107:1–13.

Kou Y, Wang C, Lai C. (1995). Role of vagal afferents in the acute ventilatory responses to inhaled wood smoke in rats. J Appl Physiol 78:2070–8.

Kristensson A, Rissler J, Löndahl J, et al. (2013). Size-resolved respiratory tract deposition of sub-micrometer aerosol particles in a residential area with wintertime wood combustion. Aerosol Air Qual Res 13:24–35.

Kubátová A, Dronen LC, Picklo MJ, Hawthorne SB. (2006). Midpolarity and nonpolar wood smoke particulate matter fractions deplete glutathione in RAW 264.7 macrophages. Chem Res Toxicol 19: 255–61.

Kunzli N, Avol E, Wu J, et al. (2006). Health effects of the 2003 southern California wildfires on children. Am J Respir Crit Care Med 174: 1221–8.

Kurmi OP, Lam KBH, Ayres JG. (2012). Indoor air pollution and the lung in low- and medium-income countries. Eur Respir J 40:239–54.

Kurmi OP, Sadhra CS, Ayres JG, Sadhra SS. (2014). Tuberculosis risk from exposure to solid fuel smoke: a systematic review and meta-analysis. J Epidemiol Community Health 68:1112–18.

Kurmi OP, Semple S, Simkhada P, et al. (2010). COPD and chronic bronchitis risk of indoor air pollution from solid fuel: a systematic review and meta-analysis. Thorax 65:221–8.

Lai C, Kou Y. (1998a). Stimulation of pulmonary rapidly adapting receptors by inhaled wood smoke in rats. J Physiol 508:597–607.

Lai C, Kou Y. (1998b). Stimulation of vagal pulmonary C fibers by inhaled wood smoke in rats. J Appl Physiol 84:30–6.
Lakshmi P, Virdi NK, Sharma A, et al. (2013). Household air pollution and stillbirths in India: analysis of the DLHS-II national survey. Environ Res 121:17–22.
Lang I, Bruckner T, Triebig G. (2008). Formaldehyde and chemosensory irritation in humans: a controlled human exposure study. Regul Toxicol Pharmacol 50:23–36.
Lee HM, Greeley GH, Englander EW. (2011). Transgenic overexpression of neuroglobin attenuates formation of smoke-inhalation-induced oxidative DNA damage, in vivo, in the mouse brain. Free Radic Biol Med 51:2281–7.
Lee HM, Greeley GH, Herndon DN, et al. (2005a). A rat model of smoke inhalation injury: influence of combustion smoke on gene expression in the brain. Toxicol Appl Pharmacol 208:255–65.
Lee HM, Hallberg LM, Greeley Jr GH, Englander EW. (2010). Differential inhibition of mitochondrial respiratory complexes by inhalation of combustion smoke and carbon monoxide, in vivo, in the rat brain. Inhal Toxicol 22:770–7.
Lee S, Baumann K, Schauer JJ, et al. (2005b). Gaseous and particulate emissions from prescribed burning in Georgia. Environ Sci Technol 39:9049–56.
Lee S, Kim HK, Yan B, et al. (2008a). Diagnosis of aged prescribed burning plumes impacting an urban area. Environ Sci Technol 42: 1438–44.
Lee T-S, Liu Y-J, Tang G-J, et al. (2008b). Wood smoke extract promotes both apoptosis and proliferation in rat alveolar epithelial type II cells: the role of oxidative stress and heme oxygenase-1*. Crit Care Med 36:2597–606.
Leonard SS, Castranova V, Chen BT, et al. (2007). Particle size-dependent radical generation from wildland fire smoke. Toxicology 236:103–13.
Leonard SS, Wang S, Shi X, et al. (2000). Wood smoke particles generate free radicals and cause lipid peroxidation, DNA damage, NFkappaB activation and TNF-alpha release in macrophages. Toxicology 150:147–57.
Li N, Kim S, Wang M, et al. (2002). Use of a stratified oxidative stress model to study the biological effects of ambient concentrated and diesel exhaust particulate matter. Inhal Toxicol 14:459–86.
Li N, Xia T, Nel AE. (2008). The role of oxidative stress in ambient particulate matter-induced lung diseases and its implications in the toxicity of engineered nanoparticles. Free Radic Biol Med 44: 1689–99.
Lin H-H, Ezzati M, Murray M. (2007). Tobacco smoke, indoor air pollution and tuberculosis: a systematic review and meta-analysis. PLoS Med 4:e20.
Lin YS, Ho C-Y, Tang G-J, Kou YR. (2001). Alleviation of wood smoke-induced lung injury by tachykinin receptor antagonist and hydroxyl radical scavenger in guinea pigs. Eur J Pharmacol 425:141–8.
Lin YS, Kou YR. (2000). Acute neurogenic airway plasma exudation and edema induced by inhaled wood smoke in guinea pigs: role of tachykinins and hydroxyl radical. Eur J Pharmacol 394:139–48.
Ling SH, Van Eeden SF. (2009). Particulate matter air pollution exposure: role in the development and exacerbation of chronic obstructive pulmonary disease. Int J Chron Obstruct Pulmon Dis 4:233–43.
Liu D, Tager IB, Balmes JR, Harrison RJ. (1992). The effect of smoke inhalation on lung function and airway responsiveness in wildland fire fighters. Am Rev Respir Dis 146:1469.
Liu P-L, Chen Y-L, Chen Y-H, et al. (2005). Wood smoke extract induces oxidative stress-mediated caspase-independent apoptosis in human lung endothelial cells: role of AIF and EndoG. Am J Physiol Lung Cell Mol Physiol 289:L739–49.
Liu Y, Goodrick SL, Stanturf JA. (2013). Future US wildfire potential trends projected using a dynamically downscaled climate change scenario. Forest Ecol Manage 294:120–35.
Long W, Tate RB, Neuman M, et al. (1998). Respiratory symptoms in a susceptible population due to burning of agricultural residue. Chest 113:351–7.
Mahalanabis D, Gupta S, Paul D, et al. (2002). Risk factors for pneumonia in infants and young children and the role of solid fuel for cooking: a case-control study. Epidemiol Infect 129:65–71.
Mar TF, Ito K, Koenig JQ, et al. (2006). PM source apportionment and health effects. 3. Investigation of inter-method variations in associations between estimated source contributions of $PM_{2.5}$ and daily mortality in Phoenix, AZ. J Exp Sci Environ Epidemiol 16: 311–20.
Martin KL, Hanigan IC, Morgan GG, et al. (2013). Air pollution from bushfires and their association with hospital admissions in Sydney, Newcastle and Wollongong, Australia 1994–2007. Aust N Z J Public Health 37:238–43.
Mccracken J, Smith KR, Stone P, et al. (2011). Intervention to lower household wood smoke exposure in Guatemala reduces ST-segment depression on electrocardiograms. Environ Health Perspect 119: 1562–8.
Mccracken JP, Smith KR, Díaz A, et al. (2007). Chimney stove intervention to reduce long-term wood smoke exposure lowers blood pressure among Guatemalan women. Environ Health Perspect 115: 996–1001.
McMahon CK, Bush PB. (1992). Forest worker exposure to airborne herbicide residues in smoke from prescribed fires in the southern United States. Am Ind Hyg Assoc J 53:265–72.
Mcgowan J, Hider P, Chacko E, Town G. (2002). Particulate air pollution and hospital admissions in Christchurch, New Zealand. Aust N Z J Public Health 26:23–9.
Mengersen K, Morawska L, Wang H, et al. (2011). The effect of housing characteristics and occupant activities on the respiratory health of women and children in Lao PDR. Sci Total Environ 409: 1378–84.
Migliaccio CT, Kobos E, King QO, et al. (2013). Adverse effects of wood smoke PM(2.5) exposure on macrophage functions. Inhal Toxicol 25:67–76.
Mills NL, Donaldson K, Hadoke PW, et al. (2009). Adverse cardiovascular effects of air pollution. Nat Clin Pract Cardiovasc Med 6:36–44.
Mirabelli MC, Künzli N, Avol E, et al. (2009). Respiratory symptoms following wildfire smoke exposure: airway size as a susceptibility factor. Epidemiology 20:451–9.
Miranda AI, Martins V, Cascão P, et al. (2012). Wildland smoke exposure values and exhaled breath indicators in firefighters. J Toxicol Environ Health Part A 75:831–43.
Molto J, Font R, Galvez A, et al. (2010). Emissions of polychlorodibenzodioxin/furan (PCDD/Fs), dioxin-like polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), and volatile organic compounds produced in the combustion of pine needles and cones. Energy Fuels 24:1030–6.
Montano M, Beccerril C, Ruiz V, et al. (2004). Matrix metalloproteinases activity in COPD associated with wood smoke. Chest J 125: 466–72.
Moore D, Copes R, Fisk R, et al. (2006). Population health effects of air quality changes due to forest fires in British Columbia in 2003: estimates from physician-visit billing data. Can J Public Health 97: 105–8.
Moran-Mendoza O, Pérez-Padilla J, Salazar-Flores M, Vazquez-Alfaro F. (2008). Wood smoke-associated lung disease: a clinical, functional, radiological and pathological description. Int J Tuberc Lung Dis 12:1092–8.
Morgan G, Sheppeard V, Khalaj B, et al. (2010). Effects of bushfire smoke on daily mortality and hospital admissions in Sydney, Australia. Epidemiology 21:47–55.
Mott JA, Mannino DM, Alverson CJ, et al. (2005). Cardiorespiratory hospitalizations associated with smoke exposure during the 1997 southeast Asian forest fires. Int J Hyg Environ Health 208: 75–85.
Mott JA, Meyer P, Mannino D, et al. (2002). Wildland forest fire smoke: health effects and intervention evaluation, Hoopa, California, 1999. West J Med 176:157.
Myatt TA, Vincent MS, Kobzik L, et al. (2011). Markers of inflammation in alveolar cells exposed to fine particulate matter from prescribed fires and urban air. J Occup Environ Med 53:1110–14.
Naeher LP, Brauer M, Lipsett M, et al. (2007). Woodsmoke health effects: a review. Inhal Toxicol 19:67–106.
Nichols JL, Owens EO, Dutton SJ, Luben TJ. (2013). Systematic review of the effects of black carbon on cardiovascular disease among individuals with pre-existing disease. Int J Public Health 58: 707–24.
Noonan CW, Ward TJ, Navidi W, Sheppard L. (2012). A rural community intervention targeting biomass combustion sources: effects on air quality and reporting of children's respiratory outcomes. Occup Environ Med 69:354–60.

Ostro BD, Lipsett MJ, Mann JK, et al. (1994). Indoor air pollution and asthma. Results from a panel study. Am J Respir Crit Care Med 149: 1400–6.

Painschab MS, Davila-Roman VG, Gilman RH, et al. (2013). Chronic exposure to biomass fuel is associated with increased carotid artery intima-media thickness and a higher prevalence of atherosclerotic plaque. Heart 99:984–91.

Papi A, Luppi F, Franco F, Fabbri LM. (2006). Pathophysiology of exacerbations of chronic obstructive pulmonary disease. Proc Am Thorac Soc 3:245–51.

Park MS, Cancio LC, Jordan BS, et al. (2004). Assessment of oxidative stress in lungs from sheep after inhalation of wood smoke. Toxicology 195:97–112.

Perez-Padilla R, Perez-Guzman C, Baez-Saldana R, Torres-Cruz A. (2001). Cooking with biomass stoves and tuberculosis: a case control study. Int J Tuberc Lung Dis 5:441–7.

Perng D-W, Chang T-M, Wang J-Y, et al. (2013). Inflammatory role of AMP-activated protein kinase signaling in an experimental model of toxic smoke inhalation injury*. Crit Care Med 41:120–32.

Phuleria HC, Fine PM, Zhu Y, Sioutas C. (2005). Air quality impacts of the october 2003 southern California wildfires. J Geophys Res 110: D07S20.

Po JY, Fitzgerald JM, Carlsten C. (2011). Respiratory disease associated with solid biomass fuel exposure in rural women and children: systematic review and meta-analysis. Thorax 66:232–9.

Poloniecki JD, Atkinson RW, De Leon AP, Anderson HR. (1997). Daily time series for cardiovascular hospital admissions and previous day's air pollution in London, UK. Occup Environ Med 54: 535–40.

Pope DP, Mishra V, Thompson L, et al. (2010). Risk of low birth weight and stillbirth associated with indoor air pollution from solid fuel use in developing countries. Epidemiology 32:70–81.

Portin H, Mielonen T, Leskinen A, et al. (2012). Biomass burning aerosols observed in Eastern Finland during the Russian wildfires in summer 2010–part 1: in-situ aerosol characterization. Atmos Environ 47:269–78.

Quinn DK, Mcgahee SM, Politte LC, et al. (2009). Complications of carbon monoxide poisoning: a case discussion and review of the literature. Prim Care Companion J Clin Psychiatry 11:74–9.

Radeloff VC, Hammer RB, Stewart SI, et al. (2005). The wildland-urban interface in the United States. Ecol Appl 15:799–805.

Ramalho R, Soares R, Couto N, Moreira A. (2011). Tachykinin receptors antagonism for asthma: a systematic review. BMC Pulm Med 11:41.

Ramanathan V, Carmichael G. (2008). Global and regional climate changes due to black carbon. Nature Geosci 1:221–7.

Ramos C, Cisneros J, Gonzalez-Avila G, et al. (2009). Increase of matrix metalloproteinases in woodsmoke-induced lung emphysema in guinea pigs. Inhal Toxicol 21:119–32.

Ramos C, Pedraza-Chaverri J, Becerril C, et al. (2013). Oxidative stress and lung injury induced by short-term exposure to wood smoke in guinea pigs. Toxicol Mech Methods 23:711–22.

Rappold AG, Stone SL, Cascio WE, et al. (2011). Peat bog wildfire smoke exposure in rural north Carolina is associated with cardiopulmonary emergency department visits assessed through syndromic surveillance. Environ Health Perspect 119:1415–20.

Raub J. 1999. Carbon monoxide. Geneva, Switzerland: World Health Organization.

Raub JA, Mathieu-Nolf M, Hampson NB, Thom SR. (2000). Carbon monoxide poisoning – a public health perspective. Toxicology 145: 1–14.

Reinhardt TE, Ottmar RD. (2004). Baseline measurements of smoke exposure among wildland firefighters. J Occup Environ Hyg 1:593–606.

Reisen F, Brown SK. (2009). Australian firefighters' exposure to air toxics during bushfire burns of autumn 2005 and 2006. Environ Int 35: 342–52.

Reisen F, Hansen D, Meyer CM. (2011). Exposure to bushfire smoke during prescribed burns and wildfires: firefighters' exposure risks and options. Environ Int 37:314–21.

Riddervold IS, Bønløkke JH, Olin A-C, et al. (2012). Effects of wood smoke particles from wood-burning stoves on the respiratory health of atopic humans. Part Fibre Toxicol 9:12.

Rinne ST, Rodas EJ, Bender BS, et al. (2006). Relationship of pulmonary function among women and children to indoor air pollution from biomass use in rural Ecuador. Respir Med 100: 1208–15.

Riojas-Rodríguez H, Romano-Riquer P, Santos-Burgoa C, Smith KR. (2001). Household firewood use and the health of children and women of Indian communities in Chiapas, Mexico. Int J Occup Environ Health 7:44–53.

Robinson MS, Zhao M, Zack L, et al. (2011). Characterization of $PM_{2.5}$ collected during broadcast and slash-pile prescribed burns of predominately ponderosa pine forests in Northern Arizona. Atmos Environ 45:2087–94.

Roemer E, Anton H, Kindt R. (1993). Cell proliferation in the respiratory tract of the rat after acute inhalation of formaldehyde or acrolein. J Appl Toxicol 13:103–7.

Romieu I, Riojas-Rodríguez H, Marrón-Mares AT, et al. (2009). Improved biomass stove intervention in rural Mexico: impact on the respiratory health of women. Am J Respir Crit Care Med 180:649–56.

Rothman N, Ford PD, Baser ME, et al. (1991). Pulmonary function and respiratory symptoms in wildland firefighters. J Occup Med 33: 1163–7.

Saha A, Rao NM, Kulkarni P, et al. (2005). Pulmonary function and fuel use: a population survey. Respir Res 6:127.

Samoli E, Aga E, Touloumi G, et al. (2006). Short-term effects of nitrogen dioxide on mortality: an analysis within the APHEA project. Eur Respir J 27:1129–38.

Samuelsen M, Cecilie Nygaard U, Løvik M. (2009). Particles from wood smoke and road traffic differently affect the innate immune system of the lung. Inhal Toxicol 21:943–51.

Sandoval J, Salas J, Martinez-Guerra ML, et al. (1993). Pulmonary arterial hypertension and cor pulmonale associated with chronic domestic woodsmoke inhalation. Chest J 103:12–20.

Sanhueza PA, Torreblanca MA, Diaz-Robles LA, et al. (2009). Particulate air pollution and health effects for cardiovascular and respiratory causes in Temuco, Chile: a wood-smoke-polluted urban area. J Air Waste Manag Assoc 59:1481–8.

Sarnat JA, Marmur A, Klein M, et al. (2008). Fine particle sources and cardiorespiratory morbidity: an application of chemical mass balance and factor analytical source-apportionment methods. Environ Health Perspect 116:559–66.

Sastry N. (2002). Forest fires, air pollution, and mortality in southeast Asia. Demography 39:1–23.

Schei MA, Hessen JO, Smith KR, et al. (2004). Childhood asthma and indoor woodsmoke from cooking in Guatemala. J Exp Sci Environ Epidemiol 14:S110–17.

Schmidl C, Marr IL, Caseiro A, et al. (2008). Chemical characterisation of fine particle emissions from wood stove combustion of common woods growing in mid-European Alpine regions. Atmos Environ 42: 126–41.

Schreuder AB, Larson TV, Sheppard L, Claiborn CS. (2006). Ambient woodsmoke and associated respiratory emergency department visits in Spokane, Washington. Int J Occup Environ Health 12:147–53.

Schwarze P, Øvrevik J, Låg M, et al. (2006). Particulate matter properties and health effects: consistency of epidemiological and toxicological studies. Hum Exp Toxicol 25:559–79.

Sehlstedt M, Dove R, Boman C, et al. (2010). Antioxidant airway responses following experimental exposure to wood smoke in man. Part Fibre Toxicol 7:21.

Serra A, Mocci F, Randaccio FS. (1996). Pulmonary function in Sardinian fire fighters. Am J Ind Med 30:78–82.

Shapiro D, Deering-Rice CE, Romero EG, et al. (2013). Activation of transient receptor potential ankyrin-1 (TRPA1) in lung cells by wood smoke particulate material. Chem Res Toxicol 26:750–8.

Siddiqui AR, Gold EB, Yang X, et al. (2008). Prenatal exposure to wood fuel smoke and low birth weight. Environ Health Perspect 116:543–9.

Sillanpää M, Saarikoski S, Hillamo R, et al. (2005). Chemical composition, mass size distribution and source analysis of long-range transported wildfire smokes in Helsinki. Sci Total Environ 350: 119–35.

Silvestrine RA, Soares-Filho BS, Nepstad D, et al. (2011). Simulating fire regimes in the Amazon in response to climate change and deforestation. Ecol Appl 21:1573–90.

Simpson IJ, Akagi S, Barletta B, et al. (2011). Boreal forest fire emissions in fresh canadian smoke plumes: C 1-c 10 volatile organic compounds (VOCs), CO_2, CO, NO_2, NO, HCN and CH_3 and CN. Atmos Chem Phys 11:6445–63.

Slaughter JC, Koenig JQ, Reinhardt TE. (2004). Association between lung function and exposure to smoke among firefighters at prescribed burns. J Occup Environ Hyg 1:45–9.

Smith-Sivertsen T, Diaz E, Pope D, et al. (2009). Effect of reducing indoor air pollution on women's respiratory symptoms and lung function: the RESPIRE randomized trial, Guatemala. Am J Epidemiol 170:211–20.

Smith KR, Mccracken JP, Weber MW, et al. (2011). Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomised controlled trial. Lancet 378:1717–26.

Smith MA, Jalaludin B, Byles JE, et al. (1996). Asthma presentations to emergency departments in Western Sydney during the January 1994 bushfires. Int J Epidemiol 25:1227–36.

Stephens SL, Ruth LW. (2005). Federal forest-fire policy in the United States. Ecol Appl 15:532–42.

Stockfelt L, Sallsten G, Almerud P, et al. (2013). Short-term chamber exposure to low doses of two kinds of wood smoke does not induce systemic inflammation, coagulation or oxidative stress in healthy humans. Inhal Toxicol 25:417–25.

Stockfelt L, Sallsten G, Olin A-C, et al. (2012). Effects on airways of short-term exposure to two kinds of wood smoke in a chamber study of healthy humans. Inhal Toxicol 24:47–59.

Sumpter C, Chandramohan D. (2013). Systematic review and meta-analysis of the associations between indoor air pollution and tuberculosis. Trop Med Int Health 18:101–8.

Sutherland ER, Make BJ, Vedal S, et al. (2005). Wildfire smoke and respiratory symptoms in patients with chronic obstructive pulmonary disease. J Allergy Clin Immunol 115:420–2.

Swiston JR, Davidson W, Attridge S, et al. (2008). Wood smoke exposure induces a pulmonary and systemic inflammatory response in firefighters. Eur Respir J 32:129–38.

Tan WC, Qiu D, Liam BL, et al. (2000). The human bone marrow response to acute air pollution caused by forest fires. Am J Respir Crit Care Med 161:1213–17.

Taylor ET, Nakai S. (2012). Prevalence of acute respiratory infections in women and children in western Sierra Leone due to smoke from wood and charcoal stoves. Int J Environ Res Public Health 9:2252–65.

Tesfaigzi Y, Singh SP, Foster JE, et al. (2002). Health effects of subchronic exposure to low levels of wood smoke in rats. Toxicol Sci 65:115–25.

Tham R, Erbas B, Akram M, et al. (2009). The impact of smoke on respiratory hospital outcomes during the 2002-2003 bushfire season, Victoria, Australia. Respirology 14:69–75.

Thompson LM, Bruce N, Eskenazi B, et al. (2011). Impact of reduced maternal exposures to wood smoke from an introduced chimney stove on newborn birth weight in rural Guatemala. Environ Health Perspect 119:1489.

Torigoe K, Hasegawa S, Numata O, et al. (2000). Influence of emission from rice straw burning on bronchial asthma in children. Pediatr Int 42:143–50.

Tosca MG, Randerson JT, Zender CS, et al. (2011). Dynamics of fire plumes and smoke clouds associated with peat and deforestation fires in Indonesia. J Geophys Res 116:D08207.

Triche EW, Belanger K, Beckett W, et al. (2002). Infant respiratory symptoms associated with indoor heating sources. Am J Respir Crit Care Med 166:1105–11.

Triche EW, Belanger K, Bracken MB, et al. (2005). Indoor heating sources and respiratory symptoms in nonsmoking women. Epidemiology 16:377–84.

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 49:775–80.

Unosson J, Blomberg A, Sandström T, et al. (2013). Exposure to wood smoke increases arterial stiffness and decreases heart rate variability in humans. Part Fibre Toxicol 10:20.

Urbanski S. (2014). Wildland fire emissions, carbon, and climate: emission factors. For Ecol Manage 317:51–60.

Van Eeden SF, Tan WC, Suwa T, et al. (2001). Cytokines involved in the systemic inflammatory response induced by exposure to particulate matter air pollutants (PM_{10}). Am J Respir Crit Care Med 164:826–30.

Vedal S, Dutton SJ. (2006). Wildfire air pollution and daily mortality in a large urban area. Environ Res 102:29–35.

Verma V, Polidori A, Schauer JJ, et al. (2009). Physicochemical and toxicological profiles of particulate matter in Los Angeles during the October 2007 southern California wildfires. Environ Sci Technol 43: 954–60.

WHO. 2006. Air quality guidelines: global update 2005: particulate matter, ozone, nitrogen dioxide, and sulfur dioxide. Geneva: World Health Organization.

Wang H, Zhu B, Shen L, Kang H. (2012). Size distributions of aerosol and water-soluble ions in Nanjing during a crop residual burning event. J Environ Sci 24:1457–65.

Ward TJ, Lincoln E. (2006). Concentrations of PM(2.5)-associated OC, EC, and PCDD/Fs measured during the 2003 wildfire season in Missoula, Montana. Environ Monit Assess 115:39–50.

Wegesser TC, Franzi LM, Mitloehner FM, et al. (2010). Lung antioxidant and cytokine responses to coarse and fine particulate matter from the great California wildfires of 2008. Inhal Toxicol 22: 561–70.

Wegesser TC, Pinkerton KE, Last JA. (2009). California wildfires of 2008: coarse and fine particulate matter toxicity. Environ Health Perspect 117:893–7.

Williams KM, Franzi LM, Last JA. (2013). Cell-specific oxidative stress and cytotoxicity after wildfire coarse particulate matter instillation into mouse lung. Toxicol Appl Pharmacol 266:48–55.

Wolf SJ, Lavonas EJ, Sloan EP, Jagoda AS. (2008). Clinical policy: critical issues in the management of adult patients presenting to the emergency department with acute carbon monoxide poisoning. J Emerg Nurs 34:e19–32.

Wong LN, Aung H, Lamé M, et al. (2011). Fine particulate matter from urban ambient and wildfire sources from California's San Joaquin valley initiate differential inflammatory, oxidative stress, and xenobiotic responses in human bronchial epithelial cells. Toxicol In Vitro 25:1895–905.

Wu J, Winer AM, Delfino RJ. (2006). Exposure assessment of particulate matter air pollution before, during, and after the 2003 southern California wildfires. Atm Environ 40:3333–48.

Wylie BJ, Coull BA, Hamer DH, et al. (2014). Impact of biomass fuels on pregnancy outcomes in central east India. Environ Health 13:1.

Xiao GG, Wang M, Li N, et al. (2003). Use of proteomics to demonstrate a hierarchical oxidative stress response to diesel exhaust particle chemicals in a macrophage cell line. J Biol Chem 278: 50781–90.

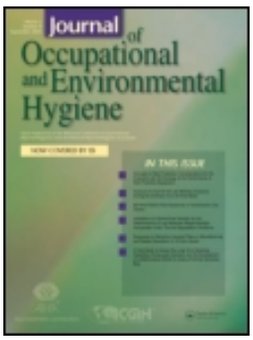
Yoschenko VI, Kashparov VA, Prostak VP, et al. (2006). Resuspension and redistribution of radionuclides during grasslands and forest fires in the Chernobyl exclusion zone: part I. Fire experiments. J Environ Radioact 86:143–63.

Yokelson RJ, Burling IR, Gilman J, et al. (2013). Coupling field and laboratory measurements to estimate the emission factors of identified and unidentified trace gases for prescribed fires. Atmos Chem Phys 13:89–116.

Zelikoff JT, Chen LC, Cohen MD, Schlesinger RB. (2002). The toxicology of inhaled woodsmoke. J Toxicol Environ Health B Crit Rev 5:269–82.

Zhu F, Qiu X, Wang J, et al. (2012). A rat model of smoke inhalation injury. Inhal Toxicol 24:356–64.

Zou Y, Li S, Zou W, et al. (2014). Upregulation of gelatinases and epithelial-mesenchymal transition in small airway remodeling associated with chronic exposure to wood smoke. PLoS One 9:e96708.



Journal of Occupational and Environmental Hygiene

ISSN: 1545-9624 (Print) 1545-9632 (Online) Journal homepage: https://www.tandfonline.com/loi/uoeh20

Personal $PM_{2.5}$ Exposure Among Wildland Firefighters Working at Prescribed Forest Burns in Southeastern United States

Olorunfemi Adetona , Kevin Dunn , Daniel B. Hall , Gary Achtemeier , Allison Stock & Luke P. Naeher

To cite this article: Olorunfemi Adetona , Kevin Dunn , Daniel B. Hall , Gary Achtemeier , Allison Stock & Luke P. Naeher (2011) Personal $PM_{2.5}$ Exposure Among Wildland Firefighters Working at Prescribed Forest Burns in Southeastern United States, Journal of Occupational and Environmental Hygiene, 8:8, 503-511, DOI: 10.1080/15459624.2011.595257

To link to this article: https://doi.org/10.1080/15459624.2011.595257

Published online: 15 Jul 2011.

Journal of Occupational and Environmental Hygiene, 8: 503–511
ISSN: 1545-9624 print / 1545-9632 online
DOI: 10.1080/15459624.2011.595257

Personal $PM_{2.5}$ Exposure Among Wildland Firefighters Working at Prescribed Forest Burns in Southeastern United States

Olorunfemi Adetona,[1] Kevin Dunn,[2] Daniel B. Hall,[3] Gary Achtemeier,[4] Allison Stock,[2] and Luke P. Naeher[1]

[1]The University of Georgia, College of Public Health, Department of Environmental Health Science, Athens, Georgia
[2]National Center for Environmental Health, Centers for Disease Control, Atlanta, Georgia
[3]The University of Georgia, Franklin College of Arts and Sciences, Department of Statistics, Athens, Georgia
[4]United States Department of Agriculture, Forest Service, Athens, Georgia

This study investigated occupational exposure to wood and vegetative smoke in a group of 28 forest firefighters at prescribed forest burns in a southeastern U.S. forest during the winters of 2003–2005. During burn activities, 203 individual person-day $PM_{2.5}$ and 149 individual person-day CO samples were collected; during non-burn activities, 37 person-day $PM_{2.5}$ samples were collected as controls. Time-activity diaries and post-work shift questionnaires were administered to identify factors influencing smoke exposure and to determine how accurately the firefighters' qualitative assessment estimated their personal level of smoke exposure with discrete responses: "none" or "very little," "low," "moderate," "high," and "very high." An average of 6.7 firefighters were monitored per burn, with samples collected on 30 burn days and 7 non-burn days. Size of burn plots ranged from 1–2745 acres (avg = 687.8). Duration of work shift ranged from 6.8–19.4 hr (avg = 10.3 hr) on burn days. Concentration of $PM_{2.5}$ ranged from 5.9–2673 $\mu g/m^3$ on burn days. Geometric mean $PM_{2.5}$ exposure was 280 $\mu g/m^3$ (95% CL = 140, 557 $\mu g/m^3$, n = 177) for burn day samples, and 16 $\mu g/m^3$ (95% CL = 10, 26 $\mu g/m^3$, n = 35) on non-burn days. Average measured $PM_{2.5}$ differed across levels of the firefighters' categorical self-assessments of exposure ($p < 0.0001$): none to very little = 120 $\mu g/m^3$ (95% CL = 71, 203 $\mu g/m^3$) and high to very high = 664 $\mu g/m^3$ (95% CL = 373, 1185 $\mu g/m^3$); $p < 0.0001$ on burn days). Time-weighted average $PM_{2.5}$ and personal CO averaged over the run times of $PM_{2.5}$ pumps were correlated (correlation coefficient estimate, r = 0.79; CLs: 0.72, 0.85). Overall occupational exposures to particulate matter were low, but results indicate that exposure could exceed the ACGIH®-recommended threshold limit value of 3 mg/m^3 for respirable particulate matter in a few extreme situations. Self-assessed exposure levels agreed with measured concentrations of $PM_{2.5}$. Correlation analysis shows that either $PM_{2.5}$ or CO could be used as a surrogate measure of exposure to woodsmoke at prescribed burns.

Keywords exposure, firefighters, particulate matter, prescribed burn, wildland, woodsmoke

Correspondence to: Luke P. Naeher, University of Georgia, EHS Department, EHS Building, Athens, GA 30602; e-mail: lnaeher@gmail.com.

The findings and conclusions in this article are those of the author(s) and do not necessarily represent the views of the Centers for Disease Control and Prevention (CDC). Mention of company names or products does not constitute endorsement by the CDC.

INTRODUCTION

Wildland firefighters are primarily responsible for wildfire suppression in wildlands, including forests, grasslands, and brush, but also engage in prescribed burning. Prescribed burns, as opposed to wildfires, are intentionally set by firefighters and are used as a land management tool for improving forage value of the forests, and reducing wildfire hazard and competing vegetation.[1] They have become such a mandatory land management practice that as much as 6 to 8 million acres of land are treated with prescribed burns by land managers each year in the southern United States alone,[2] and it is estimated that tens of thousands of firefighters across the country work at these burns annually.[3]

Although very careful planning always precedes prescribed burns, wildland firefighters can be exposed to high levels of contaminants in woodsmoke. These include carbon monoxide (CO), carbon dioxide (CO_2), nitrogen oxides,

respirable particulate matter (RPM), total suspended particulates (TSP), polycyclic aromatic hydrocarbons (PAH), benzene, aldehydes, and others.[4,5] Carbon monoxide, RPM, TSP, and aldehydes have been identified as the chief woodsmoke exposure hazards among firefighters.[4] Firefighters working at prescribed burns often work extended shifts (up to 18 hr) while engaged in hard physical labor[4] and wearing no respiratory protection. Physical labor increases minute ventilation and total exposure of the respiratory tract to particles, gases, and vapors. Therefore, firefighters are potentially at risk of serious acute and chronic health effects. Health effects that have been associated with occupational exposure to woodsmoke among wildland firefighters include reduced lung function and pulmonary and systemic inflammation.[6,7]

Studies of exposures among wildland firefighters have been conducted mainly in the western United States, which has different vegetation and weather characteristics compared with other parts of the country. These studies show exposure to particulate matter and CO could exceed occupational health standards, and that exposures were higher among firefighters working at prescribed burns compared with those working at wildfires.[4,8,9] Although conducted in a completely different environment, a study of exposure to vegetative smoke from bushfire at prescribed burns in Australia also points to the possibility of elevated exposure to RPM among wildland firefighters.[10] We assessed occupational woodsmoke exposure in wildland firefighters working at prescribed burns in a southeastern U.S. forest during the dormant (winter) burn seasons of 2003–2005. The objective of this study was to examine the association between particulate matter with median aerodynamic diameter of 2.5 μm ($PM_{2.5}$) and duration and sizes of burns, job tasks, and weather variables to identify the factors that influence exposure. We also assessed whether the firefighters could qualitatively estimate their level of exposure.

METHODS

Study Location and Population

This study was conducted at the Savannah River Site (SRS), a 198,000-acre National Environmental Research Park located in the southeastern coastal area of the United States. The U.S. Department of Agriculture Forestry Service (USFS) manages the complex's natural resources. The forest is composed of 31% hardwood or mixed pine hardwood, and 69% pine. USFS fire personnel apply prescribed burns to approximately 15,000 to 18,000 acres annually to restore the native longleaf pine/savannah communities and wetland on the site.[11] A total of 29 Forest Service firefighters working at prescribed burns participated in the study during the winters of 2003 to 2005. The group included 25 men and 4 women between 21–46 years (average: 29.8; standard deviation: 6.3) who had worked an average of 7.5 years as firefighters at the time of recruitment. Participation in the study was voluntary. A consent form was signed after the study was explained and a firefighter had agreed to participate. The study protocol was reviewed and approved by the University of Georgia Institutional Review Board for the inclusion of human subjects.

Exposure Assessment: Personal $PM_{2.5}$ and Carbon Monoxide Sampling

Full-shift personal $PM_{2.5}$ firefighter exposures were measured during prescribed burns (burn day) and on several days when firefighters did not work at burns (controls). A total of 240 samples (6.5 per day) were collected during the study, with 203 on prescribed burn days (6.7 per day). The samples were collected using Air Check Model 224-PCXR pump (SKC, Inc., Eighty Four, Pa.) attached to GK2.05 KTL Respirable/Thoracic cyclone (BGI International, Waltham, Mass.). Particulates were collected on Gelman 37-mm Teflo filter (Pall Corp., Ann Arbor, Mich.) that was loaded into the cyclone. The filter had a 100% PTFE (polytetraflouroethylene) Teflon membrane with a 2.0 μm pore size and a polymethylpentene support ring. The system is designed to have a 50% aerodynamic cutoff point of 2.5 μm.[12] Pre- and post-sampling flow rates of the pumps were measured with a Dry Cal DC-Lite Model DCL20K (Bios International, Butler, N.J.). The flow rate for the sampling unit was set at 4.0 L/min. $PM_{2.5}$ was measured in the breathing zone with the pumps attached to each firefighter's gear pack. In all, 149 real-time person-shift CO samples were collected on 19 burn days from 20 firefighters during the 2004 and 2005 burn seasons. CO samples were not collected on non-burn days. Real-time CO was measured using Pac III single-gas monitors (Draeger Safety Inc., Pittsburgh, Pa.) outfitted with CO sensors and calibrated with a 200 ppm CO certified gas standard (Calgaz, Air Liquide America Corp., Cambridge, Mass.) prior to the start of the study. Subsequently, Draeger CO monitors were zeroed with ambient air at the forest station at the beginning of each shift, and response was checked with 200 ppm calibration gas at the end of each shift. $PM_{2.5}$ samples were collected in 2003–2005, while CO was measured in 2004 and 2005.

Exposure Assessment: Questionnaire and Time-Activity Diary

A post-shift questionnaire was administered daily to the firefighters to collect data on burn characteristics, tobacco smoke exposure, and self-reported qualitative estimation of woodsmoke exposure: whether their exposure at the prescribed burns was "none to very little, low, medium, medium to high, or very high." A daily activity diary administered alongside the questionnaire was used to determine the tasks and schedule of the firefighters during their work shifts. Possible job tasks included holding, lighting, mop-up, and other activities that do not belong to these major groupings. Briefly, holding involves the maintenance of fire within boundary lines, mop-up entails the extinguishing of smoldering fire after the major burning phase, and ignition is the fire lighting process.

$PM_{2.5}$ Gravimetric Analysis

The PTFE filters were packed and stored in a refrigerator (approximately –4C) until shipment on dry ice to the University of Georgia. The filters were stored in a climate-controlled lab for a minimum of 48 hours before they were weighed pre- and post-sample collection. Both weights were measured twice with a Cahn C-35 microbalance with a sensitivity of ± 1 μg following the guidelines set in the U.S. Environmental Protection Agency's (USEPA) standard operating procedures.[13] The weight of the $PM_{2.5}$ collected on the filter was determined by subtracting the average pre-weight of the filter from its average post-weight. Adjustments were made for minor variations in temperature, barometric pressure, and humidity for all the pre- and post-weights. The time-weighted average (TWA) particulate matter concentration was calculated as the amount of $PM_{2.5}$ collected per cubic meter (m^3) of air. Field blank concentrations were subtracted from each sample to determine the final $PM_{2.5}$ concentrations.

Statistical Analysis

All analyses were done in SAS version 9.1 (SAS Institute, Inc., Cary, N.C.).

Linear mixed-effect models were used to analyze the effect of various factors on $PM_{2.5}$ exposure. A plot of residuals using the untransformed TWA $PM_{2.5}$ concentrations revealed that the constant variance assumption was not satisfied, so $PM_{2.5}$ data were log transformed before model fitting. Firefighter tasks were included in the model as dichotomous variables. Zero was assigned to a task on control days or on burn days when the firefighter had spent less than 75% of total work time on the task; 1 was assigned on burn days when at least 75% of total work time was spent working on the task. The model included terms for plot size, wind speed, shift length (all of which were centered on their means), dichotomous variables for tasks, and the interactions between the tasks and the other variables. Interaction terms were excluded for tasks that were done on very few occasions. In addition, random subject effects were included in the model to account for longitudinal within-subject correlation among the data, and random effects for the date of sample collection were included to account for possible heterogeneity in meteorological and burn conditions from day to day.

A mixed-effect model was also used to analyze how well firefighter estimation predicted actual exposure. Self-reported measures of exposure were classified as 1 to 4 depending on the subject's response in the questionnaire regarding his/her perceived level of exposure, with 1 being "none to very little," 2 being low, 3 being moderate, and 4 being "high" or "very high." Exposures classified as high and very high were collapsed into one new category because of the small sample sizes in these categories.

Finally, it was desired to measure the correlation between $PM_{2.5}$ and CO and to test whether this correlation was equal to 0. This task is complicated by the presence of longitudinal correlation within this sample from repeated measures on the subjects and because of day-to-day heterogeneity. These features preclude a simple correlation analysis. Instead, inference on the correlation between these variables was performed by fitting a bivariate linear mixed-effect model to $PM_{2.5}$ and CO simultaneously, in which random subject-specific effects and random sampling date effects for each response variable were included, and contemporaneous correlation between the two response variables was allowed and estimated.

RESULTS

In total, 240 individual $PM_{2.5}$ work shift samples were collected over the 3-year period: 203 of these were collected on days when prescribed burns were done. Thirty-seven non-burn activity samples were collected as controls from subjects working away from burns: 35 were collected on non-burn days, 2 of which were from subjects carrying out high exposure fire mop-up duties. The other two control samples were collected on a burn day from firefighters who did not work at prescribed burns. In all, 28 samples were excluded from the analyses, leaving 177 burn day and 35 non-burn day samples. Seven of the burn day samples were excluded because they were collected with pumps having stop flows more than 5% below or above the calibrated volume of 4 L/min. Sixteen burn day samples were compromised because of a problem with the cyclone, pump flow faults, or torn filters, and were also excluded. Two non-burn day samples were excluded because they were collected during high exposure fire mop-up duties. In addition, three samples collected on burn days were not used in the models because data were missing for the acreage of burn the firefighters conducted. The average duration of work shift was 10.3 hr (range = 6.8 to 19.4 hr) on burn days and 9.3 hr (range = 7.0 to 11.5 hr) on non-burn days. Samples were collected on 30 burn days with an average of 6.7 firefighters monitored per burn. The size of burn plots ranged from 1 to 2745 acres (avg = 697). Seven non-burn (control) days were monitored. The difference between average exposures on burn and non-burn days was significant. The geometric mean $PM_{2.5}$ exposure calculated from a linear mixed-effect model adjusted for firefighter task, wind speed, length of work shift, and size of burn was 280 $\mu g/m^3$ (95% CL = 140, 557 $\mu g/m^3$, n = 177) for burn day samples, and 16 $\mu g/m^3$ (95% CL = 10, 26 $\mu g/m^3$, n = 35) for non-burn day samples (Table I). The unadjusted arithmetic and geometric means by year and for all samples are also presented in Table I. Overall, $PM_{2.5}$ exposure ranged from 5.9 to 2673 $\mu g/m^3$, and there was no difference in exposure across the 3 years for all samples and neither for burn or non-burn day samples alone. A plot of the cumulative frequency distribution of $PM_{2.5}$ exposure is presented in Figure 1.

TWA $PM_{2.5}$ above 1000 $\mu g/m^3$ was experienced in 11% (n = 18) of all samples included in data analysis, while exposure was above 2000 $\mu g/m^3$ and 2500 $\mu g/m^3$ in 3% (n = 5) and 1% (n = 2) of these samples, respectively. There was no consistency within these samples regarding the subject or sample day. Filter $PM_{2.5}$ differed significantly across levels of the firefighters' self-assessed exposure ($p < 0.0001$ for samples collected on burn days), with a significant linear trend of increasing personal $PM_{2.5}$ exposure being observed at higher

TABLE I. Work Shift TWA Personal Exposure to $PM_{2.5}$ and CO

	Unadjusted				Adjusted[A]
	2003	2004	2005	Total	Total
Burn Day					
$PM_{2.5}$ Arithmetic mean (CLs) ($\mu g/m^3$)	353 (242, 464)	491 (365, 617)	507 (385, 629)	462 (389, 535)	
$PM_{2.5}$ Geomean (CLs) ($\mu g/m^3$)	215 (154, 300)	248 (184, 333)	347 (265, 456)	264 (221, 316)	280 (140, 557)
CO Geomean (CLs) (ppm)[B]		1.0 (0.07, 13)	1.1 (0.14, 9.2)	1.0 (0.09, 11.6)	
Duration of work shift- Mean (Min, Max) (hr)	9.0 (6.8, 10.5)	11.0 (7.8, 19.4)	10.1 (7.9, 14.5)	10.3 (6.8, 19.4)	
Size of Burn- Mean (Min, Max) (acres)	411 (1.0, 1900)	758 (5.0, 2745)	837 (345, 1898)	697 (1.0, 2745)	
N	43	82	52	177	
Non-Burn Day					
$PM_{2.5}$ Arithmetic mean (CLs) ($\mu g/m^3$)	26 (12, 39)	24 (14, 35)	12 (10, 15)	20 (15, 25)	
$PM_{2.5}$ Geomean (CLs) ($\mu g/m^3$)	23 (13, 43)	18 (12, 27)	12 (9.0, 15)	16 (12, 20)	16 (10, 26)
Duration of work shift-Mean (Min, Max) (hr)	8.6 (7.0, 9.0)	9.2 (9.0, 9.8)	9.9 (7.8, 11.5)	9.3 (7.0, 11.5)	
N	5	17	13	35	

Notes: N = 87 in 2004, N = 62 in 2005, N = 149 for all samples.
[A]Results were adjusted for plot size, wind speed, shift length, tasks, and the interactions between the tasks and the other variables.
[B]CO was measured only on burn days and in 2004 and 2005 alone.

levels of self-assessed exposure ($p < 0.0001$). The adjusted geometric mean exposures for all sample days estimated as none to very little by the firefighters was 120 $\mu g/m^3$ (95% CL = 71, 203 $\mu g/m^3$), and 664 $\mu g/m^3$ (95% CL = 373, 1185 $\mu g/m^3$) for exposures estimated as high or very high on samples collected on burn days (Figure 2). Only the difference between exposures estimated as moderate and those estimated as high or very high was insignificant ($p = 0.06$). Exposure

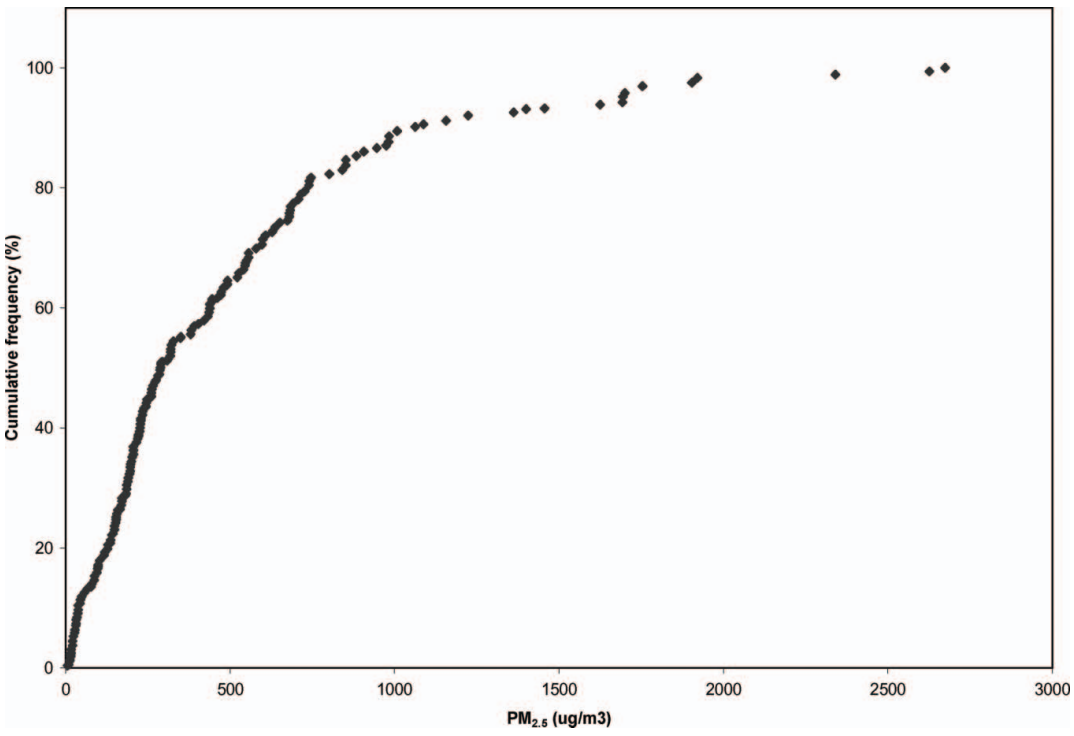


FIGURE 1. Cumulative frequency distribution for $PM_{2.5}$ exposure on burn days (N = 177)

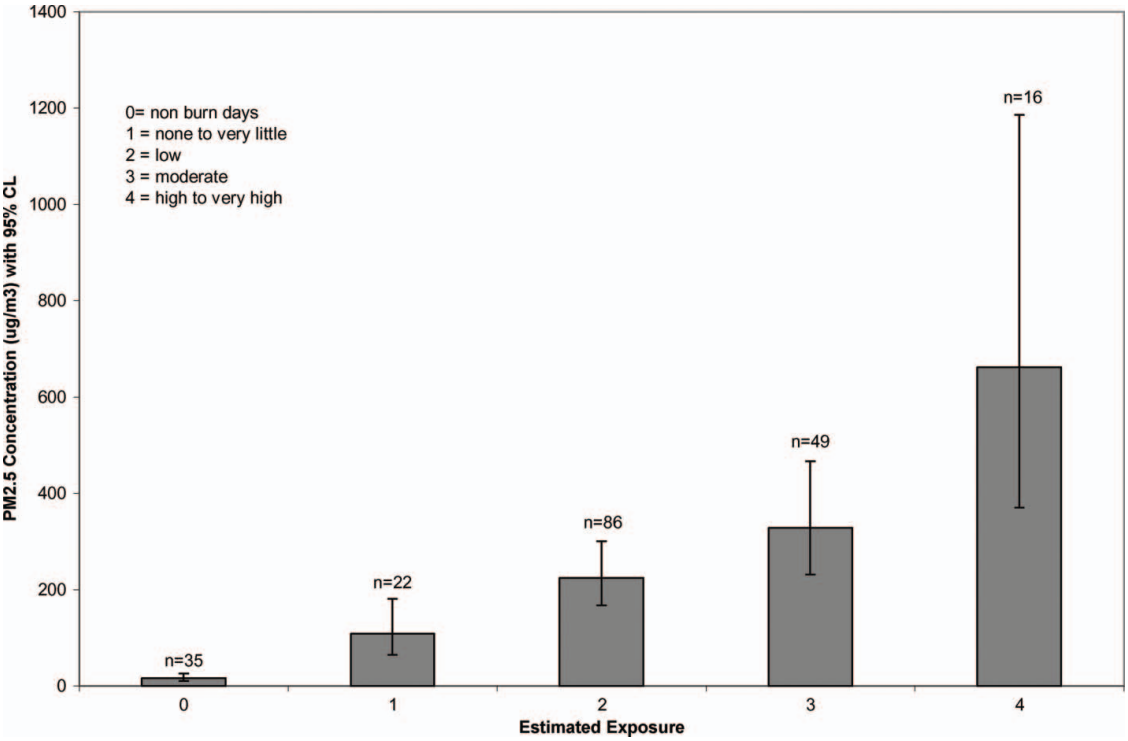


FIGURE 2. Geometric mean estimates of $PM_{2.5}$ at self-estimated exposure levels (N = 208) (N is less than 212 because some samples did not have data filled in for the self-estimation variable) ($p < 0.0001$)

was not dependent on size of burn, wind speed, or length of work time. Results of analyses suggest that type of task has an effect on exposure. However, the observed effect is solely due to tasks classified as "other" (tasks performed by the burn boss, from helicopters, or not directly at the burn). The differences between pairs of job tasks excluding the "other" category were insignificant. Figure 3 shows geometric mean $PM_{2.5}$ exposure on burn days classified according to the job task taking up at least 75% of the firefighters' work time.

In all, 149 real-time person-shift CO samples were collected during the 2004 and 2005 burn seasons. The geometric mean CO exposure (n = 149) is presented in Table I. Some pumps used for $PM_{2.5}$ sampling stopped before the end of the work shift, so for the purpose of the correlation analysis, the average CO samples were calculated for the periods for which the pumps ran. In addition, because some $PM_{2.5}$ samples were excluded from the analysis, only 134 CO/$PM_{2.5}$ pairs were used for the analysis. TWA $PM_{2.5}$ was correlated with TWA CO averaged over the run times of the $PM_{2.5}$ pumps (Pearson correlation coefficient estimate, r = 0.79; CLs: 0.72, 0.85; average duration, t = 9.3 hr) (Figure 4), and the correlation coefficient was significantly different from zero ($p < 0.0001$). Due to the increase in variance with increasing concentrations in both variables, we decided to fit the bivariate linear mixed-effect model to log-transformed values of $PM_{2.5}$ and CO simultaneously. The estimate for the Pearson correlation coefficient for this analysis was not substantially different: r = 0.73 (CLs: 0.64, 0.82).

DISCUSSION

Studies of occupational exposures to woodsmoke among wildland firefighters in the United States have revealed that they could be exposed to levels of particulate matter exceeding the OSHA permissible exposure limit (PEL) for respirable particulates (particulates with median size 3.5 μm, $PM_{3.5}$) of 5 mg/m^3 (OSHA, *Code of Federal Regulations*, Title 29) or the ACGIH threshold limit value (TLV for PM_4) of 3 mg/m^3.[14] These studies have been done mostly in forests in the western United States. The current study examines exposure among wildland firefighters in a forest in the southeastern United States where the vegetation and climate are very different.

It is difficult to make comparisons between this and other studies or the exposure standards because of the different size of particulate matter used in this study. Most of the previous forest firefighter exposure studies in the United States and the exposure standards are based on respirable particulates (with median aerodynamic diameter of 3.5 or 4 μm), while this study used particulate matter with median aerodynamic diameter of 2.5 μm ($PM_{2.5}$), defined by the U.S. Environmental Protection Agency (USEPA) as respirable particles. Various studies have shown that the aerodynamic diameter of particles in woodsmoke is mainly below 1.0 μm,[15–17] and most studies of the health effects of respirable particles have used $PM_{2.5}$ as a measure of exposure. Furthermore, we do not expect the weight concentration of $PM_{2.5}$ measured in this study to

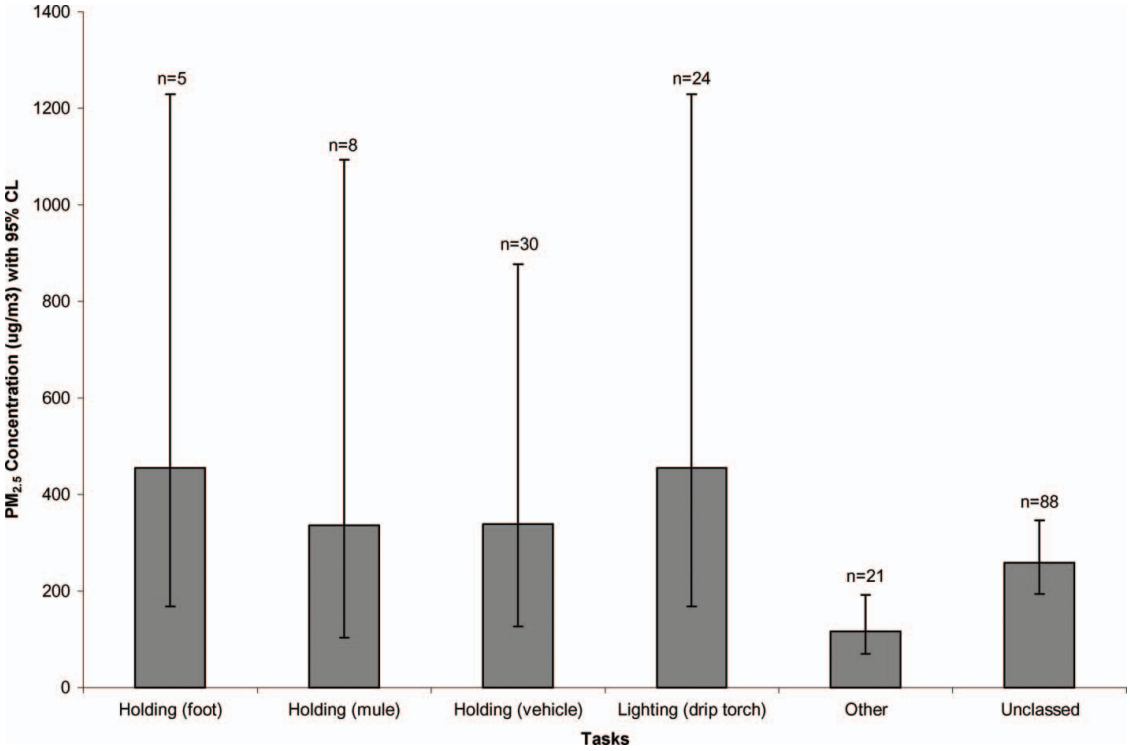


FIGURE 3. Geometric mean estimates of $PM_{2.5}$ exposure on burn days across different tasks with at least 75% of firefight work time. Holding was done on foot, on a mule (utility vehicle), or with a 4-wheel car (vehicle). Lighting was done with a drip torch. Tasks not under the major classification are categorized as "other," while the "unclassed" category is for exposures with the proportion of work shift time spent on all tasks during the particular work shift being below 75%.

be substantially different from that of $PM_{3.5}$. McMahon and Bush[18] reported a 12% difference in weight concentration between $PM_{3.5}$ and $PM_{2.3}$ from small, open burning greenhouse experiments using a 10-mm nylon cyclone. Subsequently, the measured exposures (geometric mean = 0.28 mg/m^3) seem to be lower than those reported by Reinhardt and Ottmar[8] (for $PM_{3.5}$: geometric mean = 0.63 mg/m^3; n = 200). Furthermore, higher exposures were observed among wildland firefighters during prescribed burns in an older study in the state of Georgia (for $PM_{2.3}$: median = 1.3 mg/m^3; range = 0.2–3.7 mg/m^3; n = 48).[18] However, exposure in the Georgia study was monitored only at the fire line and not over the entire work shift. In comparison, time spent performing tasks away from the fire during the work shift would have resulted in reduced TWA concentrations.

Although the geometric mean presented here indicates that the OSHA PEL or the ACGIH TLV for particulate matter is not exceeded among this group of firefighters, exposures may exceed the TLV as a few firefighters had a $PM_{2.5}$ exposure above 2500 μg/m^3. Exposure to such elevated levels of particulate matter may elicit various adverse health effects.[19–21] More specifically, woodsmoke exposure has been linked to respiratory symptoms and diseases,[5,22–26] and systemic inflammation.[27,28] Lung function decline and inflammation have also been observed among wildland firefighters post-exposure to woodsmoke.[6,7]

Daily average ambient 24-hr $PM_{2.5}$ concentrations measured by EPA monitors closest to the study site—those in South Carolina at Aiken: 1 mile NW, Edgefield: 25 miles NW, Richland: 50 miles NE, and Orangeburg: 37 miles NE, and in Augusta, Georgia: 16 miles NW—during the periods of the study were well below most of the personal exposures of the firefighters. The maximum concentration measured by any of the monitors was less than 30 μg/m^3 throughout the periods of the study.[29] Magnitudes of $PM_{2.5}$ exposure similar to those measured in this study have been observed for persons living in homes in which wood is used for cooking in rural communities in developing countries[30,31] and in ambient air in areas impacted by wildfires in the United States.[32,33]

As observed by Reinhardt and Ottmar,[8] average work shift particulate matter and CO are correlated in this study confirming that either of these two environmental markers might be used as surrogate measure of exposure to the other across a prescribed burn work shift. However, the slope of the relationship in this study appears to be steeper. This could be explained by the lower carbon monoxide exposure that was observed and, possibly, the difference in the aerodynamic size of the particles measured. Average exposure was not significantly affected by wind speed, wind direction, size of burn, or length of the work shift of the firefighter. Results show that firefighters tended to be able to predict their exposure.

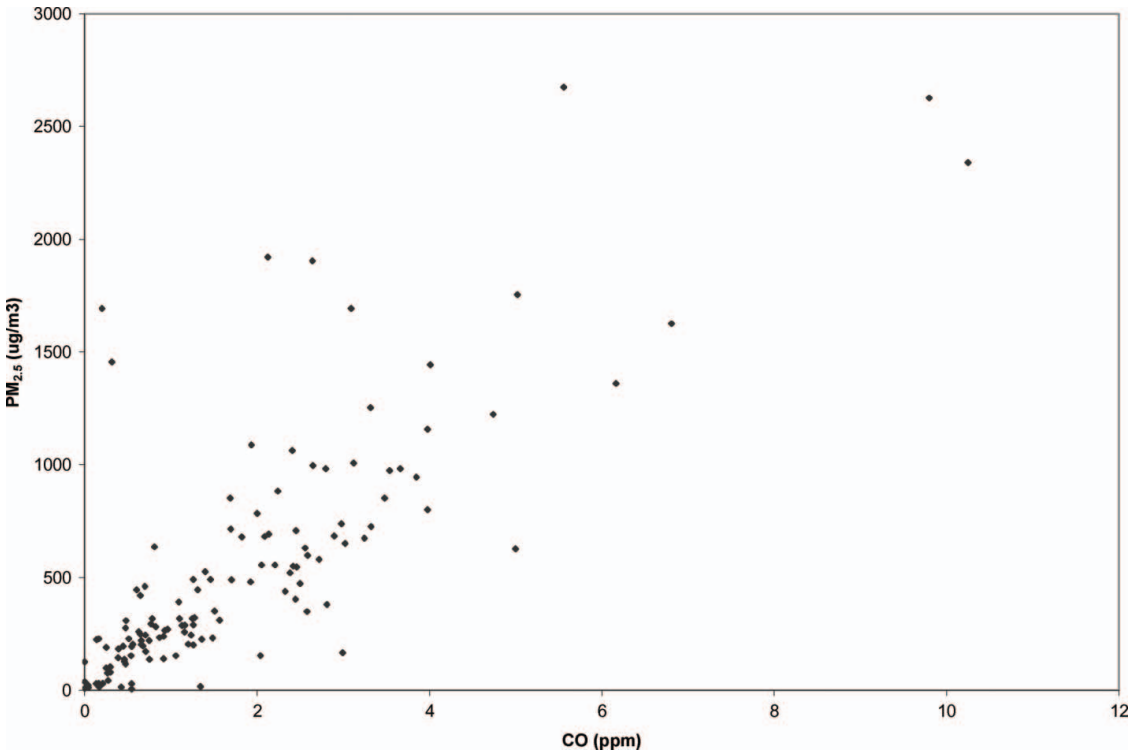


FIGURE 4. Association (scatterplot) between $PM_{2.5}$ and CO. Pearson correlation coefficient = 0.79; $p < 0.0001$ (estimated from bivariate linear mixed-effect model fitte simultaneously to $PM_{2.5}$ and CO) (N = 134).

However, the variation within each estimation class is large. The observed difference in exposure across job task was solely due to tasks relatively remote from the fires. However, the comparison was not precise because firefighters often worked multiple tasks during each work shift, making the attribution of exposure during a shift to a particular task difficult. Also, very few person-hours satisfied our criterion requiring at least 75% of the work shift to be spent doing the task, resulting in small sample sizes for most of the tasks in the analyses, but results did not change when the analysis was done with a relaxed classification and exposures were assigned to tasks the firefighters spent the majority of their time performing. Exposure was not completely captured in a few cases as some pumps failed to run the entire duration of the work shift. Also, exposure may be underreported in a few cases because firefighters sometimes put away their gear packs while working at some tasks in the field. However, we do not envisage that this would have impacted our results substantially, as there was good compliance among the subjects, and the firefighters only put away their gear packs for very short periods and only a few feet away when they did. We also kitted the subjects in this study with the samplers to minimize hindrance without compromising the results of the study. The use of time-integrated samplers to monitor exposure across the entire work shift precluded the calculation of TWA exposure at the fire line, which would have been higher than the result presented here.

CONCLUSIONS

Although the overall geometric mean $PM_{2.5}$ exposure indicates that the OSHA PEL or the ACGIH TLV for particulate matter was not surpassed, these limits may be exceeded, as some of the firefighters were exposed to very high levels of $PM_{2.5}$. The correlation between CO and $PM_{2.5}$ is potentially an exposure assessment tool for research and exposure management for firefighters working at prescribed burns.

RECOMMENDATIONS

As indicated above, the correlation between CO and $PM_{2.5}$ may be used for exposure control among wildland firefighters. CO monitors with alarms set at certain thresholds could, for example, be used to alert firefighters to a high/very high exposure situation. This could be particularly because firefighter exposure to woodsmoke may be dominated by momentary peaks.[34] However, the relationship between CO and PM may vary as indicated by the difference in the slope of the relationship in our study compared with those reported by Reinhardt and Ottmar.[8] Therefore, future studies are needed to better understand the relationship between the two pollutants/exposure proxies. For future studies, we recommend that real-time particulate matter samplers should be used, or where they are unavailable, time-integrated samplers should be run

for the duration when the firefighters are at the fire line. The completion of time-activity diaries by researchers detailed to monitor the activities of firefighters at the fire line instead of self-administered diaries, together with the use of real-time samplers, would present the researcher with data to better understand the relationship between job tasks and exposure. The use of real-time particulate matter samplers could also facilitate a better understanding of the relationship between particulate matter and CO. We also recommend that samplers be worn directly on the firefighter to avoid underreporting in cases where firefighters put away their gear packs. Some of these corrections have been and are being made in subsequent studies among this group of wildland firefighters.

ACKNOWLEDGMENTS

We acknowledge the support of John Blake, Dan Shea, Jason Demas, Mark Frizzel, Paul Linse, and Jeff Prevey of the United States Forest Service and David Macintosh of the Environmental Resource Management Inc., Needham, Massachusetts. Funding was provided by the Department of Energy–Savannah River Operations Office through the U.S. Forest Service Savannah River under Interagency Agreement DE-AI09-00SR22188.

REFERENCES

1. **Reinhardt, T.E.:** "Monitoring Firefighter Exposure to Air Toxins at Prescribed Burns of Forest and Range Biomass." Research paper PNW-RP-441. Portland, Ore.: Pacific Northwest Research Station, U.S. Department of Agriculture, Forest Service, 1991.
2. **Wade, D.D., B.L. Brock, P.H. Brose, et al.:** Fire in eastern ecosystems. In *Fire in Ecosystems: Effects of Fire on Flora*, J.K. Brown and J.K. Smith (eds.), pp 53–96. Fort Collins, Colo. Rocky Mountain Research Station: USDA Forest Service, 2000.
3. **Harrison, R., B.L. Materna, and N. Rothman:** Respiratory health hazards and lung function in wildland firefighters. *Occup. Med. 10*(*4*):857–870 (1995).
4. **Reinhardt, T.E., and R.D. Ottmar:** "Smoke Exposure at Western Wildfires." Research paper PNW-RP-525. Portland, Ore.: Pacific Northwest Research Station, U.S. Department of Agriculture, Forest Service, 2000.
5. **Naeher, L.P., M. Brauer, M. Lipsett, et al.:** Woodsmoke health effects: A review. *Inhalat. Toxicol. 19*:67–106 (2007).
6. **Betchley, C., J.Q. Koenig, G. van Belle, et al.:** Pulmonary function and respiratory symptoms in forest firefighters. *Am. J. Ind. Med. 31*(*5*):503–509 (1997).
7. **Swiston, J.R., W. Davidson, S. Attridge, et al.:** Wood smoke exposure induces a pulmonary and systemic inflammatory response in firefighters. *Eur. Respir. J. 32*:129–138 (2008).
8. **Reinhardt, T.E., and R.D. Ottmar:** Baseline measurements of smoke exposure among wildland firefighters. *J. Occup. Environ. Hyg. 1*:593–606 (2004).
9. **Materna, B.L., J.R. Jones, P.M. Sutton, et al.:** Occupational exposures in California wildland fire fighting. *Am. Ind. Hyg. Assoc. J. 53*:69–76 (1992).
10. **Reisen, F., and S. K. Brown:** Australian firefighters' exposure to air toxics during bushfire burns of autumn 2005 and 2006. *Environ. Intern. 35*(*2*):342–352 (2009).
11. "USDA Forest Service-Savannah River" [Online] Available at http://www.srs.gov/general/srfs/srfs.htm (Accessed October 8, 2009).
12. **Kenny, L.C., and R.A. Gussman:** Characterization and modelling of a family of cyclone aerosol preseperators. *J. Aerosol Sci. 28*(*4*):677–688 (1997).
13. **U.S. Environmental Protection Agency (USEPA):** *Field Standard Operating Procedures for the $PM_{2.5}$ Performance Evaluation Program. 441. Research Triangle Park, N.C.: USEPA*, Office of Air Quality Planning and Standards, 1998.
14. **ACGIH:** *Threshold Limit Value for Chemical Substances and Physical Agents, Biological Exposure Indices*. Cincinnati, Ohio: ACGIH, 2003.
15. **Kleeman, M.J., J.J. Schauer, and R. Glen:** Size and composition distribution of fine particulate matter emitted from wood burning, meat charboiling, and cigarettes. *Environ. Sci. Technol. 33*:3516–3523 (1999).
16. **Leonard, S.S., V. Castranova, B.T. Chen, et al.:** Particle size-dependent radical generation from wildland fire smoke. *Toxicology 236*:103–113 (2007).
17. **Silva, P.J., D.-Y. Liu, C.A. Noble, et al.:** Size and chemical characterization of individual particles resulting from biomass burning of local Southern California species. *Environ. Sci. Technol. 33*:3068–3076 (1999).
18. **McMahon, C.K. and P.B. Bush:** Forest worker exposure to airborne herbicide residues in smoke from prescribed fires in the southern United States. *Am. Ind. Hyg. Assoc. J. 53*:265–272 (1992).
19. **Dominici, F., R.D. Peng, M.L. Bell, et al.:** Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *JAMA 295*(*10*):1127–1134 (2006).
20. **Dockery, D.W. and C.A. Pope:** Acute respiratory effects of particulate air pollution. *Ann. Rev. Publ. Health 15*:107–132 (1994).
21. **Dockery, D.W., C.A. Pope, X. Xu, et al.:** An association between air pollution and mortality in six US cities. *N. Engl. J. Med. 329*:1753–1759 (1993).
22. **Ezzati, M., and D. M. Kammen:** Quantifying the effects of exposure to indoor air pollution from biomass combustion on acute respiratory infections in developing countries. *Environ. Health Perspect. 109*(*5*):481–488 (2001).
23. **Duclos, P., L.M. Sanderson, and M. Lipsett:** The 1987 Forest Fire Disaster in California: Assessment of emergency room visits. *Arch. Environ. Health 45*(*1*):53–58 (1990).
24. **Delfino, R.J., S. Brummel, J. Wu, et al.:** The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003. *Occup. Environ. Med. 66*:189–197 (2008).
25. **Smith, K.R., J.M. Samet, I. Romieu, et al.:** Indoor air pollution in developing countries and acute lower respiratory infections in children. *Thorax 55*:518–532 (2000).
26. **Sutherland, E.R., B.J. Make, S. Vedal, et al.:** Wildfire smoke and respiratory symptoms in patients with chronic obstructive pulmonary disease. *J. Allergy Clin. Immunol. 115*(*2*):420–422 (2005).
27. **Van Eeden, S.F., W.C. Tan, T. Suwa, et al.:** Cytokines involved in the systemic inflammatory response induced by exposure to particulate matter air pollutants (PM10). *Am. J. Respir. Crit. Care Med. 164*(*5*):826–830 (2001).
28. **Tan, W.C., D. Qiu, B.L. Liam, et al.:** The human bone marrow response to acute air pollution caused by forest fires. *Am. J. Respir. Crit. Care Med. 161*(*4*):1213–1217 (2000).
29. "Monitor Values Report—Criteria Air Pollutants." [Online] Available at http://www.epa.gov/air/data/monvals.html?st~SC~South%20Carolina (Accessed October 23, 2009).
30. **Zuk, M., L. Rojas, S. Blanco, et al.:** The impact of improved wood-burning stoves on fine particulate matter concentrations in rural Mexican homes. *J. Expos. Sci. Environ. Epidemiol. 17*:224–232 (2007).
31. **Cynthia, A., R. Edwards, M. Johnson, et al.:** Reduction in personal exposures to particulate matter and carbon monoxide as a result of the

installation of a Patsari improved cook stove in Michoacan, Mexico. *Indoor Air 18*(*2*):93–105 (2008).

32. **Ward, T.J., R.F. Hamilton, R.W. Dixon, et al.:** Characterization and evaluation of smoke tracers in PM: Results from the 2003 Montana wildfire season. *Atmos. Environ. 40*(*36*):7005–7017 (2006).
33. **Wu, J., A.M. Winer, and R.J. Delfino:** Exposure assessment of particulate matter air pollution before, during, and after the 2003 southern California wildfires. *Atmos. Environ. 40*(*18*):3333–3348 (2006).
34. **Edwards, R., M. Johnson, K.H. Dunn, et al.:** Application of real-time particle sensors to help mitigate exposures of wildland firefighters. *Arch. Environ. Occup. Health 60*:40–43 (2005).

Taylor & Francis
Taylor & Francis Group

Inhalation Toxicology

International Forum for Respiratory Research

ISSN: 0895-8378 (Print) 1091-7691 (Online) Journal homepage: https://www.tandfonline.com/loi/iiht20

Application of End-Exhaled Breath Monitoring to Assess Carbon Monoxide Exposures of Wildland Firefighters at Prescribed Burns

Kevin H. Dunn, Isabelle Devaux, Allison Stock & Luke P. Naeher

To cite this article: Kevin H. Dunn, Isabelle Devaux, Allison Stock & Luke P. Naeher (2009) Application of End-Exhaled Breath Monitoring to Assess Carbon Monoxide Exposures of Wildland Firefighters at Prescribed Burns, Inhalation Toxicology, 21:1, 55-61, DOI: 10.1080/08958370802207300

To link to this article: https://doi.org/10.1080/08958370802207300

Inhalation Toxicology, 21:55–61, 2009

ISSN: 0895-8378 print / 1091-7691 online
DOI: 10.1080/08958370802207300

informa
healthcare

Application of End-Exhaled Breath Monitoring to Assess Carbon Monoxide Exposures of Wildland Firefighters at Prescribed Burns

Kevin H. Dunn
National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, Georgia, USA

Isabelle Devaux
National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, Georgia, USA

Allison Stock
National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, Georgia, USA

Luke P. Naeher
Department of Environmental Health Science, College of Public Health, University of Georgia, Athens, Georgia, USA

Exposure to the range of combustion products from wildland fires has been demonstrated to cause respiratory irritation and decreased lung function among firefighters. The measurement of carbon monoxide (CO) has been previously shown to be highly correlated with the range of contaminants found in wildland fires. In this article, we assess the feasibility of using a simple, noninvasive biological test to assess exposure to CO for a group of wildland firefighters. Measurements of CO exposure were collected using personal monitors as well as in exhaled breath for wildland firefighters who conducted prescribed burns in February–March 2004. Overall, the CO concentrations measured in this study group were low with a shift mean of 1.87 ppm. Correspondingly, the cross-shift difference in carboxyhemoglobin as estimated from exhaled breath CO levels was also low (median increase = +0.2% carboxyhemoglobin). The use of exhaled breath measurements for CO has limitations in characterizing exposures within this worker population.

This study was conducted on the Savannah River Site (SRS), a Department of Energy industrial complex located in the southeastern coastal area of the United States. The site encompasses 198,000 acres bordering on the Savannah River. In terms of broad forest types, the SRS is about 31% hardwood or mixed pine hardwood and 69% pine (U.S. Department of Agriculture, 2008). Controlled fires are used to reduce the accumulation of combustible forest fuels. In pine stands, such as those found in the SRS, pine needles cover the ground, shrubs, and hardwoods, making wildfires more likely. The major objective during this burn season was to reduce the overall undergrowth using controlled burns.

As a part of fire management objectives, controlled fires are used extensively throughout the United States. These controlled fires, also known as prescribed burns, are used to reduce the risk of wildfire (uncontrolled fires) by reducing natural fuel load, controlling undergrowth to allow for regeneration of desirable plants, and controlling insects and diseases. In 2007, over 3 million acres of land were treated with prescribed burns in the United States (National Interagency Fire Center, 2008) Smoke management requires that the fires are conducted under strict

Received 5 March 2008; accepted 15 May 2008.
This research was supported by the U.S. Department of Agriculture Forest Service and approved by the University of Georgia Institutional Review Board for the inclusion of human subjects. The authors gratefully acknowledge Jeff Prevey, Mark Frizzell, Dan Shea, John Blake, Chris Hobson, Jason Demas, and the firefighters participating in this study.
Address correspondence to Luke P. Naeher, Department of Environmental Health Science, College of Public Health, University of Georgia, 206 Environmental Health Science Building, Athens, GA 30602-2102, USA. E-mail: LNaeher@gmail.com

conditions to reduce impact to the surrounding environment. To minimize ground-level smoke produced during the burns, prescribed burn planners account for meteorological conditions (wind speed and direction, atmospheric stability, etc.), fuel loading, fuel moisture, and a variety of other factors. However, even with appropriate planning, firefighters are exposed to moderate levels of smoke during prescribed burns.

The primary emissions from forest fires are water and carbon dioxide, which account for over 90% of the total emissions. However, fires produce many known air toxicants, including carbon monoxide, particulate matter, hydrocarbons, nitrogen oxides, and aldehydes. Previous studies of wildland firefighters have shown that the highest measured exposures were to particulate matter and carbon monoxide (Materna et al., 1992; Reinhardt & Ottmar, 2004). The health effects associated with these exposures include short-term effects such as cough, sore throat, and sore eyes due to exposure to particulates and aldehydes. Transient adverse health effects from exposure to carbon monoxide, such as headache, lightheadedness, and dizziness, have also been reported. And other health effects such as decreased lung function from exposure to smoke have been seen in several studies (Rothman et al., 1991; Betchley et al., 1997; Slaughter, Koenig et al., 2004). Decreased lung functions have been shown across workshift and occasionally across the burning season (Rothman et al., 1991; Liu et al., 1992). Despite these observed health effects, most wildland firefighters do not wear any respiratory protection.

Exposure assessments on wildland firefighters have shown higher exposures during prescribed burns than when responding to wildfires (Reinhardt & Ottmar, 2004). This research also showed that CO concentrations in smoke at project fires and prescribed burns were strongly correlated to concentrations of formaldehyde, particulate matter less than or equal to 2.5 μm in diameter (PM2.5), and acrolein. The researchers recommended the use of personal CO ambient monitors as a cost-effective way to estimate fine particulate exposure in those occupationally exposed to forest fire smoke (Reinhardt et al., 1999).Although these personal monitors are reasonable for use in some occupational settings, their cost and application may be restrictive for monitoring large firefighter populations.

Previous studies have shown a strong correlation between end-exhaled breath measurement of carbon monoxide and the level of carboxyhemoglobin (COHb) in the blood (Wald et al., 1975; Jarvis et al., 1986; Irving et al., 1988). Some researchers have evaluated the use of portable exhaled breath monitors to rapidly assess exposure to ambient CO among the general population and in firefighters (Cunnington & Hormbrey, 2002; Cone et al., 2005). In this article, we assess the feasibility of using end-exhaled breath as a determinant of CO exposure for this firefighter population.

METHODS

Study Population

A forest fire crew of 19 nonsmoking firefighters was followed throughout the fire season from February through March 2004. The fire crew was a local initial attack/prescribed fire crew stationed at the U.S. Department of Energy Savannah River Site in New Ellenton, SC. The crew's main function was conducting prescribed fires (igniting and holding). The overall group of 19 firefighters included 16 men and 3 women with an average age of 29 yr (median = 29, range 21 to 44). One firefighter was excluded due to job assignment as a fire planner, which minimized his presence at the site of the fires. Another firefighter who smoked was excluded from the study, due to the known high background of CO in expired breath. Respiratory protection was not used by any firefighters primarily due to the fact that protection for both CO and particulates would require full breathing apparatus which is not practical for wildfire fighting where mobility and weight are important considerations. Informed consent was obtained from each firefighter participating in the study in accordance with the University of Georgia/Centers for Disease Control Institutional Review Board (UGA/CDC IRB).

Over the 3-wk study period, 10 prescribed burn days were monitored. A group of up to 10 of the firefighters was sampled each day for monitoring of both real-time carbon monoxide and end exhaled breath for CO, yielding 80 person-day samples during the study period. Field personnel attempted to ensure that all available firefighters were sampled; all participated at least once during the study period. The average work shift length was 11.3 h, of which 6.8 h/shift was spent at the burn site and 4.5 h/shift was spent planning, preparing for, and traveling to/from the burn site. The prescribed burns covered an average of 692 acres per burn (median = 362, range 28 to 2745).

CO Personal Monitoring

Personal air samples for CO exposure were collected in the breathing zone of the firefighters using Draeger PAC III single gas monitors (Draeger Safety, Inc., Pittsburgh, PA) outfitted with CO sensors (see Figure 1). These monitors use an electrochemical cell for detection, have a range of 0 to 2000 ppm, and are direct-reading instruments with data-logging capabilities. The PAC III CO monitor was attached to the gear pack of each firefighter at a location that placed the monitor in the firefighter's breathing zone. The PAC III CO monitors were calibrated with a 200-ppm CO certified gas standard prior to the start of the study. During the study, the monitors were zeroed with ambient air at the forest station at the beginning of each shift and response was checked with 200 ppm gas at the end of each shift. These data were collected to provide a comparison exposure assessment method for the exhaled breath measurements.

CO Exhaled Breath

End exhaled breath CO measurements were collected from each study participant before and after each shift (see Figure 2). The subjects were asked to inhale deeply and hold their breath for 15 s to allow for the CO in the blood to escape into the lung and equilibrate prior to exhaling completely through an EC50 ToxCO exhaled breath monitor (Bedfont Scientific, USA, Medford, NJ). The exhaled breath monitors used in this study calculate percent carboxyhemoglobin (%COHb) in the blood

FIG. 1. Photo of firefighter with personal CO monitor.

based on exhaled breath CO concentrations measured by an electrochemical sensor. The %COHb estimated by the monitor is proportional to the measured CO in the breath (ppm) multiplied by a factor of 0.16, based on research conducted by Jarvis (Jarvis et al., 1986). An increase in exhaled breath concentration of 1 ppm would result in a corresponding rise in the estimated %COHb by 0.16%. The monitors were allowed to decay to background levels between each firefighter. They were calibrated with 50 ppm CO standard gas prior to the start of the study and checked at the end of the study.

Each day, the firefighters reported to a trailer at the office site to be outfitted with the PAC III personal CO monitors and for the collection of the end exhaled breath sample for the prework shift. Following the completion of the burn, workers would travel back to the office and report to the trailer again to perform the postwork shift breath sample and to return the personal CO monitor.

Data Analysis Plan

Overall, 80 person-day samples were available for analysis. All analyses were performed using Excel or SAS statistical programs. Time-weighted average CO concentrations were calculated for full-shift exposure for all monitoring periods. The full-shift CO average was used in the analysis, including time at the fires as well as in the office and in transit, since the exhaled breath samples were collected across the complete shift. Arithmetic mean and standard deviations are reported for personal CO measurements and cross shift COHb changes for each burn day. Scheffe tests were conducted to evaluate differences in the values for various group means including shift mean CO concentrations. Log transformed shift mean CO concentrations were used for these tests since log normality was confirmed using the Shapiro-Wilks test. A linear regression analysis was performed to compare the cross-shift change in end-exhaled breath CO levels with the shift average CO personal concentration.

FIG. 2. Photo of firefighter performing exhaled breath CO test.

RESULTS

CO Personal Monitoring Data

Table 1 shows the number of samples, average shift length, mean and standard deviation, and range for the shift mean CO concentration and cross-shift COHb changes for each burn day. The overall group mean shift average CO concentration was 1.87 ppm and the standard deviation was 2.31 with exposures ranging from 0.01–14.05 ppm. The overall average shift length was 11.1 h, with daily average shifts ranging from 8.9 to 13.5 h. The daily exposure was characterized by a period of minimal CO exposure while the firefighters were in the office in both the morning and evening (before and after the fire). The CO exposures at the prescribed burn sites consisted of many transient peaks in exposure typically lasting less than 1 min in duration. The real-time CO monitor data in Figure 3 illustrate the typical firefighter exposure profile for a high exposure day (day 8) and a lower exposure day (day 4).

CO Exhaled Breath Data

COHb levels decreased among 20% ($n = 16$), remained unchanged among 20% ($n = 16$) and increased among 60% of the sample ($n = 48$) (see Figure 4). The magnitude of these changes, however, was small. The absolute value of the median decrease was 0.35% (range –1.2 to –0.1% COHb) and the median increase was 0.30% (range 0.1 to 2.6% COHb). The mean levels of cross-shift COHb change between those firefighters who had decreased COHb from pre- to postshift, those who had no change in COHb measurement, and those who had increased cross-shift were statistically different ($p < .001$). However, the mean shift average CO concentrations were not significantly different between these three groups ($p = .24$) (see Figure 5).

TABLE 1
Descriptive statistics by burn day

Burn day	Sample number *n*	Average shift length (hrs)	Shift mean CO concentration (ppm)				Mean cross-shift COHb change (%COHb)			
			AM	SD	Min	Max	AM	SD	Min	Max
1	9	10.1	0.89	0.91	0.01	2.39	0.09	0.20	−0.2	0.3
2	10	10.8	1.52	0.72	0.46	2.53	0.19	0.26	0	0.8
3	9	11.1	0.83	0.46	0.17	1.29	0.11	0.13	−0.1	0.3
4	9	11.0	1.25	0.78	0.37	2.73	0.09	0.30	−0.4	0.6
5	7	8.9	0.33	0.17	0.14	0.63	−0.07	0.62	−1.2	0.6
6	7	13.5	2.07	1.08	0.01	2.97	0.31	0.22	0	0.6
7	8	11.3	2.33	1.23	0.48	4.03	0.09	0.32	−0.5	0.5
8	8	12.9	5.20	3.59	0.85	9.7	1.09	0.90	−0.1	2.6
9	6	9.6	1.02	0.38	1.57	7.2	−0.23	0.46	−0.6	0.6
10	7	12.2	3.56	4.82	0.04	14.05	0.30	0.62	−0.3	1.5
Overall Group Average			1.87	2.31			0.20	0.54		

Note: AM = Arithmetic Mean, SD = Standard. Deviation

Regression Analysis

A regression of the shift average CO exposures and the corresponding cross-shift COHB measurements is shown in Figure 6. The r-squared correlation coefficient for this data set is .49. Most personal CO concentration measurements from this study were below 5 ppm. These measurements corresponded with a cross-shift change in COHB approximately between –1% and 1%. Only those personal shift average CO exposures above about 5 ppm resulted in a consistent increase in cross-shift COHB.

DISCUSSION

Overall shift average CO exposures were low when compared to occupational exposure limits—none of the firefighters were exposed to levels exceeding the OSHA or NIOSH 8-h time-weighted average limits of 50 ppm and 35 ppm, respectively (NIOSH, 1973; CFR, 1997). These limits are both based on controlling the level of carboxyhemoglobin in the worker's blood to prevent adverse health effects. Much of the work shift was spent preparing for and traveling to/from the fires, thus reducing

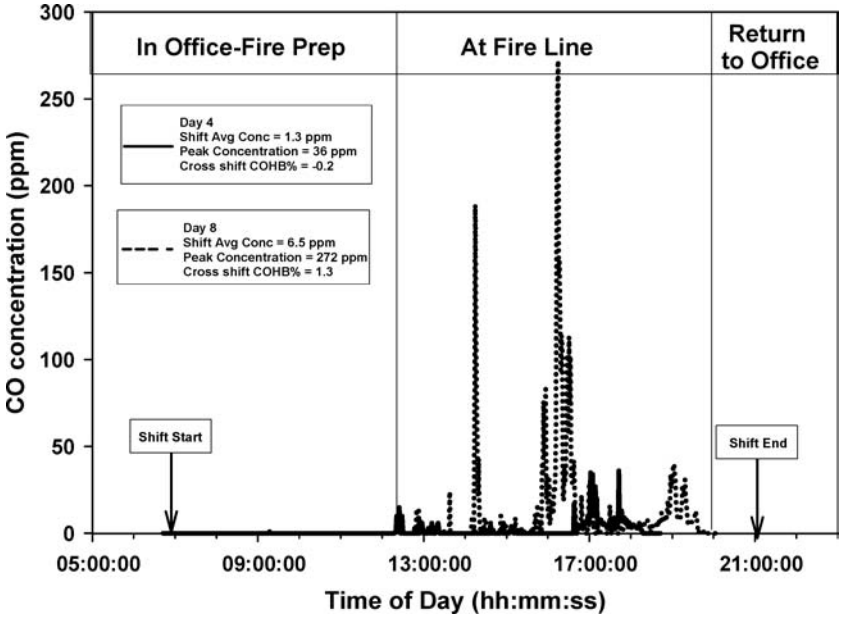


FIG. 3. Daily exposure profile for one firefighter performing prescribed burn activities on two separate days.

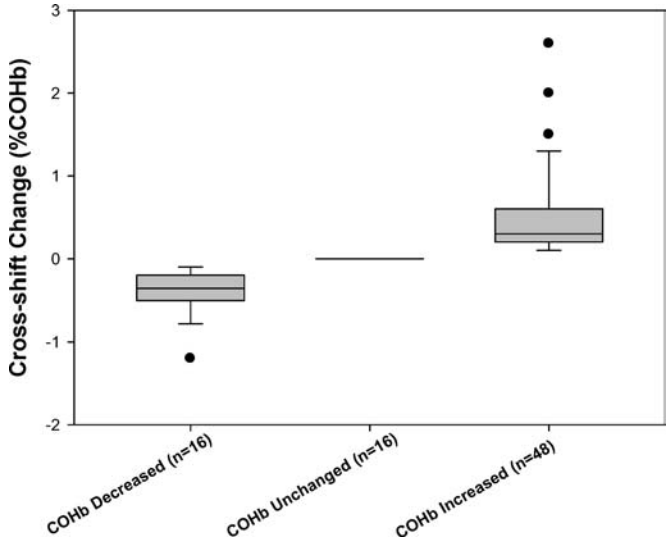


FIG. 4. Distribution of cross-shift COHb status. Note: Whiskers represent 3/2 times the interquartile range. Dots represent data points beyond that range.

overall exposure. The concentrations measured in this study were lower than previous reported CO exposures among prescribed burn wildland firefighters (Reinhardt & Ottmar, 2004). Reinhardt and Ottmar (2004) found an overall shift geometric mean exposure of 4.1 ppm among 221 firefighters at 39 prescribed burns in Oregon and Washington. Postshift %COHb measurements were also low, with the highest level observed in the study population being 2.8%, which is lower than the background level measured in tobacco smokers (5–9% COHb).

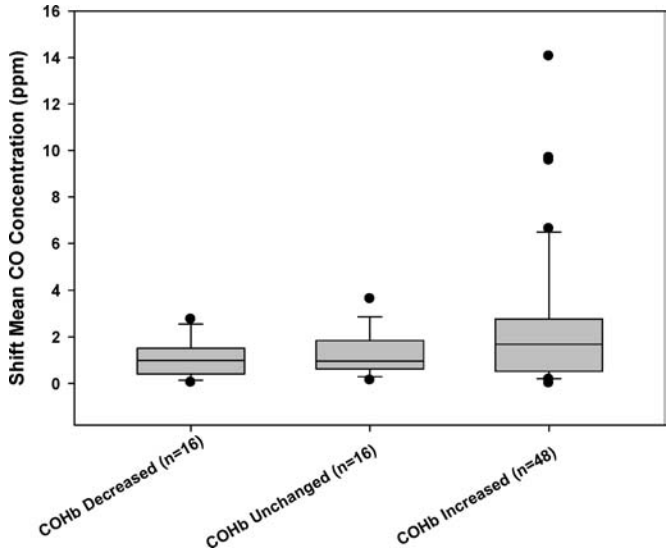


FIG. 5. Distribution of shift average CO concentrations by COHb cross-shift status. Note: Whiskers represent 3/2 times the inter-quartile range. Dots represent data points beyond that range.

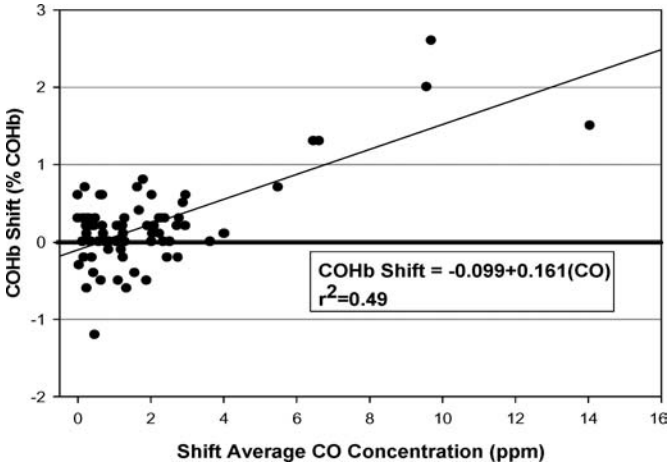


FIG. 6. Regression of personal CO monitor data with cross-shift exhaled breath analysis for the study cohort of prescribed burn firefighters.

In many cases, the postshift %COHb levels were lower than the preshift levels. This may result from higher CO exposure at periods of time away from work due to home (e.g., gas appliances, wood burning stoves, use of gas-powered tools) and transportation sources. All firefighters included in the analysis indicated that they lived in a nonsmoking home.

Endogenous levels of COHb levels range from 0.4 to 0.7%, with increased levels measured in urban populations (from 1 to 2%). The use of a cross-shift %COHb determination attempts to account for the background levels in the test subjects. However, given the variability seen in this study population (preshift COHb levels ranged from 0 to 1.5%), it is important for shift average airborne concentrations to exceed a nominal level to be predictive of exposure using this method. Given that the majority of the pre- to postshift %COHb differences seen in this study were less than 0.5%, the corresponding cross-shift change in exhaled breath CO concentration was on the order of 3 ppm or less.

The findings in this article are subject to at least two limitations. First, the exhaled breath measurements were collected about 1–2 h following cessation of exposure. This means that firefighter COHb level would have decreased approximately 10–25% from immediate postexposure concentration. Second, the nature of the exposure was characterized by short-term transient peak concentrations. The firefighters' blood COHb levels may not have reached a steady state over the work shift.

CONCLUSION

The use of prescribed burning within the United States has increased markedly over the past decade (National Interagency Fire Center, 2008). Previous studies have shown health effects in wildland firefighters associated with exposure to the variety of air contaminants in smoke. The use of carbon monoxide as a surrogate for the exposure from overall smoke components

has been proposed based on its correlation with the other major contaminants found in fires (Reinhardt & Ottmar, 2004). The use of exhaled breath CO measurements was evaluated as a simple, noninvasive method to assess firefighter exposure. However, these measurements provided limited data for characterizing exposure from this population of workers. A major challenge in monitoring this population includes the temporal variability in their exposure profile. The exposure profile was marked by periods of minimal exposure at the beginning and end of the shift with transient peak exposures throughout the burn activities. Also, the relatively low level of average exposure makes the use of end exhaled breath monitoring problematic in this worker population. Given the potential interferences of endogenous background as well as from sources such as gasoline powered tools and vehicles, the CO concentrations measured in the end-of-shift exhaled breath in this population often were less than the preshift background. Finally, end exhaled breath measurements do not provide any data on peak exposures, which may be important in explaining transient health effects in this population.

Therefore, using end exhaled breath monitoring may not be an effective tool in determining CO exposures to this group of wildland firefighters. However, in more highly exposed groups, this method may provide a quick and noninvasive way to assess exposures to carbon monoxide, a key component in smoke. More research needs to be performed to evaluate the effectiveness of using these monitors in study populations who are exposed to higher average CO concentrations. In low to moderately exposed populations, real-time CO monitors give better accuracy and discrimination for transient short-term exposure peaks.

DISCLAIMER

Mention of company names or products does not constitute endorsement by the Centers for Disease Control and Prevention. The findings and conclusions in this paper are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention/National Center for Environmental Health.

REFERENCES

Betchley, C., Koenig, J. Q., van Belle, G., Checkoway, H., and Reinhardt, T. 1997. Pulmonary Function and Respiratory Symptoms in Forest Firefighters. *Am. J. Ind. Med.* 31(5):503–509.

CFR. 1997. 29 Cfr 1910.1000, Chapter Xvii—Occupational Safety and Health Administration. Code of Federal Regulations, Table Z-1, Limits for Air Contaminants. Washington, D.C: U.S. Federal Register.

Cone, D. C., MacMillan, D. S., Van Gelder, C., Brown, D. J., Weir, S. D., and Bogucki, S. 2005. Noninvasive Fireground Assessment of Carboxyhemoglobin Levels in Firefighters. *Prehosp. Emerg. Care* 9(1):8–13.

Cunnington, A. J., and Hormbrey, P. 2002. Breath Analysis to Detect Recent Exposure to Carbon Monoxide. *Postgrad. Med. J.* 78(918):233–237.

Irving, J. M., Clark, E. C., Crombie, I. K., and Smith, W. C. 1988. Evaluation of a Portable Measure of Expired-Air Carbon Monoxide. *Prev. Med.* 17(1):109–115.

Jarvis, M. J., Belcher, M., Vesey, C., and Hutchison, D. C. 1986. Low Cost Carbon Monoxide Monitors in Smoking Assessment. *Thorax.* 41(11):886–887.

Liu, D., Tager, I. B., Balmes, J. R., and Harrison, R. J. 1992. The Effect of Smoke Inhalation on Lung Function and Airway Responsiveness in Wildland Fire Fighters. *Am. Rev. Respir. Dis.* 146(6):1469–1473.

Materna, B. L., Jones, J. R., Sutton, P. M., Rothman, N., and Harrison, R. J. 1992. Occupational Exposures in California Wildland Fire Fighting. *Am. Ind. Hyg. Assoc. J.* 53(1):69–76.

National Interagency Fire Center. 2008. Fire Information—Wildland Fire Statistics. In, http://www.nifc.gov/fire_info/ytd_state.htm. (accessed April 9, 2008).

NIOSH. 1973. *Criteria for a Recommended Standard: Occupational Exposure to Carbon Monoxide*. Vol. DHEW (NIOSH) HSM 73-11000. Cincinnati, OH: Department of Health Education and Welfare, Public Health Service, Cenetrs for Disease Control, National Institute for Occupational Safety and Health.

Reinhardt, T. E., and Ottmar, R. D. 2004. Baseline Measurements of Smoke Exposure among Wildland Firefighters. *J. Occup. Environ. Hyg.* 1(9):593–606.

Reinhardt, Timothy E., and Roger D. Ottmar, Michael J. 1999. Hallett, and Pacific Northwest Research Station (Portland Or.). *Guide to Monitoring Smoke Exposure of Wildland Firefighters*. Portland, Or.: U.S. Dept. of Agriculture, Forest Service, Pacific Northwest Research Station.

Rothman, N., Ford, D. P., Baser, M. E., Hansen, J. A., O'Toole, T., Tockman, M. S., and Strickland, P. T. 1991. Pulmonary Function and Respiratory Symptoms in Wildland Firefighters. *J. Occup. Med.* 33(11):1163–1167.

Slaughter, J. C., Koenig, J. Q., and Reinhardt, T. E. 2004. Association between Lung Function and Exposure to Smoke among Firefighters at Prescribed Burns. *J. Occup. Environ. Hyg.* 1(1):45–49.

U.S. Department of Agriculture. 2008. Usda Forest Service-Savannah River. In, http://www.srs.gov/general/srfs/srfs.htm. (accessed May 20, 2008).

Wald, N., Howard, S., Smith, P. G., and Bailey, A. 1975. Use of Carboxyhaemoglobin Levels to Predict the Development of Diseases Associated with Cigarette Smoking. *Thorax.* 30(2):133–140.

Routledge
Taylor & Francis Group

Archives of Environmental & Occupational Health

ISSN: 1933-8244 (Print) 2154-4700 (Online) Journal homepage: https://www.tandfonline.com/loi/vaeh20

Personal Carbon Monoxide Exposures Among Firefighters at Prescribed Forest Burns in the Southeastern United States

K. H. Dunn MSEE, CIH , S. Shulman PhD , A. L. Stock PhD, MPH & L. P. Naeher PhD

To cite this article: K. H. Dunn MSEE, CIH , S. Shulman PhD , A. L. Stock PhD, MPH & L. P. Naeher PhD (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, DOI: 10.1080/19338244.2011.633126

To link to this article: https://doi.org/10.1080/19338244.2011.633126

Published online: 08 Jan 2013.

Archives of Environmental & Occupational Health, Vol. 68, No. 1, 2013

Brief Communication

Personal Carbon Monoxide Exposures Among Firefighters at Prescribed Forest Burns in the Southeastern United States

K. H. Dunn, MSEE, CIH; S. Shulman, PhD; A. L. Stock, PhD, MPH; L. P. Naeher, PhD

ABSTRACT. Exposure to combustion products from wildland fires causes respiratory irritation and decreased lung function among firefighters. The authors evaluated carbon monoxide (CO) exposures of a group of wildland firefighters who conducted prescribed burns in the southeastern United States of America. A total of 149 person-days of samples were collected using data logging CO monitors. A questionnaire was administered to collect data on job tasks and self-reported smoke exposure. Overall, the highest exposures were seen amongst firefighters assigned to holding and mop-up tasks (geometric mean [GM]: 2.6 ppm), whereas the lowest were associated with lighting and jobs such as burn boss (GM: 1.6 and 0.3 ppm, respectively). The self-reported smoke exposure showed a significant linear trend with increasing CO exposure. The numbers of acres burned or burn duration, however, were not good predictors of exposure.

KEYWORDS: carbon monoxide, exposure assessment, prescribed burns, wildland firefighter

Controlled fires are used extensively as a part of forest management objectives throughout the United States of America. These prescribed burns are used to reduce the risk of wildfire by reducing natural fuel load, to control undergrowth to allow for regeneration of desirable plants, and to control insects and diseases. In 2010, over 2 million acres of land were treated with prescribed burns in the United States.[1] The current study was conducted on the Savannah River Site (SRS), a National Environmental Research Park located in the southeastern coastal area of the United States encompassing 198,000 acres bordering on the Savannah River. In terms of biodiversity, the SRS is about 21% hardwood or mixed pine hardwood and 65% pine along with meadowlands and swampy areas.[2] Approximately 20,000 acres of the SRS are treated with prescribed burns annually.[3]

Many air toxins are produced during fires, including carbon monoxide (CO), particulate matter, hydrocarbons, and nitrogen oxides. Studies of firefighters have shown that exposure to smoke from fires results in respiratory irritation, lung inflammation, eye, nose, and throat irritation, and headache as well as decreases in lung function.[4–14] Previous studies of wildland firefighters have shown that the highest measured exposures were to particulate matter and CO.[15–17] Some studies have also shown that exposure to CO is useful as a marker of overall exposure to toxic air contaminants, since it is significantly correlated with acrolein, benzene, formaldehyde, and respirable particulates.[15,16] The ability to estimate

K. H. Dunn *and* **S. Shulman** *are with the National Institute for Occupational Safety and Health, Cincinnati, Ohio, USA.* **A. L. Stock** *is with the National Center for Environmental Health, Centers for Disease Control, Atlanta, Georgia, USA.* **L. P. Naeher** *is with the Department of Environmental Health Science, College of Public Health, University of Georgia, Athens, Georgia, USA.*

exposure to smoke is important in determining potential health effects in occupational populations.

As part of a study of smoke exposure in wildland firefighters in the southeastern United States, personal monitoring for exposure to CO was performed. Quantitative and qualitative measures of exposure to smoke were collected and analyzed. In this paper, we present the results of the CO exposure monitoring as it relates to other explanatory variables, including job task, acres burned, burn duration, and self-assessed smoke exposure.

METHODS

Personal sampling

A crew of 20 nonsmoking firefighters was followed for 2 consecutive dormant (winter) burn seasons from 2004 to 2005. Participation in the study was voluntary. Informed consent was obtained from each firefighter participating in the study in accordance with the University of Georgia (UGA) and Centers for Disease Control and Prevention (CDC) institutional review boards. The overall group included 17 men and 3 women with an average age of 29 years (median 28 years, range 21–44 years). The fire crew was a local initial attack/prescribed fire crew stationed at the SRS that conducted prescribed burns. A sample of up to 11 firefighters was chosen each prescribed burn day for monitoring. Over the 2-year study period, there were a total of 19 burn days during which firefighters were sampled, providing 148 total person-days CO samples. Real-time CO exposure was measured on an average of 7.8 firefighters per burn day for an average shift length of 10.8 hours (median 10.5 hours, range 7–19 hours). The time spent at the burns ranged from 2 to 16.5 hours with an average of 6.7 hours/shift. The prescribed burns covered a range of 28 to 2745 acres (11 to 1111 hectares) with an average of 822 acres per burn (333 hectares).

Draeger PAC III CO monitors (Draeger Safety, Pittsburgh, PA) were attached to the gear pack and placed in the breathing zone of each firefighter. These monitors use an electrochemical cell for detection, have a range of 0 to 2000 parts per million (ppm), and are direct-reading instruments with data logging capabilities. The monitors were calibrated with a 200-ppm CO certified gas standard (Calgaz; Air Liquide America, Cambridge, MA) prior to the start of the study and were zeroed with ambient air at the forest station at the beginning of each shift. Monitor response was checked with 200-ppm calibration gas at the end of each shift.

A postshift questionnaire was used to collect information on daily activities, including a qualitative assessment of the smoke exposure based on a subjective scale (none to very little, low level, medium level, medium to high level, or high level). In addition, each firefighter provided a time and activity log that accounted for all tasks performed during the shift. This log was used to estimate the amount of time spent at the burn site versus that time spent in the office in preparation for prescribed burn activities. Each participant was also asked to provide information on the specific jobs that they performed while at the burn site, including the following:

Lighting—workers performing this task walked along the fire line using hand-held drip torches to ignite woody undergrowth.
Holding—workers performing this job worked to maintain the fire within established boundaries using tools such as a rake and water hose.
Mop-up—workers performing this task used hand tools and water to extinguish any smoldering debris following the completion of prescribed burns.
Other/burn boss—workers performing these tasks were located further from the fire line and were responsible for supervision of prescribed burn activities or other activities such as monitoring the fire or lighting via a helicopter.

The burn average CO concentrations were based on these questionnaires and may have overestimated or underestimated true time at the burn site due to problems with recall. However, real-time CO concentration traces taken at the time the participant indicated being at the burn site were compared with the real-time CO exposure data in order to identify any gross errors. Some firefighters changed jobs during the shift and were coded according to the job/task that was performed for a majority of the shift. This may have resulted in a dilution of exposure across several jobs/task, making differences between these tasks more difficult to assess.

Data analysis

Time-weighted average (TWA) CO concentrations were calculated for full-shift exposure. A "burn average" TWA was also calculated that accounted for exposure only during the portion of the shift that the firefighter was at the prescribed burn site. Instantaneous CO concentrations (data logged each second) were compared with the National Institute for Occupational Safety and Health (NIOSH) ceiling limit—a value that "should not be exceeded at any time."[18] Bonferroni multiple-comparison tests were conducted to evaluate the differences in TWA exposures according to each job/task category.

Data were analyzed using SAS version 9.0 (SAS Institute, Cary, NC). The data were log transformed and mixed-effect statistical models were fitted to the carbon monoxide full-shift average and burn average data. For the burn average data, because of the presence of zero values, 0.14 was added to each value before log-transforming—the use of different values has little effect on results. The data were treated as a randomized block design, with each burn compartment (land areas were divided into numbered compartments) as a random sample from a larger set. Variance terms, due to random effects,

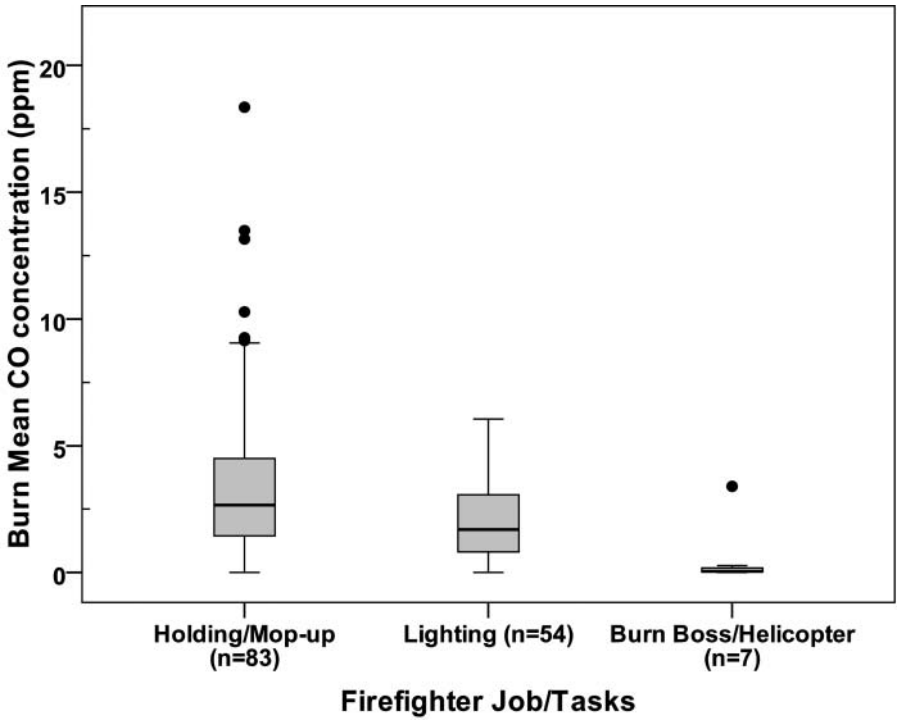


Fig. 1. Firefighter average carbon monoxide exposures while at the fire by job category/task. For each job category, the plotted symbols are as follows: (1) the solid line near the middle of the box is the median; (2) the lower and upper sides of the box are, respectively, the 25th and 75th percentiles; (3) the dashed lines extend to 1.5 times the interquartile range (IQR) beyond the 25th and 75th percentiles; and (4) isolated points are 1.5 (IQR) beyond either the 25th or 75th percentile.

were included for date, person, compartment, job activity by compartment, and date by job activity.

Simplified exposure determinant models were obtained by removing factors not statistically significant at the 5% level, based on likelihood fitting procedures. The final models included random effects for person and compartment. Residual plots were reviewed for the selected models and 1 of the 148 observations was removed because it resulted in an unacceptably large residual (representing an outlier). Full-shift and burn average TWAs were modeled to evaluate potential explanatory variables, including job activity, self-reported smoke exposure level, acres burned, duration of burn, and shift length.

RESULTS

Although instantaneous CO exposures exceeded the NIOSH ceiling limit of 200 ppm in 6 samples, overall shift average concentrations were low (GM 1.06 ppm, range 0–14 ppm) as much of the shift was spent preparing for and traveling to/from the fires. The burn mean CO concentrations were also low (GM 1.34 ppm, range 0–18 ppm) and are shown according to primary job tasks in Figure 1.

For the burn average data, model estimates are shown in Table 1. Lighting tasks produced exposures between those of the 2 extreme categories: holding and mop-up the highest and burn boss/helicopter the lowest (Figure 1), Each of these are statistically distinguishable (5% level) by Bonferroni inequalities. Results are similar for both the full-shift CO and the burn average data. The only significant factors included in the model were job task and self-assessed exposure level,

Table 1.—Mixed-Effect Model Coefficients for Factors Affecting Burn Average Exposure to Carbon Monoxide Among Firefighters at Prescribed Burns

Parameters	Coefficient estimate	*p* value
Intercept	−1.34	.0395
Job tasks		
Holding/mop-up ($n = 83$)	2.04	<.0001
Lighting ($n = 58$)	1.68	
Other/burn boss ($n = 7$)	Reference	
Self-assessed exposure level		
High level ($n = 9$)	0.96	<.0001
Low level ($n = 90$)	−0.59	
Medium level ($n = 49$)	Reference	
Burn duration	−0.031	.53
Acres burned	−0.00015	.24
Shift length	0.074	.31

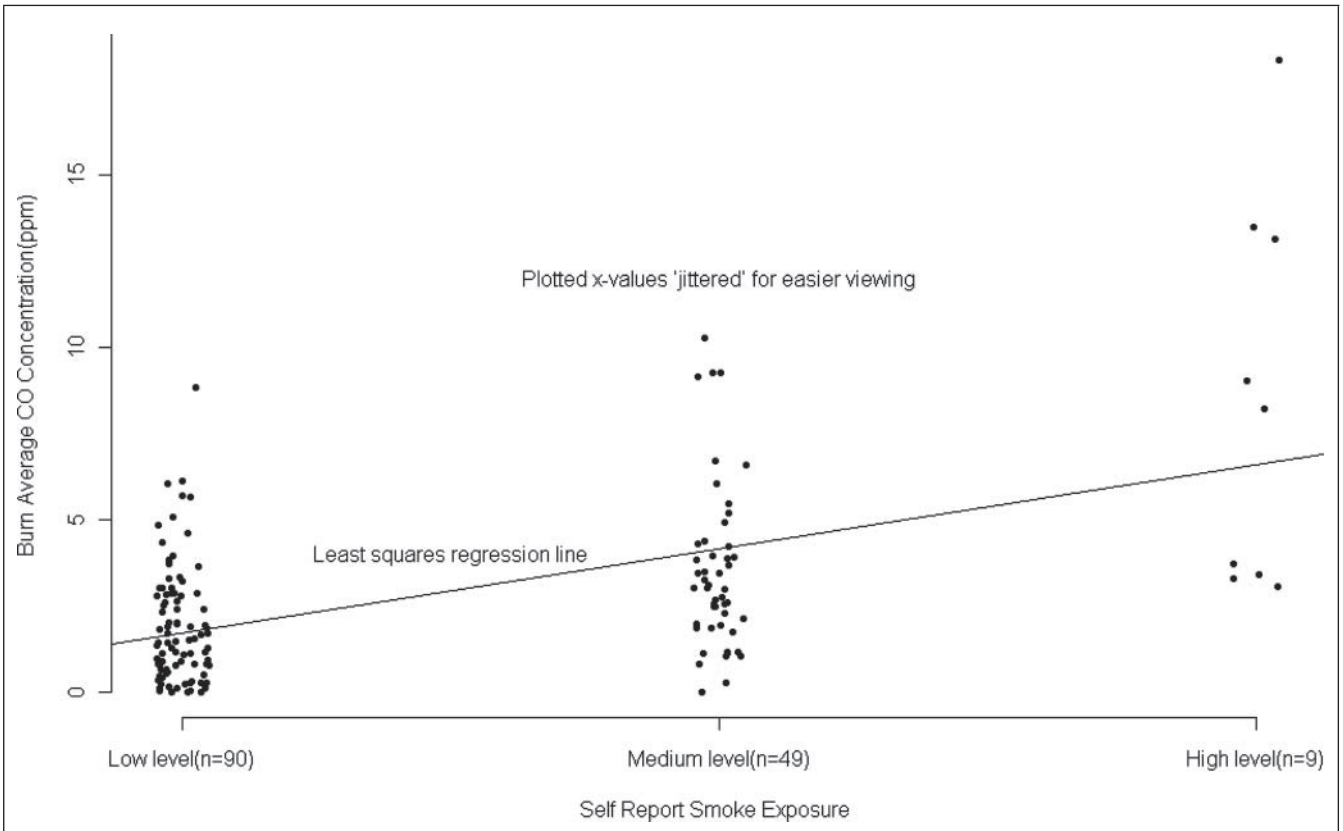


Fig. 2. Comparison of self-reported smoke levels with burn average CO concentrations.

although other factors are shown in Table 1 for informational purposes.

The mean CO concentrations for the burn average data for each self-reported smoke exposure level are shown in Figure 2. Overall, the mean exposure levels increased linearly ($p < .01$) with increasing qualitative assessment.

COMMENT

The results of this study indicate that the ascertainment of job task and self-assessment of exposure may help rank exposures among groups of firefighters. The results presented here were similar to the few published studies of wildland firefighters.[15–17,19] These studies have indicated that exposures to carbon monoxide have generally been lower than relevant occupational exposure limits. The job tasks with the highest exposures were holding and mop-up, whereas burn boss produced the lowest exposures. The results presented here indicate that the self-report scale used by firefighters can be useful for estimating carbon monoxide exposure. However, there is considerable unexplained variation in carbon monoxide exposure within each self-report category.

****

Funding and support was provided by the Department of Energy Savannah River Operations Office through the US Forest Service Savannah River under Interagency Agreement DE-AI09–00SR22188. The authors gratefully acknowledge Jeff Prevey, Paul Linse, Mark Frizzell, John Blake, Gary Achtemeier, Dan Shea, Chris Hobson, Jason Demas, and the firefighters for support and participation in this study. The findings and conclusions in this manuscript are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention. Mention of company names or products does not constitute endorsement by the Centers for Disease Control and Prevention.

For comments and further information, address correspondence to Luke Naeher, Department of Environmental Health Science, College of Public Health, University of Georgia, 206 Environmental Health Science Building, Athens, GA 30602-2102, USA.

E-mail: LNaeher@uga.edu

****

References

1. National Interagency Fire Center. Fire Information—wildland fire statistics. 2010. Available at: http://www.nifc.gov/fire_info/ytd_state.htm. Accessed August 14, 2010.
2. US Department of Energy. Natural resources management plan for the Savannah River Site. May 2005; Available at: http://www.fs.usda.gov/Internet/FSE_DOCUMENTS/stelprdb5208304.pdf.Accessed August 21, 2011.
3. US Forest Service. Facts about Savannah River Site: USDA Forest Service–Savannah River (USFS-SR). Available at: http://www.srs.gov/general/news/factsheets/usfs-sr.pdf. Accessed April 20, 2011.
4. Naeher LP, Brauer M, Lipsett M, et al. Woodsmoke health effects: a review. *Inhal Toxicol.* 2007;19:67–106.
5. Harrison R, Materna BL, Rothman N. Respiratory health hazards and lung function in wildland firefighters. *Occup Med.* 1995;10:857–870.

6. Liu D, Tager IB, Balmes JR, Harrison RJ. The effect of smoke inhalation on lung function and airway responsiveness in wildland fire fighters. *Am Rev Respir Dis.* 1992;146:1469–1473.
7. Slaughter JC, Koenig JQ, Reinhardt TE. Association between lung function and exposure to smoke among firefighters at prescribed burns. *J Occup Environ Hyg.* 2004;1:45–49.
8. Rothman N, Ford DP, Baser ME, et al. Pulmonary function and respiratory symptoms in wildland firefighters. *J Occup Med.* 1991;33:1163–1167.
9. Betchley C, Koenig JQ, van Belle G, Checkoway H, Reinhardt T. Pulmonary function and respiratory symptoms in forest firefighters. *Am J Ind Med.* 1997;31:503–509.
10. Mustajbegovic J, Zuskin E, Schachter EN, et al. Respiratory function in active firefighters. *Am J Ind Med.* 2001;40:55–62.
11. Swiston JR, Davidson W, Attridge S, Li GT, Brauer M, van Eeden SF. Wood smoke exposure induces a pulmonary and systemic inflammatory response in firefighters. *Eur Respir J.* 2008;32:129–138.
12. Gaughan DM, Cox-Ganser JM, Enright PL, et al. Acute upper and lower respiratory effects in wildland firefighters. *J Occup Environ Med.* 2008;50:1019–1028.
13. Booze TF, Reinhardt TE, Quiring SJ, Ottmar RD. A screening-level assessment of the health risks of chronic smoke exposure for wildland firefighters. *J Occup Environ Hyg.* 2004;1:296–305.
14. Serra A, Mocci F, Randaccio FS. Pulmonary function in Sardinian fire fighters. *Am J Ind Med.* 1996;30:78–82.
15. Materna BL, Jones JR, Sutton PM, Rothman N, Harrison RJ. Occupational exposures in California wildland fire fighting. *Am Ind Hyg Assoc J.* 1992;53:69–76.
16. Reinhardt TE, Ottmar RD. Baseline measurements of smoke exposure among wildland firefighters. *J Occup Environ Hyg.* 2004;1: 593–606.
17. Reisen F, Hansen D, Meyer CP. Exposure to bushfire smoke during prescribed burns and wildfires: firefighters' exposure risks and options. *Environ Int.* 2011;37:314–321.
18. National Institute for Occupational Safety and Health. *Pocket Guide to Chemical Hazards and Other Databases: Immediately Dangerous to Life and Health Concentrations*. Washington, DC: Department of Health and Human Services, National Institute for Occupational Safety and Health; 2000. Publication no. 2000–130.
19. Reh C, Letts D, Deitchman S D. U.S. Department of the Interior, National Park Service, Yosemite National Park, CA. Health Hazard Evaluation Report No. HETA-90–0365-2415. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, 1994.

HHS Public Access

Author manuscript

J Occup Environ Hyg. Author manuscript; available in PMC 2018 August 20.

Published in final edited form as:

J Occup Environ Hyg. 2017 September ; 14(9): 739–748. doi:10.1080/15459624.2017.1326700.

Lung function measures following simulated wildland firefighter exposures

Matthew D. Ferguson[1], **Erin O. Semmens**[1], **Emily Weiler**[1], **Joe Domitrovich**[2], **Mary French**[1], **Christopher Migliaccio**[1], **Charles Palmer**[3], **Charles Dumke**[3], and **Tony Ward**[1,*]

[1]Center for Environmental Health Sciences, University of Montana, Missoula, Montana, USA

[2]United States Forest Service, Missoula, Montana, USA

[3]Department of Health and Human Performance, University of Montana, Missoula, Montana, USA

Abstract

Across the world, biomass smoke is a major source of air pollution and is linked with a variety of adverse health effects. This is particularly true in the western US where wood smoke from wildland forest fires are a significant source of $PM_{2.5}$. Wildland firefighters are impacted as they experience elevated $PM_{2.5}$ concentrations over extended periods of time, often occurring during physical exertion. Various epidemiological studies have investigated wood smoke impacts on human health, including occupational field exposures experienced by wildland firefighters. As there are numerous challenges in carrying out these field studies, having the ability to research the potential health impacts to this occupational cohort in a controlled setting would provide important information that could be translated to the field setting.

To this end, we have carried out a simulated wildland firefighter exposure study in a wood smoke inhalation facility. Utilizing a randomized crossover trial design, we exposed 10 participants once to clean filtered-air, 250 $\mu g/m^3$, and 500 $\mu g/m^3$ wood stove-generated wood smoke $PM_{2.5}$. Participants exercised on a treadmill at an absolute intensity designed to simulate wildland firefighting for 1.5 hours. In addition to measured $PM_{2.5}$ smoke concentrations, mean levels of CO_2, CO, and % relative humidity were continuously monitored and recorded and were representative of occupational 'real-world' exposures. Pulmonary function was measured at three time points: before, immediately after, and 1-hour post-exposure. Although there were some reductions in FVC, FEV_1, and FVC:FEV_1 measures, results of the spirometry testing did not show significant changes in lung function. The development of this wood smoke inhalational facility provides a platform to further address unique research questions related to wood smoke exposures and associated adverse health effects.

Keywords

wood; smoke; wildland; firefighting; inhalation; spirometry

*Corresponding Author: Address: University of Montana, 32 Campus Drive, Skaggs Building Room 176, Missoula, MT 59812, Phone: (406) 243-4092, Fax: (406) 243-2807, tony.ward@umontana.edu.

INTRODUCTION

Air pollution has a major impact on human health throughout the world, and is a leading cause of premature mortality [1–3]. Both globally and throughout the US, urban areas with large populations often have elevated levels of air pollution, including airborne particulate matter (PM). This is also true for rural areas of the northern Rocky Mountain region of the US, where biomass burning (e.g., wildland fires, wood stoves, etc.) is a major source of elevated ambient and indoor $PM_{2.5}$ concentrations throughout the year [4–16].

Globally, smoke from wildland fires partly attribute to around 339,000 deaths a year [17]. Tens of thousands of wildland fires burn between 3 million and 10 million acres of land depending on year in the US alone. These fires are predicted to continue or worsen in many regions throughout the world [18]. During these wildland fire events, emissions of wood smoke $PM_{2.5}$ can impact ambient air quality in communities thousands of kilometers downwind [20]. It can also infiltrate homes resulting in indoor PM concentrations similar to levels observed outside [21–24].

With predicted increases in forest fires [18], the number of wildland firefighter crews deployed to fight these fires will also increase. These crews have been shown to experience smoke PM levels up to 2,930 $\mu g/m^3$ with average levels of exposure during wildland firefighting activities ranging from 509–558 $\mu g/m^3$, and average CO levels of exposure ranging from 1.3–1.7 ppm [25]. When working on project fires or prescribed burns, firefighters can experience average concentrations ranging from 500–630 $\mu g/m^3$ throughout an entire work shift [26]. Related health studies conducted in the field have found an overall general decrease in lung function following wildland firefighting activities [27–29].

Given the many sources of biomass smoke exposures leading to a variety of exposure scenarios, there is a need to study the health effects following similar smoke exposures in a controlled environment. Wildland firefighter exposures are unique due to sustained and elevated $PM_{2.5}$ wood smoke concentrations while also enduring considerable physical exertion. The objective of this study was to deliver wood smoke $PM_{2.5}$ (generated in wood stove) in a controlled facility to 10 human participants and determine respiratory impacts under simulated conditions typically experienced (i.e., physical and atmospheric) by wildland firefighters. Below we describe the design and methods utilized in carrying out the exposure trials and present exposure and lung function results.

METHODS

The Inhalation and Pulmonary Physiology Core within the Center for Environmental Health Sciences at the University of Montana was originally developed to conduct wood smoke exposure trials using mice, but was modified in this application to conduct human exposure trials. The study described herein is a 10-participant pilot project simulating occupational wood smoke exposures encountered by wildland firefighters. Following recruitment into the study and initial entry level measurements (Day 1), 10 individuals participated in three experimental trials, each one occurring one week apart (i.e., Day 2, Day 3, and Day 4). Each participant was blinded to his exposure assignment and was exposed, while exercising, once

to either clean filtered-air (0 μg/m^3), 250 μg/m^3, or 500 μg/m^3 wood smoke $PM_{2.5}$, in random order for 1.5 hours. Throughout each exposure, $PM_{2.5}$, CO, CO_2, and % relative humidity levels were continuously monitored and recorded. Spirometry measures were collected prior to, immediately after, and 1-hour post each exposure.

Inhalation Facility and Exposure Levels

Wood smoke for these exposure trials was generated using an older-model wood stove (Englander, England Stove Works, Inc., Monroe, VA) and routed through dilution chambers before ultimate delivery to the participant through a breathing mask. The wood used in this study was cured (~15% moisture content) western larch (*Larix occidentalis* Nutt.), which is a common species in western Montana. The technique for building and maintaining the fire was uniform throughout each exposure. Prior to each exposure trial, some remaining ash from previous burns was removed for a consistent starting ash depth (~0.5–1" deep). Each fire started with about 1 kg of wood as well as kindling (1–2 pages of newspaper). About 300 g of wood was then added every 15–20 minutes over a two-hour period, with each fire started 25–30 minutes prior to each exposure trial.

During each exposure trial, smoke pulled from the wood stove chimney was routed through dilution chambers where filtered air (Cambridge Absolute Filter, Cambridge Filter Corp., Syracuse NY) was introduced in an effort to dilute the smoke (FIGURE 1). Wood smoke was then delivered from the dilution and mixing chambers to the participant via a modified mask respirator (Hans Rudolph, Inc., Shawnee, Kansas). The major pump pulling air from the wood stove chimney and through the dilution chambers was placed in line between the chambers and the mask. This allowed air to be 'pushed' through the mask at rates (~90–100 L/min) appropriate for an individual to comfortably breath while exercising on a treadmill.

En route to the mask and following the pump, wood smoke PM first passed through 2.5 feet of flex tubing before coming to a T-valve that directed the wood smoke PM to both the mask and to a fume hood where excess wood smoke PM was exhausted. Tubing to the mask included 108" of Clean-Bor tubing (VacuMed, Ventura, CA) made of ethylene vinyl acetate. The mask utilized was a Rudolph Nasal & Mouth Breathing Face Mask with a two-way non-rebreathing T-valve. Another hose exited the mask and was directed to the fume hood for exhaust.

The exposure room (11'10" × 5'10" × 8') contained a treadmill (Model Q65, Quinton Instrument Company, Bothell, WA) attached to a control station (Model Q4000, Quinton Instrument Company, Bothell, WA), and other items intended for participant comfort. This included a fan to improve air circulation in the room. A stand within arm's reach and at eye level was also placed in front of the treadmill providing a platform for the participant to set a magazine, book, tablet, or phone. If desired, participants were also allowed to listen to music throughout their exposure trial. For comfort, the mask and tubes were suspended from the ceiling by adjustable straps. This allowed the mask to be placed at an appropriate height, reducing the burden of mask and tubing weight on the participant's head and face, and allowed the mask to move and shift with the participant while they were exercising on the treadmill.

Two $PM_{2.5}$ monitors (DustTrak, TSI, Model 8530 and Model 8534, Shoreview, MN) were used during the exposures to measure continuous readings of real-time and average $PM_{2.5}$ concentrations directly routed to the mask (see Figure 1). The first DustTrak (Model 8534) was used to adjust wood smoke $PM_{2.5}$ concentrations delivered through the dilution chambers. The second DustTrak (Model 8530) measured continuous $PM_{2.5}$ concentrations delivered to the mask just prior to inhalation. All $PM_{2.5}$ concentrations reported in this manuscript were obtained from this second DustTrak. Carbon monoxide (CO), carbon dioxide (CO_2), and % relative humidity at the mask were also monitored with a Q-Trak (TSI, Model 7565, Shoreview, MN) and collocated to the second DustTrak. This CO measurement was especially important to ensure low levels of CO during each exposure trial.

Inclusion Criteria and Recruitment

This study included 10 healthy, non-smoking males, aged 18–40 years, with no pre-existing chronic lung diseases. Participants did not have wood smoke exposures at home or work (via cigarettes or wood stoves), and had to complete a moderate physical exercise protocol three times during the study. Due to the small size of this pilot study, and to remove the potentially confounding impact of gender on findings, only males were included. Additional inclusion criteria described in more detail below under 'Day 1' included answering 'No' to all questions on a Physical Activity Readiness Questionnaire (PARQ), as well as having a VO_2 max > 40 ml/kg/min.

Following study approval from the University of Montana's Institutional Review Board, participants were recruited from the University of Montana student, faculty, and staff population. Flyers were posted throughout the campus. Upon the initial meeting with participants, enrolled volunteers were administered oral and written informed consent, and then scheduled for Day 1 measures. Participants received a stipend upon completion of each of the three exposure trials (Days 2–4, respectively).

Day 1

Day 1 of the study was used to determine eligibility for the Days 2–4 exposure trials, with inclusion/exclusion criteria intended to reduce the risk of adverse response occurrences throughout each exposure. Participants were reminded to fast for three hours before presenting. They were then asked to complete a personal information questionnaire and PARQ, and undergo a test to verify their maximum level of oxygen uptake (VO_2 max) was greater than 40 ml/kg/min. The percentage of body fat for each participant was also determined via an underwater weighing test. Personal information collected included age, height, weight, percentage of body fat, VO_2 max, and illnesses and medications taken during the study period. If participants met all the inclusion criteria, scheduling was initiated for Days 2, 3, and 4. The entire process for Day 1 took approximately 1.25 hours/participant.

Day 2–4 Exposure Trials

Following the Day 1 evaluations, participants participated in three exposure trials, each one occurring one-week apart (i.e., Day 2, Day 3, and Day 4). During the study, each participant was exposed once to either clean filtered air, 250 $\mu g/m^3$, or 500 $\mu g/m^3$ wood smoke $PM_{2.5}$ in

a double-blind randomized crossover design. During smoke exposure, participants were asked to walk on a treadmill at a set rate and incline (3.5 mph and 5.7% grade) for 1.5 hours to simulate working on a fireline, with a short (e.g., 20–30 seconds) break every 15 minutes to evaluate perceived stress and drink a predetermined amount of water. A researcher was constantly monitoring both the CO and $PM_{2.5}$ concentrations to the mask and signs of participant discomfort at all times during the exposure trials. Each of the three experimental trials took approximately 3 hours.

Pulmonary Function

Spirometry is the most widely used assessment of pulmonary function for diagnosis and prognosis of pulmonary status and disease, including chronic obstructive pulmonary disease (COPD) and other restrictive diseases [30– 33]. Evaluated spirometry measures used in this study include the volume of exhaled breath during the first second of forced expiratory air following maximum inhalation (FEV_1), the vital capacity (FVC; the maximum volume of air forced out of the lungs following maximum inhalation), and the ratio FEV_1/FVC (also known as the Tiffeneau-Pinelli index). For each of the three trials, spirometry measurements were collected from the participants before, immediately post-exposure, and 1-hour after each exposure. Each assessment was conducted by having the participant blow air rapidly and forcefully into the mouthpiece of a Koko Legend Spirometer (Ferraris Respiratory, Louisville, CO). To ensure accurate and reliable results during the pulmonary function test, a strict protocol was followed that included both specific participant instructions as well as quality control measures [34].

Quality Assurance / Quality Control

The DustTrak was zero calibrated prior to each exposure trial. The tubes connecting the flex tubing to the mask were replaced following each exposure trial, and the mask was thoroughly cleansed after each exposure. This was done by disassembling the mask, with each part thoroughly washed in warm water with mild detergent. This ensured that participants were equipped with a clean mask at the start of each exposure. The building of each fire was consistent using standardized procedures (e.g., starting mass, stoking mass, etc.). To further reduce source variability, the same people conducted the fire loading and stoking throughout the entire study.

Data Analysis

Each participant was randomly assigned an identification number at the start of the study, with all samples, questionnaire responses, physiological measurements, and other data collection forms labeled with this number. We define exposure as 1) filtered-air, 2) 250 $\mu g/m^3$ wood smoke $PM_{2.5}$, or 3) 500 $\mu g/m^3$ wood smoke $PM_{2.5}$. Due to skewness in the distributions, the presence of outliers, and the small sample size for pulmonary function measures in this pilot project, we utilized the Skillings-Mack test, a nonparametric analog to a repeated measures ANOVA that allows for unbalanced data, to evaluate if observed pre- to post-exposure changes in lung function differed significantly by wood smoke exposure condition. Comparisons were also made using Dunnett's test. These tests were performed using Excel and Prism (GraphPad, v.5.0a).

RESULTS

Environmental conditions and spirometry results are reported below. Also, due to fatigue in one participant and another participant dropping out before their final exposure, two individuals did not complete all spirometry measures.

Day 1 Measures

The participants had an average age of 26.4 (± 3.7). Average height (in inches) of all participants was 70.13 (± 3.1). The body weight (kg) and percent body fat outcomes were 79.03 (± 12.2) and 14.16 (± 2.6), respectively. All participants showed acceptable VO_2 max levels (ml/kg/min) following Day 1 test measures at 53.53 (± 7.2). No illnesses or medications were reported prior to Day 1 measures.

Exposure Concentrations

We were able to successfully deliver consistent, reproducible exposures in the wood smoke inhalation facility. FIGURE 2 presents an example of a participant's delivered smoke $PM_{2.5}$ concentrations at the mask throughout the 250 μg/m^3 and 500 μg/m^3 exposure trials. Across all trials, the average measured concentrations of $PM_{2.5}$ from filtered-air, 250 μg/m^3, and 500 μg/m^3 exposures were 5.2 (±4.9) μg/m^3, 253.9 (± 5.8) μg/m^3, and 506.2 (± 4.8) μg/m^3, respectively. Greater than 99% of $PM_{2.5}$ mass measured in the dilution chamber was in the PM_1 fraction (as measured by the DustTrak Model 8534, data not shown). The average levels of CO from all filtered-air, 250 μg/m^3, and 500 μg/m^3 exposures were 0.003 (± 0.007) ppm, 0.87 (± 0.28) ppm, and 1.87 (± 0.65) ppm, respectively. Mean levels of CO_2 from all filtered-air, 250 μg/m^3, and 500 μg/m^3 exposures were 443 (± 22) ppm, 464 (± 28) ppm, and 482 (± 21) ppm, respectively. From all filtered-air, 250 μg/m^3, and 500 μg/m^3 exposures, relative humidity was 14.1 (± 8.4) %, 12.1 (± 5.3) %, and 13.1 (± 2.7) %, respectively.

Lung Function

As presented in TABLE I, calculated "change from pre-exposure values" included normalizing each participant's post- and 1-hour post-e xposure spirometry values (FVC, FEV_1, and FVC:FEV_1) to their perspective pre-exposure levels, for each of the three exposures. This included subtracting pre-exposure values from post- and 1-hour post-exposure, for each individual exposure. This normalization decreases within-participant day-to-day variation, as well as between-participant variation.

Overall, spirometry results showed no significant changes following wood smoke $PM_{2.5}$ exposures (TABLE I). The mean pre-exposure FVC results ranged from 5.41 (0.53) - 5.61 (0.93) liters. There was no impairment in lung function measured at the post-exposure time point, but there were slight reductions in FVC for the 250 μg/m^3 and 500 μg/m^3 exposures at the 1-hour post time point (−0.07 and −0.04 liters, respectively). This same trend is observed in the FEV_1 measures. Pre-exposure baseline FEV_1 measures ranged from 4.37 (0.34) - 4.55 (0.64) liters. Following the exposures, there was a post-exposure reduction at the 250 μg/m^3 trial (−0.19 liters), and reductions of −0.27 liters (250 μg/m^3) and −0.05 liters (500 μg/m^3) measured in the 1-hour post exposure spirometry tests. Consistent with the FEV_1 measures, we saw insignificant reductions in the ratio of FVC:FEV_1 post-exposure at the 250 μg/m^3

trial (−3.40), and insignificant reductions in 1-house post exposure measures for 250 μg/m^3 (−3.02) and 500 μg/m^3 (−0.29) exposure trials.

DISCUSSION

One goal of this pilot project was to deliver specific concentrations of $PM_{2.5}$ wood smoke that simulated occupational exposures encountered by wildland firefighters. As demonstrated in FIGURE 2, the measured concentrations of $PM_{2.5}$ were consistent with (and representative of) field research studies where PM levels (and CO concentrations) were recorded during wildland firefighting activities [25]. Exposure concentrations are also representative of exposures encountered in other settings, providing future opportunities to investigate other exposure scenarios. The lower concentration of wood smoke $PM_{2.5}$ exposure in this study (250 μg/m^3) is comparable to concentrations recorded when biomass is burned for cooking or heating purposes in homes without ventilation [35], and consistent with concentrations used in other European human/biomass smoke exposure studies [36–43]. The higher level of exposure (500 μg/m^3) is comparable to human exposure studies conducted by the Environmental Protection Agency's Ghio et al. [44], where participants were exposed to an average concentration of 485 μg/m^3 over a 2-hour period. Similarly, occupational studies have reported average wood smoke PM exposures (i.e., wildland fire firefighters) in the range of 500–800 μg/m^3 [25, 26, 45]. These same studies reported average CO and CO_2 levels ranging about 1–7 ppm and 400–500 ppm, respectively.

Health Effects Associated with Wood Smoke Exposure

Most of the current knowledge regarding the adverse health effects (both acute and chronic detriments) following wood smoke exposures have come from epidemiologal studies. Ambient wildland fire PM levels exceeding 40 μg/m^3, relative to concentrations less than 10 μg/m^3 are associated with more than a doubling of observed asthmatic presentations [46]. Other observations following similar events included increased risk of allergic respiratory disease, as well as bronchial asthma, exacerbation of type II diabetes [47] and cardiovascular disease [48, 49]. Significant decreases in lung function were reported in several studies following occupational exposures (e.g. wildland firefighting) to wood smoke PM [27, 45, 50]. Additional studies have shown a general increase in emergency room and outpatient visits during and following smoke events [49, 51-54].

Human Exposure Wood Smoke Studies

The limited number of studies involving human exposures to wood smoke PM in controlled environments show varying results. TABLE II presents a summary of human wood smoke studies that have been conducted in a variety of settings. Throughout the literature, PM levels in the human exposure studies ranged from around 150 to 1000 μg/m^3, with durations of exposures from 1 to 4 hours. Studies reported a varying degree of physical activity throughout the trials, from sedentary 3-hour exposures [55] to riding an exercise bike at light effort (~70 W) for two 25-minute periods during a 4-hour exposure [$PM_{2.5}$ concentrations 243–279 μg/m^3 [43]]. The majority of the parameters (e.g., PM concentrations, duration of exposure, etc.) used in the present pilot study were within the range of those outlined in TABLE II. The route of exposure (using a mask to deliver the exposure) and the exercise

component (briskly walking on a treadmill at a set rate and incline (3.5 mph and 5.7% grade) for 1.5 hours) are notable differences between our study and those summarized in TABLE II.

Spirometry results from our study are consistent with previous studies [44, 56, 39] where, in healthy individuals, no significant changes in lung function were observed following controlled acute wood smoke exposures. Previous field studies investigating the influence of wildland fire smoke on wildland firefighter lung function, on the other hand, have shown significant effects from smoke inhalation [27, 28]. Liu et al. [28] gathered spirometry data from sixty-three "seasoned" firefighters before and after a full season of fighting wildland fires. Significant declines in mean FVC and FEV_1 values were observed post-season (0.09 and 0.15 L/s, respectively). In a comparable study, Betchley et al. [27] observed similar declines in FVC and FEV_1 in a cohort of seventy-six volunteers following a full season of fighting wildland fires. Compared to our study conducted in a controlled environment, these field studies had much longer exposure durations and higher wood smoke concentrations generated from multiple fuel types.

Study Limitations and Next Steps

The results of both epidemiological studies and the controlled human studies presented in TABLE II have demonstrated that there are a variety of adverse health effects following exposure to wood smoke. These studies have reported conflicting results and outcomes. Several of these studies found no significant pro-inflammatory responses [38, 39, 55], whereas increases were observed in others [42, 44, 56,]. Differences such as exposure levels, durations of exposure, varying physical activities, even biological media type (e.g. blood, EBC, etc.) and time of sample collection can partly explain these disparities. Also, the different lung function outcomes between short duration controlled exposures versus those following chronic exposures (i.e. wildland firefighters) suggest a possible role in exposure durations and recurrences. These outcomes should be considered in subsequent research trials.

As we designed this study to simulate exposures and environmental conditions experienced by wildland firefighters, exercise during the exposure trials was an important component of this study. As presented in TABLE II, about half of the aforementioned controlled human wood smoke exposure studies did not participate in an activity that might increase breathing and heart rate. In the studies where exercise was included, it was generally intermittent and non-strenuous. Importantly, the majority of observed effects occurred in participants that exercised intermittently during exposures. Also, due to the small size of this pilot study, only males were included. However, about 12%−16% of the wildland firefighter community is female [57]. We intend to incorporate both genders in future occupational studies.

CONCLUSION

As rising temperatures and shrinking snow pack have both been impacted by climate change, it is hypothesized that the frequency, magnitude, and intensity of wildland fires will increase during future summers. In a 2008 commissioned report, it was concluded that "the most important research question with respect to wildland fire particle emissions is the relationship between emission, acute and chronic exposure, and health effects" [58]. These

factors point to more research needed for wildland firefighters who are exposed to wood smoke $PM_{2.5}$ during occupational activities, as well as members of the public who are exposed to wildland and prescribed fire smoke in downwind populations.

Given the complexities (and dangers) of studying wood smoke exposures/health effects during actual wildland fire scenarios, our inhalational facility is novel in that it provides an opportunity to investigate human health effects following exposures to a range of relevant wood smoke PM concentrations during physical stress (including increased breathing rate) that simulates absolute intensity of a wildland firefighter. While focusing on firefighting activities, this study will also provide meaningful data on how wood smoke PM exposures might influence general and susceptible populations. In summary, this pilot study offers a unique method for delivering wood smoke PM at specific concentrations in a closed system. Controlling the physical exertion of our participants provides another innovative aspect of this study. The fire type, fuel type, mixing of filtered air with smoke effluent, and having direct control of those levels entering the mask provides a unique tool to answer important questions regarding human health impacts from wood smoke exposures. Future directions include evaluating systemic and pulmonary effects from these exposures, and investigating inflammatory outcomes and oxidative stress, including biomarkers of cardiovascular disease risk.

ACKNOWLEDGEMENTS

The authors thank Jed Syrenne, Britton Postma, Matthew Dorton, Kyle Cochrane, Emily Simpson, and Laura Porisch for data collection efforts. We are also grateful to Leon Washut and the Washut Endowment for Graduate Student Support in Biomedical Sciences at the University of Montana, as well as the study participants for the considerable time and effort put into this investigation. This research was supported by NCRR (COBRE P20RR 017670).

REFERENCES

1. Pope IC, Burnett RT, Thun MJ, et al.: Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 287(9): 1132–1141 (2002). [PubMed: 11879110]
2. Cohen AJ, Ross Anderson H, Ostro B, Pandey KD, Krzyzanowski M, Kunzli N et al.: The Global Burden of Disease Due to Outdoor Air Pollution. Journal of Toxicology and Environmental Health, Part A 68(13–14): 1301–1307 (2005). [PubMed: 16024504]
3. World Health Organization (WHO): 7 million premature deaths annually linked to air pollution. World Health Organization (2014).
4. U.S. Department of Energy: Space Heating in U.S. Homes, By Census Region, by U.S Energy Information Administration (EIA) Washington, D.C. (2009).
5. Air Quality Management Work Group: Recommendations to the Clean Air Act Advisory Committee: Phase I and Next Steps. Washington, D.C: Air Quality Management Work Group, USEPA 2005.
6. Ward TJ, and Lange T: The impact of wood smoke on ambient PM2.5 in northern Rocky Mountain valley communities. Environmental Pollution 158(3): 723–729 (2010). [PubMed: 19897293]
7. Naeher LP, Brauer M, Lipsett M, Zelikoff JT, Simpson CD, Koenig JQ et al.: Woodsmoke health effects: A review. Inhalation Toxicology 19(1): 67–106 (2007).
8. Larson T, Gould T, Simpson C, Liu LJS, Claiborn C, and Lewtas J: Source apportionment of indoor, outdoor, and personal PM2.5 in Seattle, Washington, using positive matrix factorization. Journal of the Air & Waste Management Association 54(9): 1175–1187 (2004). [PubMed: 15468670]

9. Ward TJ, and Smith GC: The 2000/2001 Missoula Valley PM2.5 chemical mass balance study, including the 2000 wildfire season - seasonal source apportionment. Atmospheric Environment 39(4): 709–717 (2005).
10. Noonan CW, Ward TJ, Navidi W, Sheppard L, Bergauff M, and Palmer C: Assessing the impact of a wood stove replacement program on air quality and children's health. Research report (162): 3–37; discussion 39–47 (2011).
11. Ward T, and Noonan C: Results of a residential indoor PM(2.5) sampling program before and after a woodstove changeout. Indoor Air 18(5): 408–415 (2008). [PubMed: 18665872]
12. Ward T, Boulafentis J, Simpson J, Hester C, Moliga T, Warden K et al.: Lessons learned from a woodstove changeout on the Nez Perce Reservation. Science of The Total Environment 409(4): 664–670 (2011). [PubMed: 21144555]
13. Allen RW, Leckie S, Millar G, and Brauer M: The impact of wood stove technology upgrades on indoor residential air quality. Atmospheric Environment 43(37): 5908–5915 (2009).
14. Noonan CW, Navidi W, Sheppard L, Palmer CP, Bergauff M, Hooper K et al.: Residential indoor PM2.5 in wood stove homes: follow-up of the Libby changeout program. Indoor Air 22(6): 492–500 (2012). [PubMed: 22607315]
15. Semmens EO, Noonan CW, Allen RW, Weiler EC, and Ward TJ: Indoor particulate matter in rural, wood stove heated homes. Environmental Research 138: 93–100 (2015). [PubMed: 25701812]
16. Jenkins PL, Phillips TJ, Mulberg EJ, and Hui SP: Activity Patterns of Californians - Use of and Proximity to Indoor Pollutant Sources. Atmospheric Environment Part a-General Topics 26(12): 2141–2148 (1992).
17. Johnston FH, Henderson SB, Chen Y, Randerson JT, Marlier M, Defries RS et al.: Estimated global mortality attributable to smoke from landscape fires. Environmental Health Perspectives 120(5): 695–701 (2012). [PubMed: 22456494]
18. McKenzie D, Shankar U, Keane RE, Stavros EN, Heilman WE, Fox DG et al.: Smoke consequences of new wildfire regimes driven by climate change. Earth's Future 2(2): 35–59 (2014).
19. Westerling A, Brown T, Schoennagel T, Swetnam T, Turner M, and Veblen T: Briefing: Climate and wildfire in western U.S. forests. In Forest conservation and management in the Anthropocene: Conference proceedings Proceedings. RMRS-P-71. Fort Collins, CO, Sample VAB, Patrick R(ed.), pp. p. 81–102: US Department of Agriculture, Forest Service. Rocky Mountain Research Station, 2014.
20. Le GE, Breysse PN, McDermott A, Eftim SE, Geyh A, Berman JD et al.: Canadian forest fires and the effects of long-range transboundary air pollution on hospitalizations among the elderly. ISPRS International Journal of Geo-Information 3(2): 713–731 (2014).
21. Barn P, Larson T, Noullett M, Kennedy S, Copes R, and Brauer M: Infiltration of forest fire and residential wood smoke: an evaluation of air cleaner effectiveness. Journal of Exposure Science and Environmental Epidemiology 18(5): 503–511 (2008). [PubMed: 18059421]
22. Henderson DE, Milford JB, and Miller SL: Prescribed burns and wildfires in Colorado: Impacts of mitigation measures on indoor air particulate matter. Journal of the Air & Waste Management Association 55(10): 1516–1526 (2005). [PubMed: 16295277]
23. Phuleria HC, Fine PM, Zhu YF, and Sioutas C: Air quality impacts of the October 2003 Southern California wildfires. Journal of Geophysical Research-Atmospheres 110(D7)(2005).
24. Sapkota A, Symons JM, Kleissl J, Wang L, Parlange MB, Ondov J et al.: Impact of the 2002 Canadian forest fires on particulate matter air quality in Baltimore City. Environmental Science & Technology 39(1): 24–32 (2005). [PubMed: 15667071]
25. Adetona O, Simpson CD, Onstad G, and Naeher LP: Exposure of wildland firefighters to carbon monoxide, fine particles, and levoglucosan. The Annals of occupational hygiene 57(8): 979–991 (2013). [PubMed: 23813888]
26. Reinhardt TE, and Ottmar RD: Baseline measurements of smoke exposure among wildland firefighters. Journal of Occupational and Environmental Hygiene 1(9): 593–606 (2004). [PubMed: 15559331]

27. Betchley C, Koenig JQ, van Belle G, Checkoway H, and Reinhardt T: Pulmonary function and respiratory symptoms in forest firefighters. American Journal of Industrial Medicine 31(5): 503–509 (1997). [PubMed: 9099351]
28. Liu D, Tager IB, Balmes JR, and Harrison RJ: The effect of smoke inhalation on lung function and airway responsiveness in wildland fire fighters. The American review of respiratory disease 146(6): 1469–1473 (1992). [PubMed: 1456562]
29. Rothman N, Ford DP, Baser ME, Hansen JA, O'Toole T, Tockman MS et al.: Pulmonary function and respiratory symptoms in wildland firefighters. Journal of occupational medicine. : official publication of the Industrial Medical Association 33(11): 1163–1167 (1991). [PubMed: 1765858]
30. Halbert RJ, Natoli JL, Gano A, Badamgarav E, Buist AS, and Mannino DM: Global burden of COPD: systematic review and meta-analysis. European Respiratory Journal 28(3): 523–532 (2006). [PubMed: 16611654]
31. Buffels J, Degryse J, Heyrman J, and Decramer M: Office spirometry significantly improves early detection of copd in general practice*: The didasco study. Chest 125(4): 1394–1399 (2004). [PubMed: 15078751]
32. Nowak RM, Gordon KR, Wroblewski DA, Tomlanovich MC, and Kvale PA: Spirometric evaluation of acute bronchial asthma. JACEP 8(1): 9–12 (1979). [PubMed: 533976]
33. Celli BR: The importance of spirometry in COPD and asthma: effect on approach to management. Chest 117(2 Suppl): 15S–19S (2000). [PubMed: 10673468]
34. Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, et al.: Standardisation of Spirometry. European Respiratory Journal. 26(2): 319–338 (2005) [PubMed: 16055882]
35. Dills RL, Paulsen M, Ahmad J, Kalman DA, Elias FN, and Simpson CD: Evaluation of urinary methoxyphenols as biomarkers of woodsmoke exposure. Environmental Science & Technology 40(7): 2163–2170 (2006). [PubMed: 16646448]
36. Riddervold IS, Bønløkke JH, Molhave L, Massling A, Jensen B, Gronborg TK et al.: Wood smoke in a controlled exposure experiment with human volunteers. Inhalation Toxicology 23(5): 277–288 (2011). [PubMed: 21506878]
37. Stockfelt L, Sallsten G, Olin AC, Almerud P, Samuelsson L, Johannesson S et al.: Effects on airways of short-term exposure to two kinds of wood smoke in a chamber study of healthy humans. Inhalation Toxicology 24(1): 47–59 (2012). [PubMed: 22220980]
38. Forchhammer L, Moller P, Riddervold IS, Bønløkke J, Massling A, Sigsgaard T et al.: Controlled human wood smoke exposure: oxidative stress, inflammation and microvascular function. Particle and fibre toxicology 9(2012).
39. Sehlstedt M, Dove R, Boman C, Pagels J, Swietlicki E, Londahl J et al.: Antioxidant airway responses following experimental exposure to wood smoke in man. Particle and fibre toxicology 7(2010).
40. Danielsen PH, Brauner EV, Barregard L, Sallsten G, Wallin M, Olinski R et al.: Oxidatively damaged DNA and its repair after experimental exposure to wood smoke in healthy humans. Mutation Research-Fundamental and Molecular Mechanisms of Mutagenesis 642(1–2): 37–42 (2008). [PubMed: 18495177]
41. Barregard L, Saellsten G, Andersson L, Almstrand AC, Gustafson P, Andersson M et al.: Experimental exposure to wood smoke: effects on airway inflammation and oxidative stress. Occupational and Environmental Medicine 65(5): 319–324 (2008). [PubMed: 17704195]
42. Sällsten G, Gustafson P, Johansson L, Johannesson S, Molnar P, Strandberg B et al.: Experimental wood smoke exposure in humans. Inhalation Toxicology 18(11): 855–864 (2006). [PubMed: 16864403]
43. Barregard L, Sallsten G, Gustafson P, Andersson L, Johansson L, Basu S et al.: Experimental exposure to wood-smoke particles in healthy humans: Effects on markers of inflammation, coagulation, and lipid peroxidation. Inhalation Toxicology 18(11): 845–853 (2006). [PubMed: 16864402]
44. Ghio AJ, Soukup JM, Case M, Dailey LA, Richards J, Berntsen J et al.: Exposure to wood smoke particles produces inflammation in healthy volunteers. Occupational and Environmental Medicine 69(3): 170–175 (2012). [PubMed: 21719562]

45. Slaughter JC, Koenig JQ, and Reinhardt TE: Association between lung function and exposure to smoke among firefighters at prescribed burns. Journal of Occupational and Environmental Hygiene 1(1): 45–49 (2004). [PubMed: 15202156]

46. Johnston FH, Kavanagh AM, Bowman DM, and Scott RK: Exposure to bushfire smoke and asthma: an ecological study. The Medical journal of Australia 176(11): 535–538 (2002). [PubMed: 12064985]

47. Filho MAP, Pereira LAA, Arbex FF, Arbex M, Conceicao GM, Santos UP et al.: Effect of air pollution on diabetes and cardiovascular diseases in Sao Paulo, Brazil. Brazilian Journal of Medical and Biological Research 41(6): 526–532 (2008). [PubMed: 18560673]

48. Park SK, Auchincloss AH, O'Neill MS, Prineas R, Correa JC, Keeler J et al.: Particulate air pollution, metabolic syndrome, and heart rate variability: the multi-ethnic study of atherosclerosis (MESA). Environmental Health Perspectives 118(10): 1406–1411 (2010). [PubMed: 20529761]

49. Mott JA, Mannino DM, Alverson CJ, Kiyu A, Hashim J, Lee T et al.: Cardiorespiratory hospitalizations associated with smoke exposure during the 1997, Southeast Asian forest fires. International Journal of Hygiene and Environmental Health 208(1–2): 75–85 (2005). [PubMed: 15881981]

50. Tepper A, Comstock GW, and Levine M: A longitudinal study of pulmonary function in fire fighters. American Journal of Industrial Medicine 20(3): 307–316 (1991). [PubMed: 1928108]

51. Duclos P, Sanderson LM, and Lipsett M: The 1987 forest fire disaster in California: assessment of emergency room visits. Archives of Environmental Health: An International Journal 45(1): 53–58 (1990).

52. Emmanuel SC: Impact to lung health of haze from forest fires: the Singapore experience. Respirology 5(2): 175–182 (2000). [PubMed: 10894108]

53. Kunzli N, Jerrett M, Mack WJ, Beckerman B, LaBree L, Gilliland F et al.: Ambient air pollution and atherosclerosis in Los Angeles. Environmental Health Perspectives 113(2): 201–206 (2005). [PubMed: 15687058]

54. Chen L, Verrall K, and Tong S: Air particulate pollution due to bushfires and respiratory hospital admissions in Brisbane, Australia. International Journal of Environmental Health Research 16(3): 181–191 (2006). [PubMed: 16611563]

55. Stockfelt L, Sallsten G, Almerud P, Basu S, and Barregard L: Short-term chamber exposure to low doses of two kinds of wood smoke does not induce systemic inflammation, coagulation or oxidative stress in healthy humans. Inhalation Toxicology 25(8): 417–425 (2013). [PubMed: 23808634]

56. Riddervold IS, Bønløkke JH, Olin AC, Gronborg TK, Schlunssen V, Skogstrand K et al.: Effects of wood smoke particles from wood-burning stoves on the respiratory health of atopic humans. Particle and fibre toxicology 9: 12 (2012). [PubMed: 22546175]

57. National Wildland Firefighter, N.W.F.: National Wildland Firefighter (NWFF) Workforce Assessment - Final Report. Washington, D.C. : United States Forest Service, 2010.

58. Austin C: "Wildland Firefighter Health Risks and Respiratory Protection" Montréal: IRSST, 2008

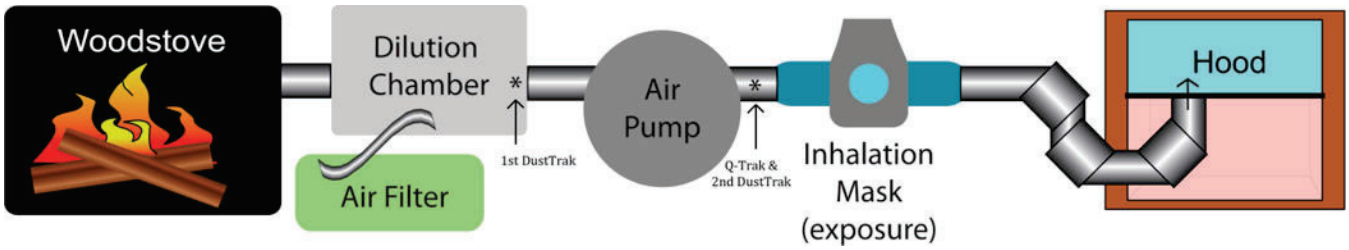


FIGURE 1.
A simplified schematic showing the path of wood smoke through the inhalation system

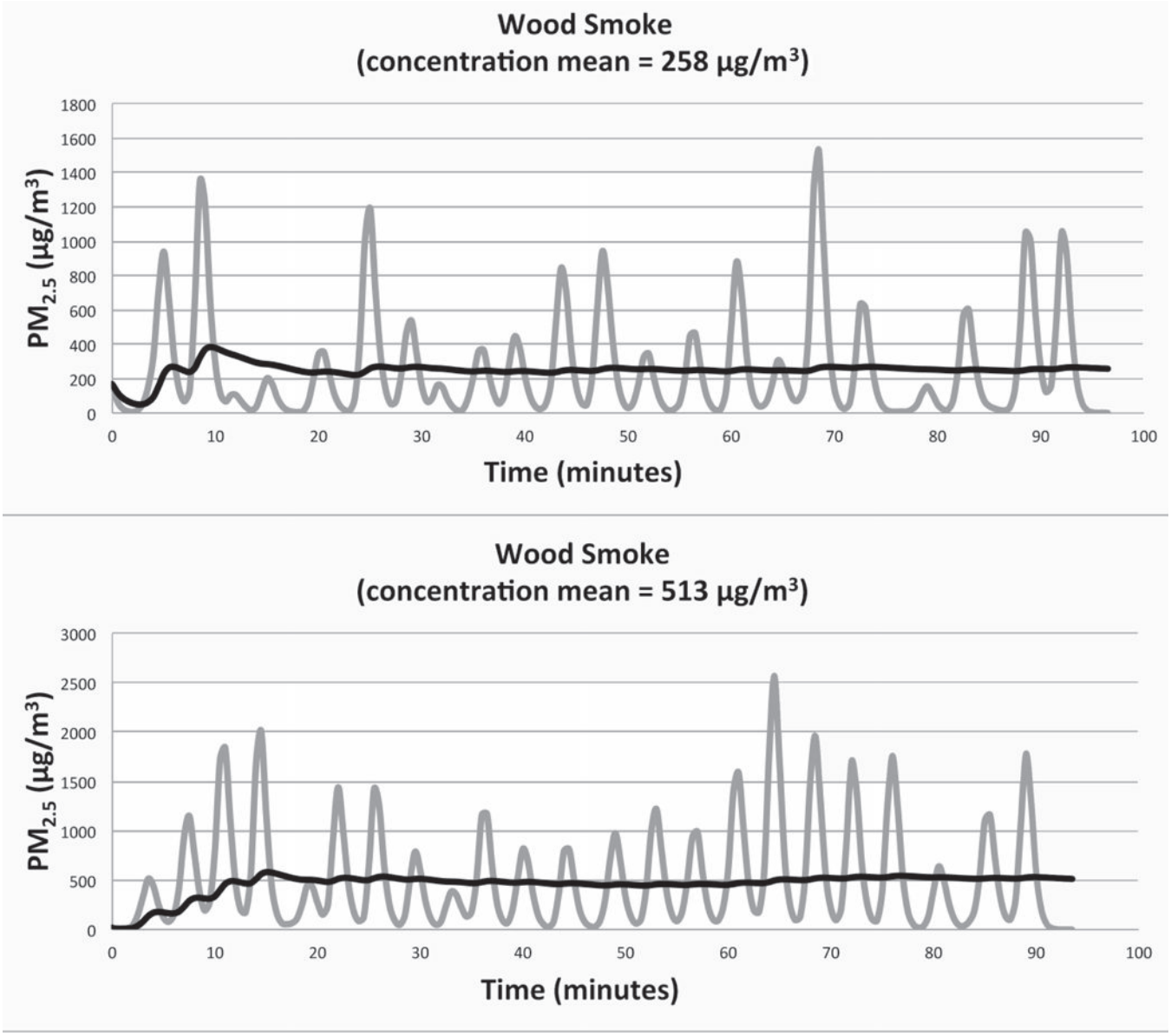


FIGURE 2.

Continuous $PM_{2.5}$ concentrations and averages plotted for a 250 µg/m^3 (top) and 500 µg/m^3 (bottom) 90-minute exposure trial, respectively.

TABLE I.

Measured Spirometry Outcomes as Averages and as Change from Pre-Exposure Levels.

	Pre-exposure (n=9)						Post-exposure (n=9)						1-hr Post-exposure (n=10)					
	Filtered-air		250 μg/m^3		500 μg/m^3		Filtered-air		250 μg/m^3		500 μg/m^3		Filtered-air		250 μg/m^3		500 μg/m^3	
Measurement category	Mean	(sd)	Mean	(sd)	Mean	(sd)	Mean	(sd)	Mean	(sd)	Mean	(sd)	Mean	(sd)	Mean	(sd)	Mean	(sd)
FVC																		
Unadjusted values (liters)	**5.41**	(0.53)	**5.58**	(0.82)	**5.61**	(0.93)	**5.37**	(0.61)	**5.62**	(0.79)	**5.69**	(0.96)	**5.37**	(0.46)	**5.51**	(0.77)	**5.57**	(0.94)
change from pre-exposure levels[A]							**−0.04**	(0.30)	**0.04**	(0.21)	**0.08**	(0.28)	**0.00**	(0.33)	**−0.07**	(0.27)	**−0.04**	(0.21)
FEV$_1$																		
Unadjusted values (liters)	**4.37**	(0.34)	**4.55**	(0.64)	**4.44**	(0.67)	**4.41**	(0.46)	**4.35**	(0.90)	**4.64**	(0.64)	**4.42**	(0.42)	**4.27**	(0.89)	**4.39**	(0.62)
change from pre-exposure levels[A]							**0.04**	(0.31)	**−0.19**	(1.00)	**0.20**	(0.36)	**0.05**	(0.48)	**−0.27**	(1.03)	**−0.05**	(0.33)
FVC:FEV$_1$																		
Unadjusted values (ratio)	**81.11**	(6.34)	**81.26**	(6.44)	**79.72**	(8.10)	**82.37**	(6.97)	**77.86**	(14.0)	**81.85**	(7.29)	**82.89**	(7.90)	**78.24**	(15.3)	**79.43**	(8.54)
change from pre-exposure levels[A]							**1.26**	(2.39)	**−3.40**	(14.2)	**2.13**	(3.33)	**1.78**	(5.66)	**−3.02**	(15.5)	**−0.29**	(4.32)

[A]Estimated changes from pre-exposure to immediate post-exposure and change from pre-exposure to 1-hour post-exposure.

TABLE II.

Summary of Controlled Human Exposure Studies.

References	N	Exercise duration	Exposure concentration(s)	Wood smoke exposure
Hunter et al. 2014	16 adult males	bike every 15 minutes	1-hour	filtered-air and ~1 mg/m^3
Bønløkke et al. 2014	24 adult males and females	at rest	3.5 hours	filtered-air (13), 222, and 385 μg/m^3
Unosson et al. 2013	14 adult males and females	bike every 15 minutes	3 hours	filtered-air and 214 μg/m^3
Stockfelt et al. 2013	16 adult males and females	at rest	3 hours	filtered-air, 146, and 295 μg/m^3
Ghio et al. 2012	10 healthy individuals[A]	bike every 15 minutes	2 hours	filtered-air and 485 μg/m^3
Stockfelt et al. 2012	16 adult males and females	at rest	3 hours	filtered-air, 146, and 295 μg/m^3
Forchhammer et al. 2012	20 adult males and females	at rest	3 hours	filtered-air (14), 220, and 354 μg/m^3
Riddervold et al. 2012	20 adult males and females	at rest	3.5 hours	filtered-air, 200, and 400 μg/m^3
Riddervold et al. 2011	20 adult males and females	at rest	3.5 hours	filtered-air, 200, and 400 μg/m^3
Sehlstedt et al. 2010	19 adult males and females	bike every 15 minutes	3 hours	filtered-air and 224 μg/m^3
Danielsen et al. 2008	13 adult males and females	25 minute bike ride, 2x	4 hours	filtered-air and 243–279μg/m^3
Barregard et al. 2008	13 adult males and females	25 minute bike ride, 2x	4 hours	filtered-air and 243–279 μg/m^3
Sällsten et al. 2006	13 adult males and females	25 minute bike ride, 2x	4 hours	filtered-air and 250 μg/m^3
Barregard et al. 2006	13 adult males and females	25 minute bike ride, 2x	4 hours	filtered-air and 243–279 μg/m^3

[A] Gender details of participating individuals not outlined in manuscript

HHS Public Access

Author manuscript

Am J Ind Med. Author manuscript; available in PMC 2015 November 02.

Published in final edited form as:

Am J Ind Med. 2014 July ; 57(7): 748–756. doi:10.1002/ajim.22331.

Arterial Stiffness, Oxidative Stress, and Smoke Exposure in Wildland Firefighters

Denise M. Gaughan, ScD[1,2,*], **Paul D. Siegel, PhD**[3], **Michael D. Hughes, PhD**[4], **Chiung-Yu Chang, ScD**[2], **Brandon F. Law, MS**[3], **Corey R. Campbell, MS**[5], **Jennifer C. Richards, MS**[6], **Stefanos F. Kales, MD**[2], **Marcia Chertok, BS**[2], **Lester Kobzik, PhD**[7], **Phuongson Nguyen, PhD**[7], **Carl R. O'Donnell, PhD**[8], **Max Kiefer, MS**[5], **Gregory R. Wagner, MD**[2], and **David C. Christiani, MD**[2]

[1]Department of Preventive Medicine and the Institute for Translational Epidemiology, Icahn School of Medicine at Mount Sinai, New York, New York

[2]Department of Environmental Health (Environmental and Occupational Medicine and Epidemiology Program), Harvard School of Public Health, Boston, Massachusetts

[3]Health Effects Laboratory Division, National Institute for Occupational Safety and Health, Morgantown, West Virginia

[4]Departmentof Biostatistics, Harvard School of Public Health, Boston, Massachusetts

[5]Western States Office, National Institute for Occupational Safety and Health, Denver, Colorado

[6]Department of Health and Exercise Science, Colorado State University, Fort Collins, Colorado

[7]Department of Environmental Health (Molecular and Integrative Physiological Sciences), Harvard School of Public Health, Boston, Massachusetts

[8]Beth Israel Deaconess Medical Center, Division of Pulmonary and Critical Care Medicine, Boston, Massachusetts

Abstract

Objectives—To assess the association between exposure, oxidative stress, symptoms, and cardiorespiratory function in wildland firefighters.

Methods—We studied two Interagency Hotshot Crews with questionnaires, pulse wave analysis for arterial stiffness, spirometry, urinary 8-iso-prostaglandin F2α (8-isoprostane) and 8-hydroxy-2′-deoxyguanosine (8-OHdG), and the smoke exposure marker (urinary levoglucosan). Arterial stiffness was assessed by examining levels of the aortic augmentation index, expressed as a percentage. An oxidative stress score comprising the average of z-scores created for 8-OHdG and 8-isoprostane was calculated.

Results—Mean augmentation index % was higher for participants with higher oxidative stress scores after adjusting for smoking status. Specifically for every one unit increase in oxidative

[*]Correspondence to: Denise M. Gaughan, ScD, Icahn School of Medicine at Mount Sinai, 17 East 102nd Street, D3-132, New York, NY10029-6574. denise.gaughan@mssm.edu.

Disclosure Statement: The authors report no conflicts of interests.

stress score the augmentation index % increased 10.5% (95% CI: 2.5, 18.5%). Higher mean lower respiratory symptom score was associated with lower percent predicted forced expiratory volume in one second/forced vital capacity.

Conclusions—Biomarkers of oxidative stress may serve as indicators of arterial stiffness in wildland firefighters.

Keywords

vascular stiffness; 8-iso-prostaglandin F2α; 8-hydroxy-2′-deoxyguanosine; spirometry; levoglucosan

BACKGROUND

Firefighters, both structural and wildland, are known to have cardiovascular and respiratory problems [Musk et al., 1979; Sardinas et al., 1986; Chia et al., 1990; Rothman et al., 1991; Guidotti and Clough, 1992; Liu et al., 1992; Materna et al., 1992; Scannell and Balmes, 1995; Betchley et al., 1997; Austin et al., 2001; Burgess et al., 2001; Kales et al., 2003; Slaughter et al., 2004; CDC, 2006; Gaughan et al., 2008; Yoo and Franke, 2009]. Cardiovascular disease (CVD) events are the leading cause of on-duty and lifetime mortality among structural (career and volunteer) firefighters [Sardinas et al., 1986; Kales et al., 2003; Yoo and Franke, 2009]. The deleterious effects of smoke exposure to structural firefighters have been extensively researched [Musk et al., 1979; Sardinas et al., 1986; Chia et al., 1990; Guidotti and Clough, 1992; Liu et al., 1992; Scannell and Balmes, 1995; Burgess et al., 2001; Kales et al., 2003; CDC, 2006; Yoo and Franke, 2009]. Exposure to particulates and other contaminants, heavy physical exertion and cardiovascular strain have been found to be among the chief health hazards associated with structural firefighting [Gledhill and Jamnik, 1992; Takeyama et al., 2005; Delfino et al., 2009]. Those findings, however, may not be generalizable to wildland firefighters for a number of reasons, including the difference in smoke composition, comparative younger age of wildland firefighters, often shorter career tenure, and the longer duration of respiratory exposures for wildland firefighters. Additionally, structural firefighters routinely wear respiratory protection when responding to fires while wildland firefighters do not.

Fine particulate exposure has been associated with acute changes in cardiovascular and pulmonary function [Vallyathan et al., 1995; Mott et al., 2005; Dominici et al., 2006; Cavallari et al., 2008; Fang et al., 2008, 2009]. Free radical mechanisms have been implicated as a contributing factor in general toxicity, inflammation, asthma, fibrogenesis, bronchopulmonary carcinogenesis, and atherosclerotic plaque formation [Jarjour and Calhoun, 1994; Leonard et al., 2007; LeBlanc et al., 2010].

Wood fires produce smoke with abundant particles in the inhalable range (<100 μm) and contain both carbon radicals and precursors. The latter are able to react with H_2O_2 after exposure to cells and generate the highly reactive hydroxyl radical (OH) from a Fenton-like reaction [Leonard et al., 2007]. The authors additionally observed that carbon radicals were stronger (per unit mass) in larger (coarse) sized particles while OH and other ROS were stronger (per unit mass) in the smaller (ultrafine) sized particles. Finally, the authors noted

that fine and ultrafine woodsmoke particles also significantly increased H_2O_2, DNA strand breaks and lipid peroxidation in exposed RAW 264.7 cells.

Pyrolysis of organic components may increase the potency and or toxicity of particulate by producing ROS. Measurement of levoglucosan, a sugar anhydride by-product of incomplete combustion of cellulose, may be used to indicate relative exposure to products of pyrolysis from burning biomass [Simoneit et al., 1999]. A recent study examined personable exposure to airborne respirable levoglucosan using a Dorr-Oliver cyclone and air sampling pump, and cross-shift changes in lung function in a population of 17 wildland firefighters for 4 days at a large wildland fire [Gaughan et al., 2014]. The authors reported that levoglucosan was found mainly in the respirable fraction, defined as under 2.5 μm, with higher concentrations during fireline construction than in mop-up operations. Furthermore, larger cross-shift declines in forced expiratory volume in one second (FEV_1) were associated with exposure to higher concentrations of respirable levoglucosan (P-value <0.05).

Urinary levoglucosan has also been investigated as a biomarker for smoke exposure. Bergauff et al. examined cross-shift changes in urinary levoglucosan in nine firefighters exposed to wood smoke in a controlled setting. They observed elevated urinary levoglucosan following smoke exposure in some but not all firefighters [Bergauff et al., 2010]. Moreover, the authors noted the contribution of dietary intake to urinary levoglucosan levels.

Urinary 8-iso-prostaglandin F2α (8-isoprostane), a biomarker of oxidative stress generated by lipid peroxidation, may serve as a biomarker for atherosclerosis [Cipollone et al., 2000]. Oxidative deoxyribonucleic acid (DNA) damage and repair has also been linked to atherosclerosis. Martinet et al. examined five human carotid endarterectomy specimens and five mammary artery specimens for 8-hydroxy-2′-deoxyguanosine (8-OHdG), an oxidized nucleoside of DNA. The authors observed an increased amount of 8-OHdG in plaques compared to the underlying media or non-atherosclerotic mammary arteries [Martinet et al., 2002]. Urinary 8-OHdG is excreted upon DNA repair and may serve as a non-invasive biomarker of global oxidative DNA damage. For instance, acute changes in urinary 8-OHdG concentrations have been reported in occupational studies examining welders [Kim et al., 2004; Nuernberg et al., 2008].

Elevated arterial stiffness is a characteristic of large artery pathology, a major contributor to CVD, and may serve as an indicator of pre-clinical atherosclerosis and/or hypertension [Blankenhorn and Kramsch, 1989; Duprez and Cohn, 2007]. Assessment of arterial stiffness is done through ultrasound or measurement of pulse wave velocity. The aortic augmentation index, is an indirect measure of systemic arterial stiffness based on pulse wave velocity and is calculated as a percentage. A recent meta-analysis demonstrated that a 10% increase in augmentation index % was associated with a 31.8% increased risk of cardiovascular events and a 34.8% increased risk of total mortality [Vlachopoulos et al., 2010]. The augmentation index % has been successfully implemented in occupational research settings [Nürnberger et al., 2002; Fang et al., 2008].

There are four types of wildland firefighter suppression crews: engine crew, hand crew, helicopter crew, and smoke jumpers. Type 1 Interagency Hotshot Crews are an elite type of hand crew, comprising up to 20 firefighters who construct fire lines using hand tools during the most dangerous phases of fire suppression.

The question addressed by the present study was whether wildland firefighting exposures are associated with oxidative stress concentrations and with pulmonary and vascular function. To answer these questions, we assessed spirometry, vascular function, symptoms, and systemic biomarkers of exposure, inflammation and oxidative stress in members of two Type 1 wildland firefighter Interagency Hotshot Crews.

METHODS

The National Interagency Fire Center (NIFC) Risk Management Committee arranged for two crews to participate in this study: the Alpine Interagency Hotshot Crew, Rocky Mountain National Park, Estes Park, CO and the Pike Interagency Hotshot Crews, Pike and San Isabel National Forests, Monument, CO. The Interagency Hotshot Crews participated in our study by completing questionnaires, pulse wave analysis and spirometry in May 2011. Serum cholesterol, and biomarkers of systemic inflammation (high sensitivity c-reactive protein (hsCRP) and fibrinogen), oxidative stress (8-isoprostane), oxidative DNA damage (8-OHdG), and smoke exposure (urinary levoglucosan) were measured. The study protocol was approved by the Harvard School of Public Health (HSPH) Institutional Review Board and the National Institute for Occupational Safety and Health (NIOSH) Human Subjects Review Board. Informed consent was obtained from each research participant.

Medical Testing Methods

Pulse wave analysis—Vascular function was measured using a pulse wave analysis system according to the manufacturer's instructions [SphygmoCor CP, Atcor Medical Pty Ltd., Sydney, Australia]. Briefly, participants were seated with the dominant arm extended onto a flat surface so that the antecubital fossa was at heart level. Following 5 min of rest, a high-fidelity micro-nanometer was used to flatten the radial artery with gentle pressure. Ten seconds of sequential pulse pressure waveforms were recorded at each reading. The waveforms were then transformed into a corresponding central aortic waveform via a validated transfer function where the systolic part of the central aortic waveform is characterized by a first peak caused by left ventricular ejection and a second peak caused by wave reflection. The difference between the two peaks reflects the degree to which the central aortic pressure is augmented by wave reflection. We calculated each participant's aortic augmentation index %, defined as the ratio of augmented pressure to pulse pressure (i.e., augmentation index % =augmented pressure/pulse pressure ×100) [Nürnberger et al., 2002] and heart rate corrected to 75 beats per minute. Larger augmentation index % values denote increased wave reflection. A minimum of three within-session recordings were obtained from each participant.

Spirometry—Pulmonary function was determined on each participant using an ultrasonic flow spirometer [EasyOne^{TM} Diagnostic Spirometry System 2001, ndd Medical Technologies, Zurich, Switzerland]. Technicians completed a NIOSH-approved spirometry

course followed American Thoracic Society (ATS) guidelines [Miller et al., 2005]. Test results were interpreted using reference values generated from the Third National Health and Nutrition Examination Survey (NHANES III) [Hankinson et al., 1999]. Airways obstruction was defined as a forced expiratory volume in the first second (FEV_1) to forced vital capacity (FVC) ratio below the lower limit of normal according to published reference equations [Pellegrino et al., 2005]. We examined percent predicted FEV_1 (FEV_1%-predicted), percent predicted FVC (FVC %-predicted), and percent predicted FEV_1/FVC (FEV_1/FVC %-predicted). We followed ATS procedure by inquiring about current medications but did not ask participants to abstain from using their medications prior to participating in this study for safety purposes. Reports were reviewed for quality by a respiratory physiologist (C.R.O) experienced in clinical pulmonary function laboratory administration.

Blood—Whole non-fasting serum samples (30 ml/sample) were collected by venous phlebotomy in EDTA tubes, and buffy coat was extracted and stored in cell lyses solution at −20°C for analyses of typical cardiovascular-related biomarkers, specifically, hsCRP and fibrinogen. We additionally examined total cholesterol, high-density lipoprotein cholesterol (HDL-cholesterol), low-density lipoprotein cholesterol (LDL-cholesterol), and triglycerides. These analyses were conducted by Quest Diagnostics Inc., Denver, CO.

Urine—Urine samples were analyzed for 8-OHDG and 8-isoprostane using competitive enzyme-linked immunoassays (EKS-350, Assay Designs, Inc., Ann Arbor, MI; 8-Isoprostane EIA Kit, Cayman Chemical Company, Ann Arbor, MI) as well as for creatinine (picric acid colorimetric assay; Oxford Biomedical Research, Oxford, MI). We also examined urine for levoglucosan concentration. Two hundred microliter of urine or levoglucosan standards (Blank, 6.25–100 μg/ml in saline) were added to 1.5 ml low retention microcentrifuge tubes. To this 30U of urease (Sigma, St. Louis, MO) was added and incubated for 1.0 hr at 37°C. After incubation, 600 μl of cold (4°C) ethanol was added to precipitate the protein. Approximately 400 mg of sodium sulfate was added volumetrically and allowed to sit for 2.0 min. Samples and standards were then centrifuged for 2.0 min at 14,000*g*. After centrifugation, 600 μl of each were transferred to clean low retention microcentrifuge tubes and evaporated to dryness using a vacuum centrifugation and gentle heat. Two hundred microliter of n-methyl-n-(trimethylsilyl) trifluoroacet (MSTFA) (Sigma Chemical Co.,) was then added to each tube, vortexed and incubated at 72°C for 1.0 hr. After derivatization samples and standards were analyzed on an Agilent 6890 gas chromatograph coupled to an Agilent 5975C mass spectrometer using a 30-m HP5-MS column (Agilent Technologies, Santa Clara, CA). Samples were injected (1 μl) in splitless mode into a 250°C inlet with a 6.0-min solvent delay. Analytes were eluted from the column using 1.0 ml/min helium and an oven temperature program as follows: 70°C for 3.0 min and then ramped at 25.0°C/min to a final temperature of 275°C. The MS source temperature was maintained at 230°C, and the quadrupole temperature was maintained at 150°C. Ions were scanned between 50 and 400 m/z. Levoglucosan from each sample was identified by the MS spectra and retention time (against the known standards) and quantified using the 204 m/z ion. The standard plot from which the samples were extrapolated used a polynomial curve fit of 204 m/z area under the curve ion count versus standard concentration. Specimens were

analyzed by NIOSH's Health Effects Laboratory Division, Allergy and Clinical Immunology Branch, Morgantown, WV.

Questionnaires—The questionnaire was based on two standardized questionnaires, the American Thoracic Society-Division of Lung Disease-78 (ATS-DLD-78) supplemented with questions from NHANES III [Ferris, 1978; Wasserfallen et al., 1997]. This modified questionnaire was designed to acquire information concerning chronic cardiovascular and respiratory conditions; lifetime diagnoses; tobacco history; symptom history; dietary intake; sleep patterns; medication use; and occupational history. A validated symptom scale, with Likert scoring where 0 =none, 1 =trivial, 2 =mild, 3 =moderate, and 4 =severe for upper and lower airways symptoms, was used to derive overall symptom scores by summing the responses to questions about 19 symptoms. Symptoms ascertained included cough, wheeze, sputum production, shortness of breath or chest tightness, and shortness of breath while walking, as well as various eye, nose, and throat symptoms. Subjects additionally completed a semi-quantitative food frequency questionnaire, adult version, 2007. Subjects were asked to report the average daily consumption of various foods in the preceding year. Responses ranged from "never" to "six or more servings per day." The food frequency questionnaire also assessed the frequency of multivitamin and mineral supplement usage. Frequency factors of related foods items were then summed to calculate the daily servings for each food group. The nutrient value of the food item was multiplied by the frequency of consumption in order to obtain macro and micro nutrient intake. Scoring was done by the Nutrition Department, Harvard School of Public Health (https://regepi.bwh.harvard.edu/health/nutrition.html) [Willett et al., 1985].

Statistical Methods—Descriptive statistics were calculated for demographic and clinical variables. Mean values among subgroups were compared using Student's *t*-test techniques. Ordinary least squares regression techniques were used to examine associations between augmentation index %, FEV_1%-predicted, FVC %-predicted, FEV_1/FVC %-predicted, 8-OHdG concentration, and 8-isoprostane concentration using the following predictor variables: levoglucosan concentration, serum lipid levels, hsCRP concentration, fibrinogen concentration, LDL-cholesterol, HDL-cholesterol, Interagency Hotshot Crew, volunteer firefighter (yes or no), cumulative time spent fighting fires (seasons), medical diagnoses, allergies, upper and lower respiratory symptom scores, and history of tobacco use. Levoglucosan, 8-isoprostane, 8-OHdG, and hsCRP values were not normally distributed and thus the data were $\log_{10}$-transformed to achieve an approximate normal distribution. We additionally calculated an "oxidative stress score" comprising z-score's of 8-isoprostane and 8-OHdG, where z-value's for each variable were calculated and then averaged to yield a score for each participant. The motivation for calculating the z-score was prompted by the possibility that while 8-isoprostane is considered a marker of lipid peroxidation and 8-OHdG has been linked to DNA damage and repair, they may both be measuring the overall level of oxidative stress in the urine. Thus, we standardized the two biomarkers of oxidative stress on one scale. These values were also examined in multivariable analysis. The z-value was calculated by subtracting the mean value from each subject's value and then dividing by the standard deviation of the values. Multivariable models were chosen based on an initial evaluation using stepwise selection techniques, followed by examining univariate

associations and sequentially adjusting for other predictors. Associations were considered statistically significant if *P*-values were <0.05 and borderline significant at ≥0.05 and <0.10. All analyses were conducted using SAS statistical software (version 9.3).

RESULTS

Thirty-eight of the 39 current members of both crews participated in the surveys in May 2011 during their training sessions at their respective home base parks (97%). One member was training off-site the day of our survey and as a result, could not participate. Members of the Pike Interagency Hotshot Crew had been exposed to smoke for 2 days at the Sand Gulch fire 4 days before the testing. The Sand Gulch fire in Wetmore, CO, was a 495-acre wildland fire at 7,000 feet elevation in high difficulty terrain. Fire behavior activity was described by crew members as high/extreme on the first day and low/smoldering on the second. It was reported that the average shift duration each day was 16 hr and it was estimated the crew members were on the fire line performing firefighting activities for 12.5 hr each day.

Participant demographic and clinical characteristics, overall and stratified by crew are shown in Tables I and II. The two crews were very similar. They were all male with a median age of 28 years and had spent a median of 3 years (seasons) working as an Interagency Hotshot Crew member. Approximately 5% of the participants were current smokers and nearly half (42%) reported current chewing tobacco use. Approximately one-third (29%) of the participants were classified as “permanent” employees, the remainder were “seasonal” hires. Additionally, six of the participants (16%) reported working as a volunteer structural firefighter off-season. Values for clinical characteristics were also comparable when examined by crew and within the normal range.

Seven participants reported a history of physician-diagnosed asthma (18%). The median age at asthma diagnosis was 7 years and the median time spent as a firefighter for these seven participants was 2 years (seasons). Among these individuals, the median FEV_1%-predicted was 105%, the median FVC %-predicted was 109%, and the median FEV_1/FVC %-predicted was 96%. Among the four individuals with current asthma, three were currently taking medication for their asthma. One of the seven participants with a history of asthma also reported a smoking history.

Four values for levoglucosan were below the limit of detection and were not included in the analysis. Detectable levels were observed for all values of 8-OHdG, 8-isoprostane, hsCRP, and fibrinogen.

In multivariable analysis, mean augmentation index % was higher for participants with higher oxidative stress scores. Specifically, for every one unit increase in oxidative stress score, mean augmentation index % increased 10.2% (10.2%, 95% CI: 1.35, 19.0%) (Table III). This association remained significant after adjusting for smoking status (*P* =0.01). No other variables were associated with augmentation index %, including our exposure variable, levoglucosan. Table III details these results.

However, higher levoglucosan concentration was positively associated with oxidative stress scores (Table IV). Specifically, for every twofold increase in $\log_{10}$ levoglucosan

concentration, mean 8-OHdG increased by $\log_{10}$ 0.14 μg/ml (95% CI: 0.02, 0.25) (regression estimate 0.41 (95% CI: 0.04, 0.79)) and 8-isoprostane increased by $\log_{10}$ 0.16 ng/ml (95% CI: 0.02, 0.29) (regression estimate 0.52 (95% CI: 0.06, 0.97)). Additionally, 8-OHdG values for participants who had recently been exposed to smoke particulate as measured by Interagency Hotshot Crew were higher than those with no recent exposure (P =0.01). This association became borderline significant after adjusting for levoglucosan (P =0.07) (Table IV).

Finally, higher lower respiratory symptom score (LRSS) was associated with lower FEV_1/FVC %-predicted after adjusting for smoking status and history of asthma. Specifically, for every twofold increase in mean LRSS, the mean FEV_1/FVC %-predicted dropped on average by 1.66% (95% CI: 0.10%, 3.22%) (regression estimate:−0.83 (95% CI: −1.61, −0.05)). No other variables were associated with FEV_1/FVC %-predicted, most notably, neither oxidative stress score nor levoglucosan.

DISCUSSION

Particulate exposure has been associated with increased arterial stiffness in occupational cohorts. Fang et al. examined changes in augmentation index % in 26 welders over 24 hr on a welding day and non-welding day. Following welding fume exposure, the authors observed an increase in afternoon augmentation index % and a decrease in next morning augmentation index % [Fang et al., 2008]. The results suggested that exposure to welding fume particulate is associated with acute adverse vascular responses. In our homogenous group of healthy workers, we observed higher augmentation index % values for participants with higher oxidative stress values which were also associated with larger levoglucosan values. A possible explanation for this finding could be that our population was younger by comparison (28 vs. 41 years) and/or our assessment occurred 4 days post-exposure to smoke.

Similar to other studies, we observed an association between cross-shift differences in oxidative DNA damage and recent occupational exposure to particulate matter. Kim et al. obtained 5 days of cross-shift urinary 8-OHdG measurements from 20 welders exposed to metal fumes. The authors reported that urinary 8-OHdG levels were significantly elevated in post-shift samples compared to those collected pre-shift [Kim et al., 2004]. Nuernberg et al. [2008] observed similar 8-OHdG elevation post-shift in 63 welders. Moreover, the authors found an unexpected inverse relationship between post-shift and 8 hr post-shift values of 8-isoprostane and $PM_{2.5}$ (P <0.05). We additionally observed a difference in 8-isoprostane by levoglucosan concentration. We did not observe a difference in mean values of 8-OHdG or 8-isoprostane between participants who identified themselves as having a history of smoking and those who did not (P =0.51 and 0.64, respectively).

Levoglucosan was positively associated with both biomarkers of oxidative stress, 8-OHdG and 8-isoprostane. Urinary levoglucosan has been shown to increase in mice after exposure to wood smoke [Migliaccio et al., 2009]. However, when examining urinary levoglucosan concentration, it is important to control for dietary confounders such as smoked or fried foods and caramel. Based on responses to the Food Frequency Questionnaire, 32 (84%)

reported consuming two slices of bacon or less once per week; 34 (89%) reported eating one candy bar or less per week. All participants reported eating fried foods at home or away four to six times per week or less, although 22 (58%) reported consuming fried foods less than once per week. Inhaled levoglucosan is probably completely eliminated from the body within 24 hr post-exposure. Thus, it is possible that the elevated levels of levoglucosan we observed were caused by diet, rather than exposure.

There is evidence that particulate matter [PM] produced by woodsmoke fires may be more toxic than PM from ambient air due to the chemical components found in the smoke [Wegesser et al., 2009]. Leonard et al. [2007] examined aerodynamically size-selected aerosol samples at a large wildland fire. The authors found smoke particles in all size fractions [from smaller than 0.056 μm to greater than 10 μm] and a bell-shaped distribution with highest overall mass fraction on filters in the fine range. The authors additionally observed that radical signals were size dependent and resulted in reactive oxygen species (ROS) generation.

Recent animal studies suggest that exposure to ultrafine particles may additionally augment cardiac dysfunction through an ROS mechanism [LeBlanc et al., 2010]. LeBlanc et al. recently demonstrated that local ROS generation can influence vascular reactivity in coronary arterioles. It follows that excessive coronary ROS generation following pulmonary woodsmoke exposure could impair endothelium-dependent arteriolar reactivity.

Seven participants reported a history of asthma; four with current asthma. Similar asthma prevalences were previously observed in this population [Gaughan et al., 2008]. We did not see a difference in %-predicted lung function values in these subjects from the rest nor did we observe an association between oxidative stress score or levoglucosan and a history of asthma. However, we did observe that higher LRSS were associated with lower FEV_1/FVC %-predicted after adjusting for asthma history and smoking status ($P < 0.05$).

We recognize several limitations to our study. Our sample size was small and the resultant lack of statistical power may have hindered our ability to observe associations if they did exist. While we did observe a significant association between higher oxidative stress scores with recent firefighting activities, the generally qualitative and self-reported nature of exposure characterization in our study may have limited our ability to have identified statistically significant cardiopulmonary effects related to firefighting exposures. Additionally, this was a cross-sectional study with essentially no control group. This design limited examination of acute changes. Our study also lacked cross-shift data during periods in which the crews were working but not fighting a fire. Future studies should examine these individuals cross-shift at a wildland fire and when they are not being exposed to smoke aerosol to ascertain whether any observed changes are part of their normal variation. However, a recent study did not find significant changes in cross-shift lung function on burn days compared to non-burn days [Adetona et al., 2011]. Also, urinary levoglucosan may be more representative of diet than as an indicator of wood-smoke exposure. Finally, all spirometric values were obtained pre-bronchodilator. Post-bronchodilator values would have allowed us to examine reversibility and may have been particularly informative for participants reporting a history of asthma.

This is the first study to examine the association between systemic oxidative stress and arterial stiffness in wildland firefighters. Future studies should additionally examine endothelial function which is governed by smooth muscle tone, collage/elastin, calcification, and other factors that affect the entire arterial wall. The two together could provide different but complementary information on vascular health.

Acknowledgments

We thank the Alpine and Pike Interagency Hotshot Crews for their participation as well as the National Interagency Fire Center, Risk Management Committee, specifically, Chad Fisher, U.S. Department of Interior, National Park Service and Larry Sutton, U.S.D.A. Forest Service for arranging for the Interagency Hotshot Crews to participate. The authors additionally thank Dr. Shona Fang, Li Su, and Lauren Cassidy for assistance preparing for the field surveys and Dr. Kristin Cummings for editorial comments.

References

Adetona O, Hall DB, Naeher LP. Lung function changes in wildland firefighters working at prescribed burns. Inhal Toxicol. 2011; 23:835–841. [PubMed: 22035123]

Austin C, Wang D, Ecobichon DJ, Dussault G. Characterization of volatile organic compounds in smoke at municipal structural fires. J Toxicol Environ Health. 2001; 63:437–458.

Bergauff MA, Ward TJ, Noonan CW, Migliaccio CT, Simpson CD, Evanoski AR, Palmer CP. Urinary levoglucosan as a biomarker of wood smoke: Results of human exposure studies. J Expo Sci Environ Epidemiol. 2010; 20:385–392. [PubMed: 19707249]

Betchley C, Koenig JQ, vanBelle G, Checkoway H, Reinhardt T. Pulmonary function and respiratory symptoms in forest firefighters. Am J Ind Med. 1997; 31:503–509. [PubMed: 9099351]

Blankenhorn DH, Kramsch DM. Reversal of atherosis and sclerosis. The two components of atherosclerosis. Circulation. 1989; 79:1–7. [PubMed: 2642753]

Burgess JL, Nanson CJ, Bolstad-Johnson DM, Gerkin R, Hysong TA, Lantz RC, Sherrill DL, Crutchfield CD, Quan SF, Bernard AM, et al. Adverse respiratory effects following overhaul in firefighters. J Occup Environ Med. 2001; 43:467–473. [PubMed: 11382182]

Cavallari JM, Eisen EA, Fang SC, Schwartz J, Hauser R, Herrick RF, Christiani DC. PM 2.5 metal exposures and nocturnal heart rate variability: A panel study of boilermaker construction workers. Environ Health. 2008; 7:36. [PubMed: 18613971]

Centers for Disease Control and Prevention [CDC]. Fatalities among volunteer and career firefighters–United States, 1994–2004. CDC MMWR Morb Mortal Wkly Rep. 2006; 55(16):453–455. [PubMed: 16645570]

Chia KS, Jeyaratnam J, Chan TB, Lim TK. Airway responsiveness of firefighters after smoke exposure. Br J Ind Med. 1990; 47:524–527. [PubMed: 2393631]

Cipollone F, Ciabattoni G, Patrignani P, Pasquale M, Di Gregorio D, Bucciarelli T, Davì G, Cuccurullo F, Patrono C. Oxidant stress and aspirin-insensitive thromboxane biosynthesis in severe unstable angina. Circulation. 2000; 102:1007–1013. [PubMed: 10961965]

Delfino RJ, Brummel S, Wu J, Stern H, Ostro B, Lipsett M, Winer A, Street DH, Zhang L, Tjoa T, et al. The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003. Occup Environ Med. 2009; 66:189–197. [PubMed: 19017694]

Dominici F, Peng RD, Bell ML, Pham L, McDermott A, Zeger SL, Samet JM. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA. 2006; 295:1127–1134. [PubMed: 16522832]

Duprez DA, Cohn JN. Arterial stiffness as a risk factor for coronary atherosclerosis. Curr Atheroscler Rep. 2007; 9:139–144. [PubMed: 17877923]

Fang SC, Eisen EA, Cavallari JM, Mittleman MA, Christiani DC. Acute changes in vascular function among welders exposed to metal-rich particulate matter. Epidemiology. 2008; 19:217–225. [PubMed: 18300696]

Fang SC, Cavallari JM, Eisen EA, Chen JC, Mittleman MA, Christiani DC. Vascular function, inflammation, and variations in cardiac autonomic responses to particulate matter among welders. Am J Epidemiol. 2009; 169:848–856. [PubMed: 19153215]

Ferris BG. Epidemiology standardization project. Am Rev Respir Dis. 1978; 108:1–113. [PubMed: 742764]

Gaughan DM, Cox-Ganser JM, Enright PL, Castellan RM, Wagner GR, Hobbs GR, Bledsoe TA, Siegel PD, Kreiss K, Weissman DN. Acute upper and lower respiratory effects in wildland firefighters. J Occup Environ Med. 2008; 50:1019–1028. [PubMed: 18784550]

Gaughan DM, Piacitelli CA, Chen BT, Law BF, Virji MA, Edwards NT, Enright PL, Schwegler-Berry DE, Leonard SS, Wagner GR, et al. Exposures and cross-shift lung function declines in wildland firefighters. J Occup Environ Hyg. 2014 Feb 25. Epub ahead of print. 10.1080/15459624.2014.895372

Gledhill N, Jamnik VK. Characterization of the physical demands of firefighting. Can J Sport Sci. 1992; 17:207–213. [PubMed: 1325260]

Guidotti TL, Clough VM. Occupational health concerns of firefighting. Annu Rev Public Health. 1992; 13:151–171. [PubMed: 1599583]

Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. Am J Respir Crit Care Med. 1999; 159:179–187. [PubMed: 9872837]

Jarjour NN, Calhoun WJ. Enhanced production of oxygen radicals in asthma. J Lab Clin Med. 1994; 123:131–136. [PubMed: 8288953]

Kales SN, Soteriades ES, Christoudias SG, Christiani DC. Firefighters and on-duty deaths from coronary heart disease: A case control study. Environ Health. 2003; 2:14. [PubMed: 14613487]

Kim JY, Mukherjee S, Ngo LC, Christiani DC. Urinary 8-hydroxy-2′-deoxyguanosine as a biomarker of oxidative DNA damage in workers exposed to fine particulates. Environ Health Perspect. 2004; 112:666–671. [PubMed: 15121508]

LeBlanc AJ, Moseley AM, Chen BT, Frazer D, Castranova V, Nurkiewicz TR. Nanoparticle inhalation impairs coronary microvascular reactivity via a local reactive oxygen species-dependent mechanism. Cardiovasc Toxicol. 2010; 10:27–36. [PubMed: 20033351]

Leonard SS, Castranova V, Chen BT, Schwegler-Berry D, Hoover M, Piacitelli C, Gaughan DM. Particle size-dependent radical generation from wildland fire smoke. Toxicology. 2007; 236:103–113. [PubMed: 17482744]

Liu D, Tager IB, Balmes JR, Harrison RJ. The effect of smoke inhalation on lung function and airway responsiveness in wildland firefighters. Am Rev Respir Dis. 1992; 146:1469–1473. [PubMed: 1456562]

Martinet W, Knaapen MW, De Meyer GR, Herman AG, Kockx MM. Elevated levels of oxidative DNA damage and DNA repair enzymes in human atherosclerotic plaques. Circulation. 2002; 106:927–932. [PubMed: 12186795]

Materna BL, Jones JR, Sutton PM, Rothman N, Harrison RJ. Occupational exposures in California wildland firefighting. Am Ind Hyg Assoc J. 1992; 53:69–76. [PubMed: 1317093]

Migliaccio CT, Bergauff MA, Palmer CP, Simpson CD, Evanoski AR, Palmer CP. Urinary levoglucosan as a biomarker of wood smoke exposure: Observations in a mouse model and in children. Environ Health Perspect. 2009; 117:74–79. [PubMed: 19165390]

Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, Crapo R, Enright P, van der Grinten CP, Gustafsson P, et al. Standardisation of spirometry. Eur Respir J. 2005; 26:319–338. [PubMed: 16055882]

Mott JA, Mannino DM, Alverson CJ, Kiyu A, Hashim J, Lee T, Falter K, Redd SC. Cardiorespiratory hospitalizations associated with smoke exposure during the 1997Southeast Asian forest fires. Int J Hyg Environ Health. 2005; 208:75–85. [PubMed: 15881981]

Musk AW, Smith TJ, Peters JM, McLaughlin E. Pulmonary function in firefighters: Acute changes in ventilatory capacity and their correlates. Br J Ind Med. 1979; 36:29–34. [PubMed: 444439]

Public Use Data File Documentation Number 76200. Hyattsville, MD: Centers for Disease Control and Prevention; 1996. NHANES III Adult and Examination Data Files [CD-ROM].

Nuernberg AM, Boyce PD, Cavallari JM, Fang SC, Eisen EA, Christiani DC. Urinary 8- isoprostane and 8-OHdG concentrations in boilermakers with welding exposure. J Occup Environ Med. 2008; 50:182–189. [PubMed: 18301175]

Nürnberger J, Keflioglu-Scheiber A, OpazoSaez AM, Wenzel RR, Philipp T, Schäfers RF. Augmentation index is associated with cardiovascular risk. J Hypertens. 2002; 20:2407–2414. [PubMed: 12473865]

Pellegrino R, Viegi G, Brusasco V, Crapo RO, Burgos F, Casaburi R, Coates A, van der Grinten CP, Gustafsson P, Hankinson J, et al. Interpretative strategies for lung function tests. Eur Respir J. 2005; 26:948–968. [PubMed: 16264058]

Rothman N, Ford DP, Baser ME, Hansen JA, O'Toole T, Tockman MS, Strickland PT. Pulmonary function and respiratory symptoms in wildland firefighters. J Occup Med. 1991; 33:1163–1167. [PubMed: 1765858]

Sardinas A, Miller JW, Hansen H. Ischemic heart disease mortality of firemen and policemen. Am J Public Health. 1986; 76:1140–1141. [PubMed: 3740340]

Scannell CH, Balmes JR. Pulmonary effects of firefighting. Occup Med. 1995; 10:789–801. [PubMed: 8903749]

Simoneit BRT, Schauer JJ, Nolte CG, Oros DR, Elias VO, Fraser MP, Rogge WF, Cass GR. Levoglucosan, a tracer for cellulose in biomass burning and atmospheric particles. Atmos Environ. 1999; 33:173–182.

Slaughter JC, Koenig JQ, Reinhardt TE. Association between lung function and exposure to smoke among firefighters at prescribed burns. J Occup Environ Hyg. 2004; 1:45–49. [PubMed: 15202156]

Takeyama H, Itani T, Tachi N, Sakamura O, Murata K, Inoue T, Takanishi T, Suzumura H, Niwa S. Effects of shift schedules on fatigue and physiological functions among firefighters during night duty. Ergonomics. 2005; 48:1–11. [PubMed: 15764302]

Vallyathan V, Castranova V, Pack D, Leonard S, Shumaker J, Hubbs AF, Shoemaker DA, Ramsey DM, Pretty JR, McLaurin JL. Freshly fractured quartz inhalation leads to enhanced lung injury and inflammation: Potential role of free radicals. Am J Respir Crit Care Med. 1995; 152:1003–1009. [PubMed: 7663775]

Vlachopoulos C, Aznaouridis K, O'Rourke MF, Safar ME, Baou K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with central haemodynamics: A systematic review and meta-analysis. Eur Heart J. 2010; 31:1865–1871. [PubMed: 20197424]

Wasserfallen JB, Gold K, Schulman KA, Baraniuk JN. Development and validation of a rhinoconjunctivitis and asthma symptom score for use as an outcome measure in clinical trials. J Allergy Clin Immunol. 1997; 100:16–22. [PubMed: 9257782]

Wegesser TC, Pinkerton KE, Last JA. California wildfires of 2008: coarse and fine particulate matter toxicity. Environ Health Perspect. 2009; 117:893–897. [PubMed: 19590679]

Willett WC, Sampson L, Stampfer MJ, Rosner B, Bain C, Witschi J, Hennekens CH, Speizer FE. Reproducibility and validity of a semiquantitative food frequency questionnaire. Am J Epidemiol. 1985; 122:51–65. [PubMed: 4014201]

Yoo HL, Franke WD. Prevalence of cardiovascular disease risk factors in volunteer firefighters. J Occup Environ Med. 2009; 51:958–962. [PubMed: 19620889]

TABLE I

Demographic Characteristics of Interagency Hotshot Crew Members

Variable	All (N =38)	Alpine interagency hotshot crew (n =18)	Pike interagency hotshot crew (n =20)
Age (years)[a]	29 (4.34)	29.9 (5.44)	28.2 (2.97)
Time spent as a firefighter (years)[a]	3.65 (3.38)	4.12 (3.89)	3.25 (2.92)
Male %	100	100	100
White, non-Hispanic n, %	34 (90)	15 (83)	19 (95)
Current smoker n, %	2 (5)	1 (6)	1(5)
Former smoker n, %	10 (27)	2 (11)	9 (45)
Current chewing tobacco (yes vs. no) n, %	16 (42)	7 (39)	9 (45)
Volunteer firefighter n, %	6 (16)	5 (28)	1 (5)
Permanent employee n, %	11 (29)	5 (28)	6 (30)

[a]Mean value (standard deviation).

TABLE II

Clinical Characteristics of Interagency Hotshot Crew Members

Variable	All (N =38)	Alpine interagency hotshot crew (n =18)	Pike interagency hotshot crew (n =20)
Augmentation index % (adjusted for heart rate to 75 BPM)[a]	5 (13)	10 (11)	1.0 (13)[b]
Hypertension (ever) n, %	3 (8)	2 (11)	1 (5)
Elevated cholesterol (ever) n, %	4 (10)	3 (17)	1 (5)
Total cholesterol (mg/dl)[a]	170 (35.8)	175 (40.1)	164 (28.4)
HDL-cholesterol (mg/dl)[a]	55.3 (10.6)	54.6 (11.4)	56.25 (9.81)
LDL-cholesterol (mg/dl)[a]	93.8 (28.1)	101 (32.1)	82.9 (16.5)
Triglycerides (mg/dl)[a]	109 (61.4)	97.6 (39.1)	125.8 (84.0)
HsCRP (mg/L)[a]	1.25 (1.95)	1.23 (2.11)	1.28 (1.78)
Fibrinogen (mg/dl)[a]	270.2 (60.2)	295 (41.2)	221 (64.3)
Log_{10} 8-isoprostane (ng/ml)[a]	−0.14 (0.42)	−0.20 (0.41)	−0.08 (0.42)
Log_{10} 8-OHdG (ng/ml)[a,c]	0.75 (0.36)	0.61 (0.39)	0.88 (0.29)[b]
Oxidative stress score[a,d]	0.41 (0.45)	0.48 (0.45)	0.34 (0.45)
Log_{10} levoglucosan (μg/ml)[a]	1.17 (0.31)	1.14 (0.25)	1.20 (0.37)
Asthma (ever) n, %	7 (18)	6 (33)	1 (5)
Allergies (ever) n, %	17 (45)	11 (61)	6 (30)
Upper respiratory symptom score[a,e]	7.63 (7.1)	7.28 (7.11)	7.95 (7.21)
Lower respiratory symptom score[a,f]	2.34 (3.87)	1.56 (2.52)	3.05 (4.73)
Pulmonary function[a]			
FEV_1%-predicted	103 (10.2)	101 (10.1)	104 (10.5)
FVC%-predicted	107 (13.0)	104 (11.4)	111 (13.7)
FEV_1/FVC	95.6 (9.14)	97.1 (9.80)	94.3 (8.53)

[a] Mean value and standard deviation.

[b] Significantly different from Alpine Interagency Hotshot Crew at *P*-value <0.05.

[c] 8-hydroxy-2′-deoxyguanosine (8-OHdG).

[d] Derived from the average of z-scores for 8-isoprostane and 8-hydroxy-2′-deoxyguanosine(8-OHdG).

[e] Range 0–24; higher score denotes more frequent symptoms.

[f] Range 0–19; higher score denotes more frequent symptoms.

TABLE III

Predictors of Arterial Stiffness (Augmentation Index %), Linear Regression Estimates and 95% CIs

Variable[a]	Unadjusted	Adjusted[b]
Smoking history (ever)	11.2 (2.81, 19.7)	11.5 (3.73, 19.3)
Oxidative stress score[c]	10.2 (1.35, 19.0)	10.5 (2.51, 18.5)

[a] Age, chewing tobacco status, HDL-cholesterol, LDL-cholesterol, triglycerides, high sensitivity CRP, fibrinogen, allergy history, lower respiratory symptom score, upper respiratory symptom score, Interagency Hotshot Crew, FEV_1 %-predicted, FVC %-predicted, and FEV_1/FVC %-predicted were not significantly associated with mean augmentation index % values.

[b] Estimates controlling for all other specified variables in the Table III.

[c] Average z-scores of $\log_{10}$ values of 8-hydroxy-2′-deoxyguanosine (8-OHdG) and 8-isoprostane.

TABLE IV

Predictors of Oxidative DNA Damage and Repair (8-OHdG[a]), Linear Regression Estimates and 95% CIs

Log_{10} 8-OHdG [a,b]	Unadjusted	Adjusted[c]
Log_{10} levoglucosan[d] concentration	0.45 (0.07, 0.84)	0.41 (0.04, 0.79)
Interagency Hotshot Crew	0.27 (0.05, 0.50)	0.21 (−0.02, 0.45)

[a] 8-hydroxy-2′-deoxyguanosine (8-OHdG).

[b] Smoking/chewing tobacco status, hsCRP, HDL-cholesterol, LDL-C, triglycerides, fibrinogen, asthma history, allergy history, lower respiratory symptom score, upper respiratory symptom score, FEV_1%-predicted FVC %-predicted, and FEV_1/FVC %-predicted, and age were not significantly associated with mean log_{10} 8-hydroxy-2′-deoxyguanosine (8-OHdG) values.

[c] Estimates controlling for all other specified variables in the table.

[d] Levoglucosan (LG).

HHS Public Access

Author manuscript

Am J Ind Med. Author manuscript; available in PMC 2015 November 02.

Published in final edited form as:

Am J Ind Med. 2014 July ; 57(7): 748–756. doi:10.1002/ajim.22331.

Arterial Stiffness, Oxidative Stress, and Smoke Exposure in Wildland Firefighters

Denise M. Gaughan, ScD[1,2,*], **Paul D. Siegel, PhD**[3], **Michael D. Hughes, PhD**[4], **Chiung-Yu Chang, ScD**[2], **Brandon F. Law, MS**[3], **Corey R. Campbell, MS**[5], **Jennifer C. Richards, MS**[6], **Stefanos F. Kales, MD**[2], **Marcia Chertok, BS**[2], **Lester Kobzik, PhD**[7], **Phuongson Nguyen, PhD**[7], **Carl R. O'Donnell, PhD**[8], **Max Kiefer, MS**[5], **Gregory R. Wagner, MD**[2], and **David C. Christiani, MD**[2]

[1]Department of Preventive Medicine and the Institute for Translational Epidemiology, Icahn School of Medicine at Mount Sinai, New York, New York

[2]Department of Environmental Health (Environmental and Occupational Medicine and Epidemiology Program), Harvard School of Public Health, Boston, Massachusetts

[3]Health Effects Laboratory Division, National Institute for Occupational Safety and Health, Morgantown, West Virginia

[4]Departmentof Biostatistics, Harvard School of Public Health, Boston, Massachusetts

[5]Western States Office, National Institute for Occupational Safety and Health, Denver, Colorado

[6]Department of Health and Exercise Science, Colorado State University, Fort Collins, Colorado

[7]Department of Environmental Health (Molecular and Integrative Physiological Sciences), Harvard School of Public Health, Boston, Massachusetts

[8]Beth Israel Deaconess Medical Center, Division of Pulmonary and Critical Care Medicine, Boston, Massachusetts

Abstract

Objectives—To assess the association between exposure, oxidative stress, symptoms, and cardiorespiratory function in wildland firefighters.

Methods—We studied two Interagency Hotshot Crews with questionnaires, pulse wave analysis for arterial stiffness, spirometry, urinary 8-iso-prostaglandin F2α (8-isoprostane) and 8-hydroxy-2′-deoxyguanosine (8-OHdG), and the smoke exposure marker (urinary levoglucosan). Arterial stiffness was assessed by examining levels of the aortic augmentation index, expressed as a percentage. An oxidative stress score comprising the average of z-scores created for 8-OHdG and 8-isoprostane was calculated.

Results—Mean augmentation index % was higher for participants with higher oxidative stress scores after adjusting for smoking status. Specifically for every one unit increase in oxidative

[*]Correspondence to: Denise M. Gaughan, ScD, Icahn School of Medicine at Mount Sinai, 17 East 102nd Street, D3-132, New York, NY10029-6574. denise.gaughan@mssm.edu.

Disclosure Statement: The authors report no conflicts of interests.

stress score the augmentation index % increased 10.5% (95% CI: 2.5, 18.5%). Higher mean lower respiratory symptom score was associated with lower percent predicted forced expiratory volume in one second/forced vital capacity.

Conclusions—Biomarkers of oxidative stress may serve as indicators of arterial stiffness in wildland firefighters.

Keywords

vascular stiffness; 8-iso-prostaglandin F2α; 8-hydroxy-2′-deoxyguanosine; spirometry; levoglucosan

BACKGROUND

Firefighters, both structural and wildland, are known to have cardiovascular and respiratory problems [Musk et al., 1979; Sardinas et al., 1986; Chia et al., 1990; Rothman et al., 1991; Guidotti and Clough, 1992; Liu et al., 1992; Materna et al., 1992; Scannell and Balmes, 1995; Betchley et al., 1997; Austin et al., 2001; Burgess et al., 2001; Kales et al., 2003; Slaughter et al., 2004; CDC, 2006; Gaughan et al., 2008; Yoo and Franke, 2009]. Cardiovascular disease (CVD) events are the leading cause of on-duty and lifetime mortality among structural (career and volunteer) firefighters [Sardinas et al., 1986; Kales et al., 2003; Yoo and Franke, 2009]. The deleterious effects of smoke exposure to structural firefighters have been extensively researched [Musk et al., 1979; Sardinas et al., 1986; Chia et al., 1990; Guidotti and Clough, 1992; Liu et al., 1992; Scannell and Balmes, 1995; Burgess et al., 2001; Kales et al., 2003; CDC, 2006; Yoo and Franke, 2009]. Exposure to particulates and other contaminants, heavy physical exertion and cardiovascular strain have been found to be among the chief health hazards associated with structural firefighting [Gledhill and Jamnik, 1992; Takeyama et al., 2005; Delfino et al., 2009]. Those findings, however, may not be generalizable to wildland firefighters for a number of reasons, including the difference in smoke composition, comparative younger age of wildland firefighters, often shorter career tenure, and the longer duration of respiratory exposures for wildland firefighters. Additionally, structural firefighters routinely wear respiratory protection when responding to fires while wildland firefighters do not.

Fine particulate exposure has been associated with acute changes in cardiovascular and pulmonary function [Vallyathan et al., 1995; Mott et al., 2005; Dominici et al., 2006; Cavallari et al., 2008; Fang et al., 2008, 2009]. Free radical mechanisms have been implicated as a contributing factor in general toxicity, inflammation, asthma, fibrogenesis, bronchopulmonary carcinogenesis, and atherosclerotic plaque formation [Jarjour and Calhoun, 1994; Leonard et al., 2007; LeBlanc et al., 2010].

Wood fires produce smoke with abundant particles in the inhalable range (<100 μm) and contain both carbon radicals and precursors. The latter are able to react with H_2O_2 after exposure to cells and generate the highly reactive hydroxyl radical (OH) from a Fenton-like reaction [Leonard et al., 2007]. The authors additionally observed that carbon radicals were stronger (per unit mass) in larger (coarse) sized particles while OH and other ROS were stronger (per unit mass) in the smaller (ultrafine) sized particles. Finally, the authors noted

that fine and ultrafine woodsmoke particles also significantly increased H_2O_2, DNA strand breaks and lipid peroxidation in exposed RAW 264.7 cells.

Pyrolysis of organic components may increase the potency and or toxicity of particulate by producing ROS. Measurement of levoglucosan, a sugar anhydride by-product of incomplete combustion of cellulose, may be used to indicate relative exposure to products of pyrolysis from burning biomass [Simoneit et al., 1999]. A recent study examined personable exposure to airborne respirable levoglucosan using a Dorr-Oliver cyclone and air sampling pump, and cross-shift changes in lung function in a population of 17 wildland firefighters for 4 days at a large wildland fire [Gaughan et al., 2014]. The authors reported that levoglucosan was found mainly in the respirable fraction, defined as under 2.5 μm, with higher concentrations during fireline construction than in mop-up operations. Furthermore, larger cross-shift declines in forced expiratory volume in one second (FEV_1) were associated with exposure to higher concentrations of respirable levoglucosan (P-value <0.05).

Urinary levoglucosan has also been investigated as a biomarker for smoke exposure. Bergauff et al. examined cross-shift changes in urinary levoglucosan in nine firefighters exposed to wood smoke in a controlled setting. They observed elevated urinary levoglucosan following smoke exposure in some but not all firefighters [Bergauff et al., 2010]. Moreover, the authors noted the contribution of dietary intake to urinary levoglucosan levels.

Urinary 8-iso-prostaglandin F2α (8-isoprostane), a biomarker of oxidative stress generated by lipid peroxidation, may serve as a biomarker for atherosclerosis [Cipollone et al., 2000]. Oxidative deoxyribonucleic acid (DNA) damage and repair has also been linked to atherosclerosis. Martinet et al. examined five human carotid endarterectomy specimens and five mammary artery specimens for 8-hydroxy-2′-deoxyguanosine (8-OHdG), an oxidized nucleoside of DNA. The authors observed an increased amount of 8-OHdG in plaques compared to the underlying media or non-atherosclerotic mammary arteries [Martinet et al., 2002]. Urinary 8-OHdG is excreted upon DNA repair and may serve as a non-invasive biomarker of global oxidative DNA damage. For instance, acute changes in urinary 8-OHdG concentrations have been reported in occupational studies examining welders [Kim et al., 2004; Nuernberg et al., 2008].

Elevated arterial stiffness is a characteristic of large artery pathology, a major contributor to CVD, and may serve as an indicator of pre-clinical atherosclerosis and/or hypertension [Blankenhorn and Kramsch, 1989; Duprez and Cohn, 2007]. Assessment of arterial stiffness is done through ultrasound or measurement of pulse wave velocity. The aortic augmentation index, is an indirect measure of systemic arterial stiffness based on pulse wave velocity and is calculated as a percentage. A recent meta-analysis demonstrated that a 10% increase in augmentation index % was associated with a 31.8% increased risk of cardiovascular events and a 34.8% increased risk of total mortality [Vlachopoulos et al., 2010]. The augmentation index % has been successfully implemented in occupational research settings [Nürnberger et al., 2002; Fang et al., 2008].

There are four types of wildland firefighter suppression crews: engine crew, hand crew, helicopter crew, and smoke jumpers. Type 1 Interagency Hotshot Crews are an elite type of hand crew, comprising up to 20 firefighters who construct fire lines using hand tools during the most dangerous phases of fire suppression.

The question addressed by the present study was whether wildland firefighting exposures are associated with oxidative stress concentrations and with pulmonary and vascular function. To answer these questions, we assessed spirometry, vascular function, symptoms, and systemic biomarkers of exposure, inflammation and oxidative stress in members of two Type 1 wildland firefighter Interagency Hotshot Crews.

METHODS

The National Interagency Fire Center (NIFC) Risk Management Committee arranged for two crews to participate in this study: the Alpine Interagency Hotshot Crew, Rocky Mountain National Park, Estes Park, CO and the Pike Interagency Hotshot Crews, Pike and San Isabel National Forests, Monument, CO. The Interagency Hotshot Crews participated in our study by completing questionnaires, pulse wave analysis and spirometry in May 2011. Serum cholesterol, and biomarkers of systemic inflammation (high sensitivity c-reactive protein (hsCRP) and fibrinogen), oxidative stress (8-isoprostane), oxidative DNA damage (8-OHdG), and smoke exposure (urinary levoglucosan) were measured. The study protocol was approved by the Harvard School of Public Health (HSPH) Institutional Review Board and the National Institute for Occupational Safety and Health (NIOSH) Human Subjects Review Board. Informed consent was obtained from each research participant.

Medical Testing Methods

Pulse wave analysis—Vascular function was measured using a pulse wave analysis system according to the manufacturer's instructions [SphygmoCor CP, Atcor Medical Pty Ltd., Sydney, Australia]. Briefly, participants were seated with the dominant arm extended onto a flat surface so that the antecubital fossa was at heart level. Following 5 min of rest, a high-fidelity micro-nanometer was used to flatten the radial artery with gentle pressure. Ten seconds of sequential pulse pressure waveforms were recorded at each reading. The waveforms were then transformed into a corresponding central aortic waveform via a validated transfer function where the systolic part of the central aortic waveform is characterized by a first peak caused by left ventricular ejection and a second peak caused by wave reflection. The difference between the two peaks reflects the degree to which the central aortic pressure is augmented by wave reflection. We calculated each participant's aortic augmentation index %, defined as the ratio of augmented pressure to pulse pressure (i.e., augmentation index % =augmented pressure/pulse pressure ×100) [Nürnberger et al., 2002] and heart rate corrected to 75 beats per minute. Larger augmentation index % values denote increased wave reflection. A minimum of three within-session recordings were obtained from each participant.

Spirometry—Pulmonary function was determined on each participant using an ultrasonic flow spirometer [EasyOne^{TM} Diagnostic Spirometry System 2001, ndd Medical Technologies, Zurich, Switzerland]. Technicians completed a NIOSH-approved spirometry

course followed American Thoracic Society (ATS) guidelines [Miller et al., 2005]. Test results were interpreted using reference values generated from the Third National Health and Nutrition Examination Survey (NHANES III) [Hankinson et al., 1999]. Airways obstruction was defined as a forced expiratory volume in the first second (FEV_1) to forced vital capacity (FVC) ratio below the lower limit of normal according to published reference equations [Pellegrino et al., 2005]. We examined percent predicted FEV_1 (FEV_1%-predicted), percent predicted FVC (FVC %-predicted), and percent predicted FEV_1/FVC (FEV_1/FVC %-predicted). We followed ATS procedure by inquiring about current medications but did not ask participants to abstain from using their medications prior to participating in this study for safety purposes. Reports were reviewed for quality by a respiratory physiologist (C.R.O) experienced in clinical pulmonary function laboratory administration.

Blood—Whole non-fasting serum samples (30 ml/sample) were collected by venous phlebotomy in EDTA tubes, and buffy coat was extracted and stored in cell lyses solution at −20°C for analyses of typical cardiovascular-related biomarkers, specifically, hsCRP and fibrinogen. We additionally examined total cholesterol, high-density lipoprotein cholesterol (HDL-cholesterol), low-density lipoprotein cholesterol (LDL-cholesterol), and triglycerides. These analyses were conducted by Quest Diagnostics Inc., Denver, CO.

Urine—Urine samples were analyzed for 8-OHDG and 8-isoprostane using competitive enzyme-linked immunoassays (EKS-350, Assay Designs, Inc., Ann Arbor, MI; 8-Isoprostane EIA Kit, Cayman Chemical Company, Ann Arbor, MI) as well as for creatinine (picric acid colorimetric assay; Oxford Biomedical Research, Oxford, MI). We also examined urine for levoglucosan concentration. Two hundred microliter of urine or levoglucosan standards (Blank, 6.25–100 μg/ml in saline) were added to 1.5 ml low retention microcentrifuge tubes. To this 30U of urease (Sigma, St. Louis, MO) was added and incubated for 1.0 hr at 37°C. After incubation, 600 μl of cold (4°C) ethanol was added to precipitate the protein. Approximately 400 mg of sodium sulfate was added volumetrically and allowed to sit for 2.0 min. Samples and standards were then centrifuged for 2.0 min at 14,000*g*. After centrifugation, 600 μl of each were transferred to clean low retention microcentrifuge tubes and evaporated to dryness using a vacuum centrifugation and gentle heat. Two hundred microliter of n-methyl-n-(trimethylsilyl) trifluoroacet (MSTFA) (Sigma Chemical Co.,) was then added to each tube, vortexed and incubated at 72°C for 1.0 hr. After derivatization samples and standards were analyzed on an Agilent 6890 gas chromatograph coupled to an Agilent 5975C mass spectrometer using a 30-m HP5-MS column (Agilent Technologies, Santa Clara, CA). Samples were injected (1 μl) in splitless mode into a 250°C inlet with a 6.0-min solvent delay. Analytes were eluted from the column using 1.0 ml/min helium and an oven temperature program as follows: 70°C for 3.0 min and then ramped at 25.0°C/min to a final temperature of 275°C. The MS source temperature was maintained at 230°C, and the quadrupole temperature was maintained at 150°C. Ions were scanned between 50 and 400 m/z. Levoglucosan from each sample was identified by the MS spectra and retention time (against the known standards) and quantified using the 204 m/z ion. The standard plot from which the samples were extrapolated used a polynomial curve fit of 204 m/z area under the curve ion count versus standard concentration. Specimens were

analyzed by NIOSH's Health Effects Laboratory Division, Allergy and Clinical Immunology Branch, Morgantown, WV.

Questionnaires—The questionnaire was based on two standardized questionnaires, the American Thoracic Society-Division of Lung Disease-78 (ATS-DLD-78) supplemented with questions from NHANES III [Ferris, 1978; Wasserfallen et al., 1997]. This modified questionnaire was designed to acquire information concerning chronic cardiovascular and respiratory conditions; lifetime diagnoses; tobacco history; symptom history; dietary intake; sleep patterns; medication use; and occupational history. A validated symptom scale, with Likert scoring where 0 =none, 1 =trivial, 2 =mild, 3 =moderate, and 4 =severe for upper and lower airways symptoms, was used to derive overall symptom scores by summing the responses to questions about 19 symptoms. Symptoms ascertained included cough, wheeze, sputum production, shortness of breath or chest tightness, and shortness of breath while walking, as well as various eye, nose, and throat symptoms. Subjects additionally completed a semi-quantitative food frequency questionnaire, adult version, 2007. Subjects were asked to report the average daily consumption of various foods in the preceding year. Responses ranged from "never" to "six or more servings per day." The food frequency questionnaire also assessed the frequency of multivitamin and mineral supplement usage. Frequency factors of related foods items were then summed to calculate the daily servings for each food group. The nutrient value of the food item was multiplied by the frequency of consumption in order to obtain macro and micro nutrient intake. Scoring was done by the Nutrition Department, Harvard School of Public Health (https://regepi.bwh.harvard.edu/health/nutrition.html) [Willett et al., 1985].

Statistical Methods—Descriptive statistics were calculated for demographic and clinical variables. Mean values among subgroups were compared using Student's *t*-test techniques. Ordinary least squares regression techniques were used to examine associations between augmentation index %, FEV_1%-predicted, FVC %-predicted, FEV_1/FVC %-predicted, 8-OHdG concentration, and 8-isoprostane concentration using the following predictor variables: levoglucosan concentration, serum lipid levels, hsCRP concentration, fibrinogen concentration, LDL-cholesterol, HDL-cholesterol, Interagency Hotshot Crew, volunteer firefighter (yes or no), cumulative time spent fighting fires (seasons), medical diagnoses, allergies, upper and lower respiratory symptom scores, and history of tobacco use. Levoglucosan, 8-isoprostane, 8-OHdG, and hsCRP values were not normally distributed and thus the data were $\log_{10}$-transformed to achieve an approximate normal distribution. We additionally calculated an "oxidative stress score" comprising z-score's of 8-isoprostane and 8-OHdG, where z-value's for each variable were calculated and then averaged to yield a score for each participant. The motivation for calculating the z-score was prompted by the possibility that while 8-isoprostane is considered a marker of lipid peroxidation and 8-OHdG has been linked to DNA damage and repair, they may both be measuring the overall level of oxidative stress in the urine. Thus, we standardized the two biomarkers of oxidative stress on one scale. These values were also examined in multivariable analysis. The z-value was calculated by subtracting the mean value from each subject's value and then dividing by the standard deviation of the values. Multivariable models were chosen based on an initial evaluation using stepwise selection techniques, followed by examining univariate

associations and sequentially adjusting for other predictors. Associations were considered statistically significant if *P*-values were <0.05 and borderline significant at ≥0.05 and <0.10. All analyses were conducted using SAS statistical software (version 9.3).

RESULTS

Thirty-eight of the 39 current members of both crews participated in the surveys in May 2011 during their training sessions at their respective home base parks (97%). One member was training off-site the day of our survey and as a result, could not participate. Members of the Pike Interagency Hotshot Crew had been exposed to smoke for 2 days at the Sand Gulch fire 4 days before the testing. The Sand Gulch fire in Wetmore, CO, was a 495-acre wildland fire at 7,000 feet elevation in high difficulty terrain. Fire behavior activity was described by crew members as high/extreme on the first day and low/smoldering on the second. It was reported that the average shift duration each day was 16 hr and it was estimated the crew members were on the fire line performing firefighting activities for 12.5 hr each day.

Participant demographic and clinical characteristics, overall and stratified by crew are shown in Tables I and II. The two crews were very similar. They were all male with a median age of 28 years and had spent a median of 3 years (seasons) working as an Interagency Hotshot Crew member. Approximately 5% of the participants were current smokers and nearly half (42%) reported current chewing tobacco use. Approximately one-third (29%) of the participants were classified as “permanent” employees, the remainder were “seasonal” hires. Additionally, six of the participants (16%) reported working as a volunteer structural firefighter off-season. Values for clinical characteristics were also comparable when examined by crew and within the normal range.

Seven participants reported a history of physician-diagnosed asthma (18%). The median age at asthma diagnosis was 7 years and the median time spent as a firefighter for these seven participants was 2 years (seasons). Among these individuals, the median FEV_1%-predicted was 105%, the median FVC %-predicted was 109%, and the median FEV_1/FVC %-predicted was 96%. Among the four individuals with current asthma, three were currently taking medication for their asthma. One of the seven participants with a history of asthma also reported a smoking history.

Four values for levoglucosan were below the limit of detection and were not included in the analysis. Detectable levels were observed for all values of 8-OHdG, 8-isoprostane, hsCRP, and fibrinogen.

In multivariable analysis, mean augmentation index % was higher for participants with higher oxidative stress scores. Specifically, for every one unit increase in oxidative stress score, mean augmentation index % increased 10.2% (10.2%, 95% CI: 1.35, 19.0%) (Table III). This association remained significant after adjusting for smoking status (*P* =0.01). No other variables were associated with augmentation index %, including our exposure variable, levoglucosan. Table III details these results.

However, higher levoglucosan concentration was positively associated with oxidative stress scores (Table IV). Specifically, for every twofold increase in $\log_{10}$ levoglucosan

concentration, mean 8-OHdG increased by $\log_{10}$ 0.14 μg/ml (95% CI: 0.02, 0.25) (regression estimate 0.41 (95% CI: 0.04, 0.79)) and 8-isoprostane increased by $\log_{10}$ 0.16 ng/ml (95% CI: 0.02, 0.29) (regression estimate 0.52 (95% CI: 0.06, 0.97)). Additionally, 8-OHdG values for participants who had recently been exposed to smoke particulate as measured by Interagency Hotshot Crew were higher than those with no recent exposure (P =0.01). This association became borderline significant after adjusting for levoglucosan (P =0.07) (Table IV).

Finally, higher lower respiratory symptom score (LRSS) was associated with lower FEV_1/FVC %-predicted after adjusting for smoking status and history of asthma. Specifically, for every twofold increase in mean LRSS, the mean FEV_1/FVC %-predicted dropped on average by 1.66% (95% CI: 0.10%, 3.22%) (regression estimate:−0.83 (95% CI: −1.61, −0.05)). No other variables were associated with FEV_1/FVC %-predicted, most notably, neither oxidative stress score nor levoglucosan.

DISCUSSION

Particulate exposure has been associated with increased arterial stiffness in occupational cohorts. Fang et al. examined changes in augmentation index % in 26 welders over 24 hr on a welding day and non-welding day. Following welding fume exposure, the authors observed an increase in afternoon augmentation index % and a decrease in next morning augmentation index % [Fang et al., 2008]. The results suggested that exposure to welding fume particulate is associated with acute adverse vascular responses. In our homogenous group of healthy workers, we observed higher augmentation index % values for participants with higher oxidative stress values which were also associated with larger levoglucosan values. A possible explanation for this finding could be that our population was younger by comparison (28 vs. 41 years) and/or our assessment occurred 4 days post-exposure to smoke.

Similar to other studies, we observed an association between cross-shift differences in oxidative DNA damage and recent occupational exposure to particulate matter. Kim et al. obtained 5 days of cross-shift urinary 8-OHdG measurements from 20 welders exposed to metal fumes. The authors reported that urinary 8-OHdG levels were significantly elevated in post-shift samples compared to those collected pre-shift [Kim et al., 2004]. Nuernberg et al. [2008] observed similar 8-OHdG elevation post-shift in 63 welders. Moreover, the authors found an unexpected inverse relationship between post-shift and 8 hr post-shift values of 8-isoprostane and $PM_{2.5}$ (P <0.05). We additionally observed a difference in 8-isoprostane by levoglucosan concentration. We did not observe a difference in mean values of 8-OHdG or 8-isoprostane between participants who identified themselves as having a history of smoking and those who did not (P =0.51 and 0.64, respectively).

Levoglucosan was positively associated with both biomarkers of oxidative stress, 8-OHdG and 8-isoprostane. Urinary levoglucosan has been shown to increase in mice after exposure to wood smoke [Migliaccio et al., 2009]. However, when examining urinary levoglucosan concentration, it is important to control for dietary confounders such as smoked or fried foods and caramel. Based on responses to the Food Frequency Questionnaire, 32 (84%)

reported consuming two slices of bacon or less once per week; 34 (89%) reported eating one candy bar or less per week. All participants reported eating fried foods at home or away four to six times per week or less, although 22 (58%) reported consuming fried foods less than once per week. Inhaled levoglucosan is probably completely eliminated from the body within 24 hr post-exposure. Thus, it is possible that the elevated levels of levoglucosan we observed were caused by diet, rather than exposure.

There is evidence that particulate matter [PM] produced by woodsmoke fires may be more toxic than PM from ambient air due to the chemical components found in the smoke [Wegesser et al., 2009]. Leonard et al. [2007] examined aerodynamically size-selected aerosol samples at a large wildland fire. The authors found smoke particles in all size fractions [from smaller than 0.056 μm to greater than 10 μm] and a bell-shaped distribution with highest overall mass fraction on filters in the fine range. The authors additionally observed that radical signals were size dependent and resulted in reactive oxygen species (ROS) generation.

Recent animal studies suggest that exposure to ultrafine particles may additionally augment cardiac dysfunction through an ROS mechanism [LeBlanc et al., 2010]. LeBlanc et al. recently demonstrated that local ROS generation can influence vascular reactivity in coronary arterioles. It follows that excessive coronary ROS generation following pulmonary woodsmoke exposure could impair endothelium-dependent arteriolar reactivity.

Seven participants reported a history of asthma; four with current asthma. Similar asthma prevalences were previously observed in this population [Gaughan et al., 2008]. We did not see a difference in %-predicted lung function values in these subjects from the rest nor did we observe an association between oxidative stress score or levoglucosan and a history of asthma. However, we did observe that higher LRSS were associated with lower FEV_1/FVC %-predicted after adjusting for asthma history and smoking status ($P < 0.05$).

We recognize several limitations to our study. Our sample size was small and the resultant lack of statistical power may have hindered our ability to observe associations if they did exist. While we did observe a significant association between higher oxidative stress scores with recent firefighting activities, the generally qualitative and self-reported nature of exposure characterization in our study may have limited our ability to have identified statistically significant cardiopulmonary effects related to firefighting exposures. Additionally, this was a cross-sectional study with essentially no control group. This design limited examination of acute changes. Our study also lacked cross-shift data during periods in which the crews were working but not fighting a fire. Future studies should examine these individuals cross-shift at a wildland fire and when they are not being exposed to smoke aerosol to ascertain whether any observed changes are part of their normal variation. However, a recent study did not find significant changes in cross-shift lung function on burn days compared to non-burn days [Adetona et al., 2011]. Also, urinary levoglucosan may be more representative of diet than as an indicator of wood-smoke exposure. Finally, all spirometric values were obtained pre-bronchodilator. Post-bronchodilator values would have allowed us to examine reversibility and may have been particularly informative for participants reporting a history of asthma.

This is the first study to examine the association between systemic oxidative stress and arterial stiffness in wildland firefighters. Future studies should additionally examine endothelial function which is governed by smooth muscle tone, collage/elastin, calcification, and other factors that affect the entire arterial wall. The two together could provide different but complementary information on vascular health.

Acknowledgments

We thank the Alpine and Pike Interagency Hotshot Crews for their participation as well as the National Interagency Fire Center, Risk Management Committee, specifically, Chad Fisher, U.S. Department of Interior, National Park Service and Larry Sutton, U.S.D.A. Forest Service for arranging for the Interagency Hotshot Crews to participate. The authors additionally thank Dr. Shona Fang, Li Su, and Lauren Cassidy for assistance preparing for the field surveys and Dr. Kristin Cummings for editorial comments.

References

Adetona O, Hall DB, Naeher LP. Lung function changes in wildland firefighters working at prescribed burns. Inhal Toxicol. 2011; 23:835–841. [PubMed: 22035123]

Austin C, Wang D, Ecobichon DJ, Dussault G. Characterization of volatile organic compounds in smoke at municipal structural fires. J Toxicol Environ Health. 2001; 63:437–458.

Bergauff MA, Ward TJ, Noonan CW, Migliaccio CT, Simpson CD, Evanoski AR, Palmer CP. Urinary levoglucosan as a biomarker of wood smoke: Results of human exposure studies. J Expo Sci Environ Epidemiol. 2010; 20:385–392. [PubMed: 19707249]

Betchley C, Koenig JQ, vanBelle G, Checkoway H, Reinhardt T. Pulmonary function and respiratory symptoms in forest firefighters. Am J Ind Med. 1997; 31:503–509. [PubMed: 9099351]

Blankenhorn DH, Kramsch DM. Reversal of atherosis and sclerosis. The two components of atherosclerosis. Circulation. 1989; 79:1–7. [PubMed: 2642753]

Burgess JL, Nanson CJ, Bolstad-Johnson DM, Gerkin R, Hysong TA, Lantz RC, Sherrill DL, Crutchfield CD, Quan SF, Bernard AM, et al. Adverse respiratory effects following overhaul in firefighters. J Occup Environ Med. 2001; 43:467–473. [PubMed: 11382182]

Cavallari JM, Eisen EA, Fang SC, Schwartz J, Hauser R, Herrick RF, Christiani DC. PM 2.5 metal exposures and nocturnal heart rate variability: A panel study of boilermaker construction workers. Environ Health. 2008; 7:36. [PubMed: 18613971]

Centers for Disease Control and Prevention [CDC]. Fatalities among volunteer and career firefighters–United States, 1994–2004. CDC MMWR Morb Mortal Wkly Rep. 2006; 55(16):453–455. [PubMed: 16645570]

Chia KS, Jeyaratnam J, Chan TB, Lim TK. Airway responsiveness of firefighters after smoke exposure. Br J Ind Med. 1990; 47:524–527. [PubMed: 2393631]

Cipollone F, Ciabattoni G, Patrignani P, Pasquale M, Di Gregorio D, Bucciarelli T, Davì G, Cuccurullo F, Patrono C. Oxidant stress and aspirin-insensitive thromboxane biosynthesis in severe unstable angina. Circulation. 2000; 102:1007–1013. [PubMed: 10961965]

Delfino RJ, Brummel S, Wu J, Stern H, Ostro B, Lipsett M, Winer A, Street DH, Zhang L, Tjoa T, et al. The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003. Occup Environ Med. 2009; 66:189–197. [PubMed: 19017694]

Dominici F, Peng RD, Bell ML, Pham L, McDermott A, Zeger SL, Samet JM. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA. 2006; 295:1127–1134. [PubMed: 16522832]

Duprez DA, Cohn JN. Arterial stiffness as a risk factor for coronary atherosclerosis. Curr Atheroscler Rep. 2007; 9:139–144. [PubMed: 17877923]

Fang SC, Eisen EA, Cavallari JM, Mittleman MA, Christiani DC. Acute changes in vascular function among welders exposed to metal-rich particulate matter. Epidemiology. 2008; 19:217–225. [PubMed: 18300696]

Fang SC, Cavallari JM, Eisen EA, Chen JC, Mittleman MA, Christiani DC. Vascular function, inflammation, and variations in cardiac autonomic responses to particulate matter among welders. Am J Epidemiol. 2009; 169:848–856. [PubMed: 19153215]

Ferris BG. Epidemiology standardization project. Am Rev Respir Dis. 1978; 108:1–113. [PubMed: 742764]

Gaughan DM, Cox-Ganser JM, Enright PL, Castellan RM, Wagner GR, Hobbs GR, Bledsoe TA, Siegel PD, Kreiss K, Weissman DN. Acute upper and lower respiratory effects in wildland firefighters. J Occup Environ Med. 2008; 50:1019–1028. [PubMed: 18784550]

Gaughan DM, Piacitelli CA, Chen BT, Law BF, Virji MA, Edwards NT, Enright PL, Schwegler-Berry DE, Leonard SS, Wagner GR, et al. Exposures and cross-shift lung function declines in wildland firefighters. J Occup Environ Hyg. 2014 Feb 25. Epub ahead of print. 10.1080/15459624.2014.895372

Gledhill N, Jamnik VK. Characterization of the physical demands of firefighting. Can J Sport Sci. 1992; 17:207–213. [PubMed: 1325260]

Guidotti TL, Clough VM. Occupational health concerns of firefighting. Annu Rev Public Health. 1992; 13:151–171. [PubMed: 1599583]

Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. Am J Respir Crit Care Med. 1999; 159:179–187. [PubMed: 9872837]

Jarjour NN, Calhoun WJ. Enhanced production of oxygen radicals in asthma. J Lab Clin Med. 1994; 123:131–136. [PubMed: 8288953]

Kales SN, Soteriades ES, Christoudias SG, Christiani DC. Firefighters and on-duty deaths from coronary heart disease: A case control study. Environ Health. 2003; 2:14. [PubMed: 14613487]

Kim JY, Mukherjee S, Ngo LC, Christiani DC. Urinary 8-hydroxy-2′-deoxyguanosine as a biomarker of oxidative DNA damage in workers exposed to fine particulates. Environ Health Perspect. 2004; 112:666–671. [PubMed: 15121508]

LeBlanc AJ, Moseley AM, Chen BT, Frazer D, Castranova V, Nurkiewicz TR. Nanoparticle inhalation impairs coronary microvascular reactivity via a local reactive oxygen species-dependent mechanism. Cardiovasc Toxicol. 2010; 10:27–36. [PubMed: 20033351]

Leonard SS, Castranova V, Chen BT, Schwegler-Berry D, Hoover M, Piacitelli C, Gaughan DM. Particle size-dependent radical generation from wildland fire smoke. Toxicology. 2007; 236:103–113. [PubMed: 17482744]

Liu D, Tager IB, Balmes JR, Harrison RJ. The effect of smoke inhalation on lung function and airway responsiveness in wildland firefighters. Am Rev Respir Dis. 1992; 146:1469–1473. [PubMed: 1456562]

Martinet W, Knaapen MW, De Meyer GR, Herman AG, Kockx MM. Elevated levels of oxidative DNA damage and DNA repair enzymes in human atherosclerotic plaques. Circulation. 2002; 106:927–932. [PubMed: 12186795]

Materna BL, Jones JR, Sutton PM, Rothman N, Harrison RJ. Occupational exposures in California wildland firefighting. Am Ind Hyg Assoc J. 1992; 53:69–76. [PubMed: 1317093]

Migliaccio CT, Bergauff MA, Palmer CP, Simpson CD, Evanoski AR, Palmer CP. Urinary levoglucosan as a biomarker of wood smoke exposure: Observations in a mouse model and in children. Environ Health Perspect. 2009; 117:74–79. [PubMed: 19165390]

Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, Crapo R, Enright P, van der Grinten CP, Gustafsson P, et al. Standardisation of spirometry. Eur Respir J. 2005; 26:319–338. [PubMed: 16055882]

Mott JA, Mannino DM, Alverson CJ, Kiyu A, Hashim J, Lee T, Falter K, Redd SC. Cardiorespiratory hospitalizations associated with smoke exposure during the 1997Southeast Asian forest fires. Int J Hyg Environ Health. 2005; 208:75–85. [PubMed: 15881981]

Musk AW, Smith TJ, Peters JM, McLaughlin E. Pulmonary function in firefighters: Acute changes in ventilatory capacity and their correlates. Br J Ind Med. 1979; 36:29–34. [PubMed: 444439]

Public Use Data File Documentation Number 76200. Hyattsville, MD: Centers for Disease Control and Prevention; 1996. NHANES III Adult and Examination Data Files [CD-ROM].

Nuernberg AM, Boyce PD, Cavallari JM, Fang SC, Eisen EA, Christiani DC. Urinary 8- isoprostane and 8-OHdG concentrations in boilermakers with welding exposure. J Occup Environ Med. 2008; 50:182–189. [PubMed: 18301175]

Nürnberger J, Keflioglu-Scheiber A, OpazoSaez AM, Wenzel RR, Philipp T, Schäfers RF. Augmentation index is associated with cardiovascular risk. J Hypertens. 2002; 20:2407–2414. [PubMed: 12473865]

Pellegrino R, Viegi G, Brusasco V, Crapo RO, Burgos F, Casaburi R, Coates A, van der Grinten CP, Gustafsson P, Hankinson J, et al. Interpretative strategies for lung function tests. Eur Respir J. 2005; 26:948–968. [PubMed: 16264058]

Rothman N, Ford DP, Baser ME, Hansen JA, O'Toole T, Tockman MS, Strickland PT. Pulmonary function and respiratory symptoms in wildland firefighters. J Occup Med. 1991; 33:1163–1167. [PubMed: 1765858]

Sardinas A, Miller JW, Hansen H. Ischemic heart disease mortality of firemen and policemen. Am J Public Health. 1986; 76:1140–1141. [PubMed: 3740340]

Scannell CH, Balmes JR. Pulmonary effects of firefighting. Occup Med. 1995; 10:789–801. [PubMed: 8903749]

Simoneit BRT, Schauer JJ, Nolte CG, Oros DR, Elias VO, Fraser MP, Rogge WF, Cass GR. Levoglucosan, a tracer for cellulose in biomass burning and atmospheric particles. Atmos Environ. 1999; 33:173–182.

Slaughter JC, Koenig JQ, Reinhardt TE. Association between lung function and exposure to smoke among firefighters at prescribed burns. J Occup Environ Hyg. 2004; 1:45–49. [PubMed: 15202156]

Takeyama H, Itani T, Tachi N, Sakamura O, Murata K, Inoue T, Takanishi T, Suzumura H, Niwa S. Effects of shift schedules on fatigue and physiological functions among firefighters during night duty. Ergonomics. 2005; 48:1–11. [PubMed: 15764302]

Vallyathan V, Castranova V, Pack D, Leonard S, Shumaker J, Hubbs AF, Shoemaker DA, Ramsey DM, Pretty JR, McLaurin JL. Freshly fractured quartz inhalation leads to enhanced lung injury and inflammation: Potential role of free radicals. Am J Respir Crit Care Med. 1995; 152:1003–1009. [PubMed: 7663775]

Vlachopoulos C, Aznaouridis K, O'Rourke MF, Safar ME, Baou K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with central haemodynamics: A systematic review and meta-analysis. Eur Heart J. 2010; 31:1865–1871. [PubMed: 20197424]

Wasserfallen JB, Gold K, Schulman KA, Baraniuk JN. Development and validation of a rhinoconjunctivitis and asthma symptom score for use as an outcome measure in clinical trials. J Allergy Clin Immunol. 1997; 100:16–22. [PubMed: 9257782]

Wegesser TC, Pinkerton KE, Last JA. California wildfires of 2008: coarse and fine particulate matter toxicity. Environ Health Perspect. 2009; 117:893–897. [PubMed: 19590679]

Willett WC, Sampson L, Stampfer MJ, Rosner B, Bain C, Witschi J, Hennekens CH, Speizer FE. Reproducibility and validity of a semiquantitative food frequency questionnaire. Am J Epidemiol. 1985; 122:51–65. [PubMed: 4014201]

Yoo HL, Franke WD. Prevalence of cardiovascular disease risk factors in volunteer firefighters. J Occup Environ Med. 2009; 51:958–962. [PubMed: 19620889]

TABLE I

Demographic Characteristics of Interagency Hotshot Crew Members

Variable	All (N =38)	Alpine interagency hotshot crew (n =18)	Pike interagency hotshot crew (n =20)
Age (years)[a]	29 (4.34)	29.9 (5.44)	28.2 (2.97)
Time spent as a firefighter (years)[a]	3.65 (3.38)	4.12 (3.89)	3.25 (2.92)
Male %	100	100	100
White, non-Hispanic n, %	34 (90)	15 (83)	19 (95)
Current smoker n, %	2 (5)	1 (6)	1(5)
Former smoker n, %	10 (27)	2 (11)	9 (45)
Current chewing tobacco (yes vs. no) n, %	16 (42)	7 (39)	9 (45)
Volunteer firefighter n, %	6 (16)	5 (28)	1 (5)
Permanent employee n, %	11 (29)	5 (28)	6 (30)

[a]Mean value (standard deviation).

TABLE II

Clinical Characteristics of Interagency Hotshot Crew Members

Variable	All (N =38)	Alpine interagency hotshot crew (n =18)	Pike interagency hotshot crew (n =20)
Augmentation index % (adjusted for heart rate to 75 BPM)[a]	5 (13)	10 (11)	1.0 (13)[b]
Hypertension (ever) n, %	3 (8)	2 (11)	1 (5)
Elevated cholesterol (ever) n, %	4 (10)	3 (17)	1 (5)
Total cholesterol (mg/dl)[a]	170 (35.8)	175 (40.1)	164 (28.4)
HDL-cholesterol (mg/dl)[a]	55.3 (10.6)	54.6 (11.4)	56.25 (9.81)
LDL-cholesterol (mg/dl)[a]	93.8 (28.1)	101 (32.1)	82.9 (16.5)
Triglycerides (mg/dl)[a]	109 (61.4)	97.6 (39.1)	125.8 (84.0)
HsCRP (mg/L)[a]	1.25 (1.95)	1.23 (2.11)	1.28 (1.78)
Fibrinogen (mg/dl)[a]	270.2 (60.2)	295 (41.2)	221 (64.3)
Log_{10} 8-isoprostane (ng/ml)[a]	−0.14 (0.42)	−0.20 (0.41)	−0.08 (0.42)
Log_{10} 8-OHdG (ng/ml)[a,c]	0.75 (0.36)	0.61 (0.39)	0.88 (0.29)[b]
Oxidative stress score[a,d]	0.41 (0.45)	0.48 (0.45)	0.34 (0.45)
Log_{10} levoglucosan (μg/ml)[a]	1.17 (0.31)	1.14 (0.25)	1.20 (0.37)
Asthma (ever) n, %	7 (18)	6 (33)	1 (5)
Allergies (ever) n, %	17 (45)	11 (61)	6 (30)
Upper respiratory symptom score[a,e]	7.63 (7.1)	7.28 (7.11)	7.95 (7.21)
Lower respiratory symptom score[a,f]	2.34 (3.87)	1.56 (2.52)	3.05 (4.73)
Pulmonary function[a]			
FEV_1%-predicted	103 (10.2)	101 (10.1)	104 (10.5)
FVC%-predicted	107 (13.0)	104 (11.4)	111 (13.7)
FEV_1/FVC	95.6 (9.14)	97.1 (9.80)	94.3 (8.53)

[a] Mean value and standard deviation.

[b] Significantly different from Alpine Interagency Hotshot Crew at *P*-value <0.05.

[c] 8-hydroxy-2′-deoxyguanosine (8-OHdG).

[d] Derived from the average of z-scores for 8-isoprostane and 8-hydroxy-2′-deoxyguanosine(8-OHdG).

[e] Range 0–24; higher score denotes more frequent symptoms.

[f] Range 0–19; higher score denotes more frequent symptoms.

TABLE III

Predictors of Arterial Stiffness (Augmentation Index %), Linear Regression Estimates and 95% CIs

Variable[a]	Unadjusted	Adjusted[b]
Smoking history (ever)	11.2 (2.81, 19.7)	11.5 (3.73, 19.3)
Oxidative stress score[c]	10.2 (1.35, 19.0)	10.5 (2.51, 18.5)

[a] Age, chewing tobacco status, HDL-cholesterol, LDL-cholesterol, triglycerides, high sensitivity CRP, fibrinogen, allergy history, lower respiratory symptom score, upper respiratory symptom score, Interagency Hotshot Crew, FEV_1 %-predicted, FVC %-predicted, and FEV_1/FVC %-predicted were not significantly associated with mean augmentation index % values.

[b] Estimates controlling for all other specified variables in the Table III.

[c] Average z-scores of $\log_{10}$ values of 8-hydroxy-2′-deoxyguanosine (8-OHdG) and 8-isoprostane.

TABLE IV

Predictors of Oxidative DNA Damage and Repair (8-OHdG[a]), Linear Regression Estimates and 95% CIs

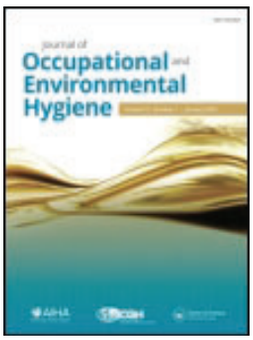
Log_{10} 8-OHdG [a,b]	Unadjusted	Adjusted[c]
Log_{10} levoglucosan[d] concentration	0.45 (0.07, 0.84)	0.41 (0.04, 0.79)
Interagency Hotshot Crew	0.27 (0.05, 0.50)	0.21 (−0.02, 0.45)

[a] 8-hydroxy-2′-deoxyguanosine (8-OHdG).

[b] Smoking/chewing tobacco status, hsCRP, HDL-cholesterol, LDL-C, triglycerides, fibrinogen, asthma history, allergy history, lower respiratory symptom score, upper respiratory symptom score, FEV_1%-predicted FVC %-predicted, and FEV_1/FVC %-predicted, and age were not significantly associated with mean log_{10} 8-hydroxy-2′-deoxyguanosine (8-OHdG) values.

[c] Estimates controlling for all other specified variables in the table.

[d] Levoglucosan (LG).



Taylor & Francis
Taylor & Francis Group

Journal of Occupational and Environmental Hygiene

ISSN: 1545-9624 (Print) 1545-9632 (Online) Journal homepage: https://www.tandfonline.com/loi/uoeh20

Exposures and Cross-shift Lung Function Declines in Wildland Firefighters

Denise M. Gaughan, Chris A. Piacitelli, Bean T. Chen, Brandon F. Law, M. Abbas Virji, Nicole T. Edwards, Paul L. Enright, Diane E. Schwegler-Berry, Stephen S. Leonard, Gregory R. Wagner, Lester Kobzik, Stefanos N. Kales, Michael D. Hughes, David C. Christiani, Paul D. Siegel, Jean M. Cox-Ganser & Mark D. Hoover

To cite this article: Denise M. Gaughan, Chris A. Piacitelli, Bean T. Chen, Brandon F. Law, M.b Ab as Virji, Nicole T. Edwards, Paul L. Enright, Diane E. Schwegler-Berry, Stephen S. Leonard,b Gregory R. Wagner, Lester Kobzik, Stefanos N. Kales, Michael D.bHughes, David C. Christiani,b Paul D. Siegel, Jean M. Cox-Ganser & Mark D. Hoover (2014) Expobures and Cross-shift Lungb Function Declines in Wildland Firefighters, Journal of Occupational and Environmental Hygiene,b 11:9, 591-603, DOI: 10.1080/15459624.2014.895372b

To link to this article: https://doi.org/10.1080/15459624.2014.895372m

Journal of Occupational and Environmental Hygiene, 11: 591–603
ISSN: 1545-9624 print / 1545-9632 online

DOI: 10.1080/15459624.2014.895372

Exposures and Cross-shift Lung Function Declines in Wildland Firefighters

Denise M. Gaughan,[1-3] Chris A. Piacitelli,[2] Bean T. Chen,[4] Brandon F. Law,[4] M. Abbas Virji,[2] Nicole T. Edwards,[2] Paul L. Enright,[5] Diane E. Schwegler-Berry,[4] Stephen S. Leonard,[4] Gregory R. Wagner,[3] Lester Kobzik,[6] Stefanos N. Kales,[3] Michael D. Hughes,[7] David C. Christiani,[3] Paul D. Siegel,[4] Jean M. Cox-Ganser,[2] and Mark D. Hoover[2]

[1]Department of Preventive Medicine and the Institute for Translational Epidemiology, Icahn School of Medicine at Mount Sinai, New York, New York
[2]Division of Respiratory Disease Studies, National Institute for Occupational Safety and Health, Morgantown, West Virginia
[3]Department of Environmental Health (Environmental and Occupational Medicine and Epidemiology Program), Harvard School of Public Health, Boston, Massachusetts
[4]Health Effects Laboratory Division, National Institute for Occupational Safety and Health, Morgantown, West Virginia
[5]Department of Medicine, College of Medicine, University of Arizona, Tucson, Arizona
[6]Department of Environmental Health (Molecular and Integrative Physiological Sciences), Harvard School of Public Health, Boston, Massachusetts
[7]Department of Biostatistics, Harvard School of Public Health, Boston, Massachusetts

Respiratory problems are common among wildland firefighters. However, there are few studies directly linking occupational exposures to respiratory effects in this population. Our objective was to characterize wildland fire fighting occupational exposures and assess their associations with cross-shift changes in lung function. We studied 17 members of the Alpine Interagency Hotshot Crew with environmental sampling and pulmonary function testing during a large wildfire. We characterized particles by examining size distribution and mass concentration, and conducting elemental and morphological analyses. We examined associations between cross-shift lung function change and various analytes, including levoglucosan, an indicator of wood smoke from burning biomass. The levoglucosan component of the wildfire aerosol showed a predominantly bimodal size distribution: a coarse particle mode with a mass median aerodynamic diameter about 12 μm and a fine particle mode with a mass median aerodynamic diameter < 0.5 μm. Levoglucosan was found mainly in the respirable fraction and its concentration was higher for fire line construction operations than for mop-up operations. Larger cross-shift declines in forced expiratory volume in one second were associated with exposure to higher concentrations of respirable levoglucosan ($p < 0.05$). Paired analyses of real-time personal air sampling measurements indicated that higher carbon monoxide (CO) concentrations were correlated with higher particulate concentrations when examined by mean values, but not by individual data points. However, low CO concentrations did not provide reliable assurance of concomitantly low particulate concentrations. We conclude that inhalation of fine smoke particles is associated with acute lung function decline in some wildland firefighters. Based on short-term findings, it appears important to address possible long-term respiratory health issues for wildland firefighters. [Supplementary materials are available for this article. Go to the publisher's online edition of Journal of Occupational and Environmental Hygiene *for the following free supplemental resources: a file containing additional information on historical studies of wildland fire exposures, a file containing the daily-exposure-severity questionnaire completed by wildland firefighter participants at the end of each day, and a file containing additional details of the investigation of correlations between carbon monoxide concentrations and other measured exposure factors in the current study.]*

Keywords occupational lung disease, exposure assessment, levoglucosan

Address correspondence to: Jean Cox-Ganser, Division of Respiratory Disease Studies, National Institute for Occupational Safety and Health, 1095 Willowdale Road, MS 2800, Morgantown, WV 26505; e-mail: Jcoxganser@cdc.gov

INTRODUCTION

According to wildland fire statistics collected by the U.S. Government, the number and intensity of fires has grown

over the past decades with approximately 6 million acres being destroyed in an average North American wildland fire season.[1] The year 2012 involved extremely arid conditions (i.e., the worst drought since 1934) and 9.3 million acres burned in wildfires (http://www.ncdc.noaa.gov/). The growing annual extent and intensity of these fires spurs increasing concern for the respiratory health of wildland firefighters, including the more than 34,000 who are seasonally or permanently employed by the federal government. Studies examining respiratory symptoms, pulmonary function, and inflammatory markers in wildland firefighters have found increases in upper and lower airways inflammation and symptoms, airways hyper-responsiveness, and declines in lung function across a work shift and a season.[2–8]

The chief inhalation hazards associated with wildland fire fighting have been identified as carbon monoxide (CO) and respirable particulate matter (<10 μm).[5,8–13] Pyrolysis of organic components may increase the potency and or toxicity of particulate by producing radical and oxygenated species. Oxygen free radicals are highly reactive and have been implicated in lung injury, including asthma.[14] Measurement of levoglucosan (LG), a sugar anhydride byproduct of incomplete combustion of cellulose, may be used to indicate relative exposure to products of pyrolysis from burning biomass.[15]

While general observational information exists on adverse health effects and exposures associated with wildland fire fighting, studies directly linking respiratory function and exposure characteristics are lacking. Therefore, the objectives of our current study were to characterize exposures of wildland firefighters during a large wildfire, explore correlations between exposures and fire fighting activity, and examine the effects of these exposures on cross-shift lung function changes. Our secondary objective was to support improved selection of sampling methods for future studies.

METHODS

Study Population

The study population consisted of members of the Alpine Interagency Hotshot Crew (IHC) based in Rocky Mountain National Park, Colorado, who fought the Red Eagle Fire from August 7–10, 2006 (Table I). The Red Eagle Fire burned over 34,000 acres in Glacier National Park and on the adjoining Blackfeet Tribal Land in Montana. IHCs are elite crews that primarily conduct fire-line construction using hand tools during the most dangerous phases of wildfire suppression. Fire line construction involves clearing vegetation and exposing bare soil by cutting, scraping, or digging to create a break in fuel. IHCs also engage in mop-up operations, which involve extinguishing or removing burning material along or near a fire line after the fire has been controlled. Each crew comprises 20 members. Jobs include: "Lead workers" who oversee all crew operations; "Sawyers" who are the chainsaw operators who clear the way for fire line construction; "Swampers" who shadow the Sawyers, removing the fallen debris; and "Line workers" who construct the fire line.

TABLE I. Characteristics of Interagency Hotshot Crew (IHC) Study Participants

Variable	Study Participants n = 17
Median age, years	26 (23, 35)[A]
Median time spent as member of IHC, months	1 (1, 12)[A]
Male n,%	16, 94%
White, non-Hispanic n,%	16, 94%
Never smoker n,%	15, 88%
Former smoker n,%	2, 12%
Asthma, physician-diagnosed (current) n,%	1, 6%
Allergies (ever) n,%	9, 53%
Spirometry Results[B]	
• Median FEV_1% predicted	104 (86, 115)[A]
• Median FVC% predicted	104 (91, 125)[A]
• Median FEV_1/FVC	0.80 (0.73, 0.87)[A]

[A](10^{th}, 90^{th} percentiles).
[B]Spirometry values obtained at first pre-shift assessment.

The study protocol was approved by the NIOSH Human Subjects Review Board and informed consent was obtained from each research participant.

Medical Survey

Demographic and clinical variables for the crew members, as well as baseline spirometry measurements, were obtained during a preseason assessment conducted several months before the Red Eagle Fire; those results have been reported.[3]

Exhaled Breath Carbon Monoxide

Exhaled breath CO was measured twice daily—approximately one hour before the work shift began and one hour after the shift concluded—on each participant using a breath CO monitor according to the manufacturer's instructions (Micro 4 Smokerlyzer, Bedfont Scientific, Medford, N.J.). The elapsed time between the end of shift and the measurement of exhaled breath CO was recorded for each subject. The elapsed time provided for washout of residual inhaled CO from the lung volume. The medical monitoring location was situated at the base camp, at a location away from cooking, parking, and portable generator areas to avoid interference from local sources of CO. Ambient CO concentration was measured at the base camp where the pre-/post-shift exhaled breath measurements were obtained.

Spirometry

Spirometry was conducted twice daily, immediately following exhaled breath CO measurement, on each participant. Technicians who had completed a NIOSH-approved spirometry course followed American Thoracic Society (ATS) guidelines[16] using an ultrasonic flow spirometer (EasyOne

Diagnostic Spirometry System 2001, ndd Medical Technologies, Zurich, Switzerland).

We used equations for predicted values and lower limits of normal derived from the Third National Health and Nutrition Examination Survey (NHANES III) data.[17] We defined an individual cross-shift forced expiratory volume in 1 sec (FEV_1) decline of 10% or greater as significant;[18] we also examined declines of 5% or greater. We followed ATS procedure by inquiring about current medications, but we did not ask participants to abstain from using their medications, given safety concerns.

Exposure Questionnaire

Participants were asked daily, at the shift's conclusion, what job duties they had performed and to rate the severity (none, mild, moderate, or severe) and duration of his or her exposure to smoke and his or her exposure to dust during the preceding shift. Because use of the qualitative terms "none," "mild," "moderate," and "severe" is subjective, our instructions to the subjects were that they should base their responses on their individual perception of the smoke and dust conditions.

Exposure Assessment

Details on each environmental sampling method and associated instrumentation used in the assessment of exposure are provided in Table II. For each of the four days, each crew member agreed to wear a real-time (RT) personal breathing zone CO monitor (Industrial Scientific Corporation, Oakdale, Pa.) and one of the following samplers: a filter cassette with a 10-mm nylon respirable cyclone to measure respirable particulate concentration and either respirable LG concentration or respirable crystalline silica concentration; a personal cascade impactor to measure particle size distribution, total concentrations of particulates and LG, and respirable concentrations of particulates and LG; or a *personal*DataRAM (*p*DR) monitor (Thermo Scientific Corporation, Franklin, Mass.) to measure RT particulate in the size range of 0.1 to 10 μm.

The CO monitor was calibrated in the field using a certified canister of 100 ppm CO. The *personal*DataRAM was factory-calibrated annually, which provided an initial qualitative measure for use in side-by-side sampling with a filter/cyclone combination in the field. Because *personal*DataRAM response is dependent on the size distribution and refractive index of the particles sampled, sampling response to field aerosols are likely to be different from the response to calibration aerosols used at the factory. Samplers were worn for the duration of the work shift.

Arrays of the above-identified instrumentation, along with a closed-face cassette for total particulate sampling and a filter cassette with a 10-mm nylon respirable cyclone to collect samples for EC/OC analysis, were positioned 1.3 m above the ground in sampling "baskets" adjacent to the fire line and at the base camp (located approximately five miles from burn activity) to collect area samples for the duration of the work shift. Each basket also included an open-face cassette that was activated for a 1-hour period during the shift to collect particles for elemental analyses by energy dispersive x-ray analysis (EDX) and for morphological analyses by scanning electron microscopy (SEM). Closed-face versions of the filter cassettes were selected for collection of the total particulate samples to minimize unwanted collection of inertially dispersed debris such as chainsaw cuttings; open-face versions of the filter cassettes were selected for the shorter-term collection of a more uniformly distributed array of particles on the filter for microscopic examination.

Sample analyses for LG and for elemental particle composition and morphology were performed at the National Institute for Occupational Safety and Health (NIOSH); all other analyses were performed at a contract laboratory accredited by the American Industrial Hygiene Association (AIHA®).

Statistical Methods

Descriptive statistics were calculated for demographic and clinical variables, as well as for self-reported smoke exposures over the shift immediately preceding the medical assessment. Crew members' breathing zones were sampled, and their results were analyzed according to job (Lead worker, Line worker, Sawyer, or Swamper) and operation (fire line construction or mop-up).

The airborne concentration and aerodynamic size distribution of particulate mass were determined for each personal cascade impactor sample. Composite values of mass concentration as a function of particle size were calculated as averages for each stage across all samples. The concentration of the respirable fraction of particulate mass was calculated using standard cascade impactor correction factors.[19] The airborne concentration and aerodynamic size distribution of LG were determined from the personal cascade impactor samples. LG respirable concentrations were also determined from the respirable cyclone samples.

The geometric mean (GM) concentration and geometric standard deviation (GSD) of the concentration distribution were calculated for each analyte. Concentration values below the limit of detection (LOD) were treated using the maximum likelihood estimation method, consistent with conventional practice following a review of the data's distribution.[20] One-way and multifactor analysis of variance techniques were used to compare mean values of log-transformed analyte concentrations by job, operation, and location. Spearman correlation coefficients were calculated to examine associations between paired RT CO and RT particulate concentrations (for both time-weighted average (TWA) and individual 1-min interval values). Spearman correlations were also examined for associations between TWA CO and the following analytes: TWA total particulate concentrations, TWA respirable particulate concentrations (for both impactor and cyclone samples), LG (total and respirable), and organic carbon.

Mean cross-shift changes in FEV_1 (ΔFEV_1) and in exhaled breath CO (ΔCO) were investigated using paired difference

TABLE II. Environmental Sampling Methods

Analyte	Analytical Method	Sample Type: Area (A) and/or Personal (P)	Sampling Device and Media	Flow Rate (lpm)
Particle size distribution	Gravimetric analysis by NIOSH Method 0500	A, P	Marple 8-stage Personal Cascade Impactor, Model 298 (Thermo Scientific Corporation, Franklin, Mass.) with 34-mm diameter radial-cut polyvinyl chloride (PVC) substrates (first stage greased); stage cut points of 21.3, 14.8, 6.0, 3.5, 1.6, 0.93, and 0.52 μm; followed by a 5-μm-pore-size PVC final filter	2.0
Particle elemental and morphological analyses	Energy dispersive x-ray spectrometry (EDX) and scanning electron microscopy (SEM) for particle profiles; filter carbon tape-mounted on aluminum stub and sputter-coated with gold/palladium (SPI Supplies, West Chester, Pa.); EDX conducted with IMIX Microanalysis Imaging System (Princeton Gamma-Tech, Princeton, N.J.); SEM analyses conducted with model JEM6400 (JEOL, Tokyo, Japan)	A	Open-face cassette with 37-mm diameter, 0.8-μm pore size polycarbonate filter	2.0
Total particulate	Gravimetric analysis by NIOSH Method 0500	A	Closed-face cassette with 37-mm diameter, 5-μm pore size PVC filter	2.0
Respirable particulate	Gravimetric analysis by NIOSH Method 0600	A, P	10-mm nylon respirable dust cyclone followed by 37-mm diameter, 5-μm pore size PVC filter	1.7
Real-time particulate (0.1 to 10 μm)	Passive flow direct-reading instrument	A, P	*personal*DataRAM *p*DR-1000-AN monitor (Thermo Scientific Corporation, Franklin, Mass.) at 1-min averaging periods	N/A
Real-time carbon monoxide	Passive flow direct-reading instrument	A, P	T82^{TM} Single-Gas Monitor (Industrial Scientific Corporation, Oakdale, PA) at 1-min averaging period	N/A
Levoglucosan	Levoglucosan extracted from filter using methanol and extract evaporated to dryness under nitrogen; each sample and standard derivatized using Tri-Sil reagent (Thermo Scientific, Rockford, Ill.), and then quantified by gas chromatography-mass spectrometry	P	Samples from personal cascade impactors were sent for this analysis after gravimetric analysis	2.0
		A, P	Subset of respirable dust cyclone samples were sent for this analysis after gravimetric analysis	1.7
Crystalline silica	X-ray diffraction by NIOSH Method 7500	A, P	Subset of respirable dust cyclone samples were sent for this analysis after gravimetric analysis	1.7
Elemental carbon/ organic carbon	Thermal-optical analysis and flame ionization detection by NIOSH Method 5040	A	10-mm nylon respirable dust cyclone followed by 37-mm diameter binder-free quartz fiber filter	1.7

Note: N/A indicates that an active flow rate value is not applicable to the passive flow devices.

TABLE III. Personal Concentration Results by Job

Analyte[A]	Job	N	GM	GSD	MIN	MAX
Particulate, Total-Impactor (mg/m^3)	Lead	1	1.16	N/A[B]	N/A	N/A
	Line	9	1.99	1.51	0.92	3.12
	Sawyer	3	2.80	1.55	1.71	3.93
	Swamper	3	2.27	1.28	1.78	2.93
Particulate, Respirable-Impactor (mg/m^3)	Lead	1	0.88	N/A	N/A	N/A
	Line	9	0.35	1.58	0.20	0.72
	Sawyer	3	0.41	1.87	0.22	0.78
	Swamper	3	0.40	1.23	0.33	0.49
Particulate, Respirable-Cyclone (mg/m^3)	Lead	2	0.14	1.33	0.11	0.17
	Line	13	0.33	2.49	0.13	2.18
	Sawyer	7	0.52	1.96	0.27	2.07
	Swamper	7	0.69	1.59	0.27	1.03
Particulate-Real-time (mg/m^3)[C]	Line	10	0.60	1.60	0.29	1.49
	Sawyer	2	1.68	1.48	1.27	2.22
	Swamper	1	1.40	N/A	N/A	N/A
Carbon monoxide (ppm)	Lead	7	0.58	1.23	0.50	0.83
	Line	36	0.67	1.52	0.50	2.83
	Sawyer	12	8.19	1.64	4.15	16.5
	Swamper	12	6.24	1.71	2.51	14.6
Levoglucosan, Total-Impactor (μg/m^3)	Lead	1	0.65	N/A	N/A	N/A
	Line	8	3.04	2.41	1.43	20.4
	Sawyer	3	1.31	2.70	0.61	4.02
	Swamper	3	2.66	3.06	0.75	6.20
Levoglucosan, Respirable-Impactor (μg/m^3)	Lead	1	0.38	N/A	N/A	N/A
	Line	8	1.48	3.66	0.22	18.8
	Sawyer	3	0.50	3.48	0.22	2.12
	Swamper	3	0.92	2.83	0.30	2.42
Levoglucosan, Respirable-Cyclone (μg/m^3)	Lead	1	0.22	N/A	N/A	N/A
	Line	8	1.25	4.35	0.36	21.6
	Sawyer	2	0.21	1.01	0.21	0.21
	Swamper	3	3.24	3.73	0.71	7.08

Note: GM = geometric mean.
GSD = geometric standard deviation.
[A]Results for crystalline silica were below the minimum quantifiable concentration of 0.025 mg/m^3 and are not tabulated.
[B]N/A indicates not applicable for single value.
[C]No RT measurement obtained for lead worker.

Student t-tests. We used the MIXED procedure for repeated measures with a first-order autoregressive correlation structure (SAS software version 9.1, SAS Institute Inc., Cary, N.C.) to examine univariate and multivariate associations between individual ΔFEV_1 and several predictor variables, some of which were categorized by distributional tertiles (including total particulate concentration, respirable particulate concentration as measured by impactor, respirable particulate concentration as measured by cyclone, total LG concentration, respirable LG concentration as measured by impactor, and peak dust exposure). Similar univariate and multivariate associations were examined between exhaled breath ΔCO and the above-noted predictor variables.

Respirable LG concentration, as measured by filtration following a 10-mm respirable cyclone, and RT TWA particulate concentration each had distinct bimodal distributions and were categorized accordingly. Specifically, participants with values of respirable LG concentration exceeding 1 μg/m^3 were classified as having high LG exposure. Similarly, participants with values of RT TWA particulate concentration exceeding 1 mg/m^3 were classified as having high RT TWA particulate exposure. High peak RT particulate concentration (defined as one or more peaks greater than 10 mg/m^3) was also analyzed. We additionally examined age, a history of allergies, and pre-season FEV_1. Heterogeneities in the cohort were insufficient to support analyses of relationships involving race/ethnicity, gender, and smoking.

For individuals with more than one measurement for a specific analyte, categorization was based on the average of the measured values. For all analyses, we considered two-sided

TABLE IV. Personal Concentration Results by Crew Operation

	Crew Operation[A]	N	GM	GSD	MIN	MAX
Particulate, Total-Impactor (mg/m^3)	Fire line construction	8	2.28	1.43	1.16	3.93
	Mop-up	3	1.86	1.88	0.92	3.12
Particulate, Respirable-Impactor (mg/m^3)	Fire line construction	8	0.45	1.62	0.24	0.88
	Mop-up	3	0.38	1.92	0.20	0.72
Particulate, Respirable-Cyclone (mg/m^3)	Fire line construction	14	0.49	2.59	0.11	2.18
	Mop-up	8	0.51	1.96	0.17	1.14
Particulate-Real-time (mg/m^3)	Fire line construction	7	1.04	1.93	0.29	2.22
	Mop-up	5	0.51	1.21	0.42	0.68
Carbon monoxide (ppm)	Fire line construction	34	1.93	3.84	0.50	16.5
	Mop-up	16	1.24	3.06	0.50	6.64
Levoglucosan, Total-Impactor (μg/m^3)	Fire line construction	8	3.64	2.67	0.65	20.4
	Mop-up	2	1.13	1.79	0.75	1.70
Levoglucosan, Respirable-Impactor (μg/m^3)	Fire line construction	8	1.88	3.07	0.38	18.8
	Mop-up	2	0.26	1.25	0.22	0.31
Levoglucosan, Respirable-Cyclone (μg/m^3)	Fire line construction	7	2.59	5.20	0.36	21.6
	Mop-up	4	0.28	1.75	0.21	0.64

Note: GM = geometric mean; GSD = geometric standard deviation.
[A]Decreased sample numbers attributable to exclusion of day where crew split its time between fire line construction and mop-up.

$p < 0.05$ as indicating statistical significance and two-sided $p > 0.05$ but $p < 0.10$ as indicating borderline statistical significance.

RESULTS

Of the 20 members of the Alpine IHC, 18 were on active assignment with the crew during the time of the Red Eagle Fire. All 18 participated in the environmental measurement portion of the study and 17 of them (94%) participated in the medical portion of the study (Table I). Participants had a median age of 26 years, and were primarily Caucasian, non-Hispanic males (94%). Approximately 12% were former smokers and about 88% had never smoked. Nearly 53% reported having allergies and one participant reported current physician-diagnosed asthma. Median pulmonary function values obtained at the first pre-shift assessment were about 104% of predicted.

TABLE V. Area Concentration Results by Location

Analyte[A,B]	Location	N	GM	GSD	MIN	MAX
Particulate, Total-Impactor (mg/m^3)	Base camp	2	0.27	1.27	0.22	0.31
	Fire line	5	0.66	1.82	0.41	1.85
Particulate Total-Cassette (mg/m^3)	Base camp	2	0.14	1.01	0.14	0.14
	Fire line	5	0.53	2.16	0.19	1.48
Particulate, Respirable-Impactor (mg/m^3)	Base camp	2	0.13	1.15	0.12	0.14
	Fire line	5	0.35	2.23	0.16	1.29
Particulate, Respirable-Cyclone (mg/m^3)	Base camp	4	0.09	1.50	0.06	0.15
	Fire line	10	0.27	2.98	0.03	0.99
Particulate-Real-time (mg/m^3)	Base camp	2	0.38	1.10	0.35	0.41
	Fire line	3	0.59	2.77	0.19	1.31
Carbon monoxide (ppm)	Base camp	2	1.16	1.16	1.08	1.23
	Fire line	4	2.72	1.90	1.30	6.18
Levoglucosan, Respirable-Cyclone (μg/m^3)	Base camp	2	0.39	1.13	0.36	0.43
	Fire line	5	4.99	7.19	0.27	32.9
Organic Carbon, Respirable-Cyclone (mg/m^3)	Base camp	2	0.07	1.02	0.07	0.07
	Fire line	5	0.24	2.27	0.07	0.71

Note: GM = geometric mean; GSD = geometric standard deviation.
[A]Results for elemental carbon are not tabulated because only one value from a cyclone sample taken at the fire line was at the minimum detectable concentration (MDC) of 0.01 ug/m^3, and all other values were below that concentration.
[B]Results for crystalline silica were below the MDC of 0.005 mg/m^3 and are not tabulated.

Exposure Assessment

A total of 125 personal and 53 area air samples were collected during fire line construction (two full days and one half day) and mop-up operations (one full day and one half day). The average duration of the work shift was 12 hr. GM and GSD analyte concentrations from personal samplers are listed by job and crew operation in Tables III and IV, respectively. However, concentration means for the day when the crew split its time between fire line construction and mop-up operations are not reported in Table IV. GMs and GSD analyte concentrations from area samplers are reported by location in Table V.

Particle Size Distribution

Results of particle size distributions assessed using data from 13 personal impactor samples are illustrated in Figure 1. Figure 1A shows the mass-based distribution as a function of particle size. Wildfire smoke particles were present in all size ranges from smaller than 0.52 μm to greater than 21.3 μm. Note that airborne particle mass is mostly associated with larger particles, while the levoglucosan component of the wildfire smoke aerosol showed a primarily bimodal size distribution: a coarse particle mode with an MMAD greater than 15 μm and a fine particle mode with an MMAD less than 0.5 μm. When examined by percent mass, nearly half the particulate collected (44%) was found on the first stage of the impactor (in the very coarse range with effective cutoff diameter of 21.3 μm). The second largest percent mass collected (13%) was found on the backup filter of the impactor (in the ultrafine range with cutoff diameter < 0.52 μm). Figure 1B shows the distribution of airborne LG by particle size fractions. The largest mass fraction was found on the first stage of the impactor making up 27% of the total mass collected. However, nearly two-thirds of the levoglucosan collected (71%) was found in the respirable range.

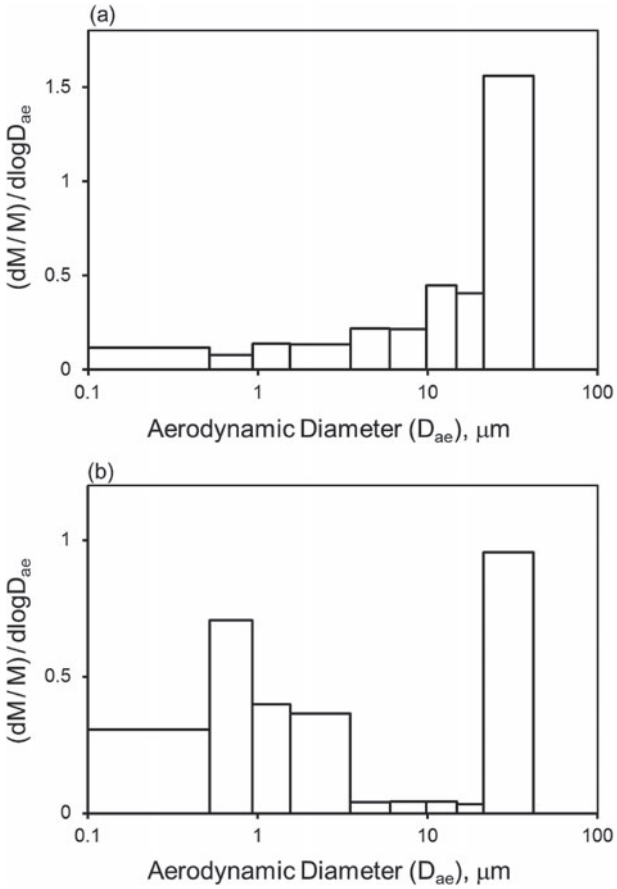


FIGURE 1. Normalized personal cascade impactor particle size distributions for (a) airborne particle mass and (b) airborne levoglucosan. Note that airborne particle mass is mostly associated with larger particles, while airborne levoglucosan is bimodally distributed.

Elemental and Morphological Analyses

The particulate profiles based on EDX and SEM techniques created from seven filter cassette samples obtained at the base camp and fire line revealed three distinctive types of particles: a crystal-like particle comprising mainly titanium, iron, and aluminum silicate (Figure 2A) (other similarly configured particles also showed lead, calcium, and/or magnesium); a spherical, tar-like particle containing mainly carbon (Figure 2B); and particles with an aggregate configuration of many tar-like particles (Figure 2C). There was no observed difference in the composition or type in the samples by location.

Total and Respirable Particulate Concentrations

Assessment of personal particulate concentration using data from 16 impactor samples and 29 cyclone samples showed that GMs for total particulate concentration did not vary significantly by job or crew operation. The GM for the respirable particulate portion of the impactors for all operations was 0.39 mg/m^3 (18% of total particulate). Although the only impactor measurement of total particulate exposure to Lead workers (who are responsible for supervision) appeared lower than exposures to the workers who are engaged in direct cutting, digging, and other fire line construction activities, the respirable fraction of that Lead worker's exposure measured by impactor was higher (0.88 mg/m^3) than those of the Line workers (0.35 mg/m^3), Sawyers (0.41 mg/m^3), and Swampers (0.40 mg/m^3), and, overall, the respirable particulate concentrations from the impactors did not vary significantly by job or crew operation.

Respirable particulates as measured by cyclone, however, were found to vary by job, ranging from a GM of 0.14 mg/m^3 for Lead workers, to a GM of 0.33 mg/m^3 for Line workers, to a GM of 0.69 mg/m^3 for Swampers. The influence of work type (e.g., Lead workers being involved in supervision versus other workers being directly involved in fire line construction) was reflected in the significantly higher respirable particulate concentrations that were observed for Swampers compared to Lead workers (GM = 0.14 mg/m^3) ($p < 0.05$) and Line workers (GM = 0.33 mg/m^3) ($p < 0.05$). Respirable particulate concentrations were also significantly higher for Sawyers (GM = 0.52 mg/m^3) than for Lead workers ($p < 0.05$).

Respirable particulate concentration as measured by cyclone did not vary significantly by crew operation.

Area respirable particulate concentration, as assessed using data from 14 cyclone samples, did not differ by location. The value of one sample obtained at a sampling rate of 1.7 L/min for 344 min during a digging operation was below the minimum detectable concentration (MDC) of 0.05 mg/m^3, based on an analytical LOD of 30 μg per sample. Total particulate concentration, as measured by cassette samples, was higher at the fire line (GM = 0.53 mg/m^3) than at base camp (GM = 0.14 mg/m^3). However, this difference was only marginally significant (p = 0.07).

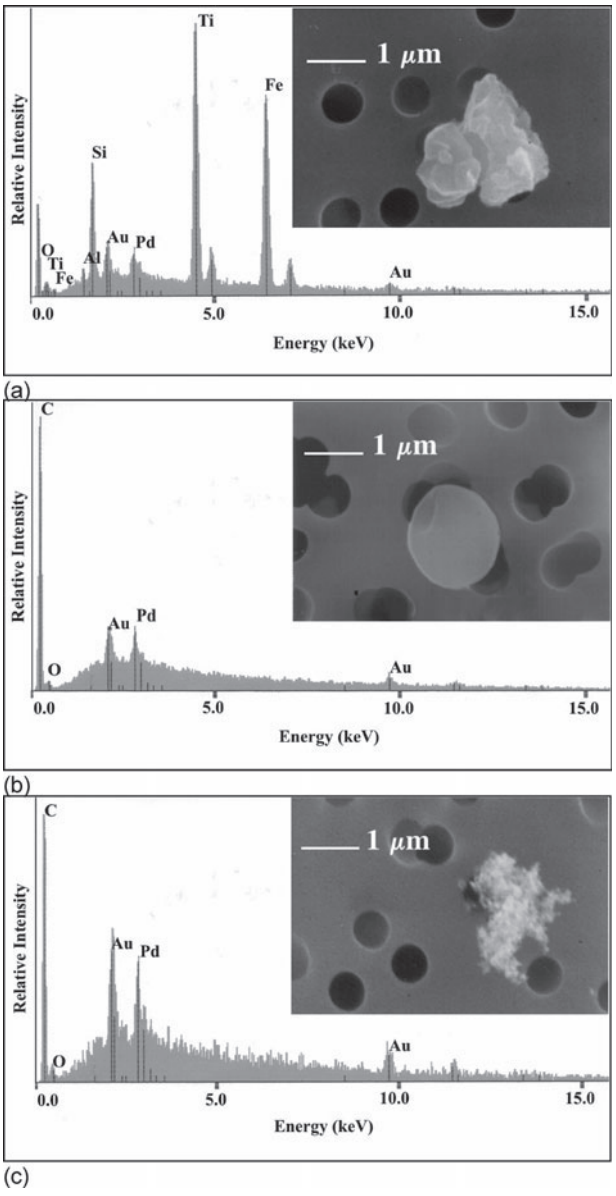


FIGURE 2. Elemental Particle Profile and Electron Micrograph of Particles: (a) samples obtained at base camp indicate a crystal-like particle comprised mostly of titanium, aluminum silicate, and iron; (b) samples obtained at the fire line revealed a spherical, tar-like particle containing mainly carbon; and (c) and other samples obtained at the fire line appear to have an aggregate configuration of many tar-like particles. The gold (Au) and palladium (Pd) peaks are from the sputter coating on the samples and are not constituents of the particles. The active diameter of EDX interrogation was approximately 1 μm and particle composition did not appear to vary with beam location on particle surface.

Real-time Particulate Concentrations

There were 7908 1-min interval data points from 13 personal RT particulate samplers. RT particulate concentration varied by crew operation. The TWA RT particulate concentration during fire line construction (GM = 1.04 mg/m^3) was significantly higher than during mop-up operations (GM = 0.51 mg/m^3) ($p < 0.05$). RT particulate concentration also differed by job, being significantly higher for Sawyers (GM = 1.7 mg/m^3) than for Line workers (GM = 0.60 mg/m^3) ($p < 0.05$). There were 2,431 1-min interval data points from five area RT particulate samplers. RT particulate concentration did not vary by location.

Personal particulate concentrations exceeded 1 mg/m^3 1707 times (i.e., during 21% of all measured intervals) (n = 10 participants); 5 mg/m^3 211 times (2.6%) (n = 10 participants), and 10 mg/m^3 62 times (< 1%) (n = 6 participants). Area particulate concentrations exceeded 1 mg/m^3 434 times (25%) (5 samples), 5 mg/m^3 23 times (1.1%) (1 sample), and 10 mg/m^3 6 times (< 1%) (1 sample).

Carbon Monoxide Concentrations

There were 45,035 1-min interval data points from 67 personal CO samples. Mean values differed by job. The CO concentrations for Swampers (GM = 6.24 parts per million (ppm)) and Sawyers (GM = 8.19 ppm) were significantly higher than those for Line workers (GM = 0.67 ppm) ($p < 0.001$) or Lead workers (GM = 0.58 ppm) ($p < 0.001$). Mean CO concentrations did not differ significantly by crew operation.

There were 3330 1-min interval data points from six area CO samplers. Mean values did not differ significantly by location. TWA CO values never exceeded the NIOSH recommended exposure limit (REL) of 35 ppm or the Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) of 25 ppm. Nine personal samples, including eight Sawyers or Swampers, exceeded the NIOSH ceiling exposure limit of 200 ppm from one to four times during his or her shift.

Levoglucosan (LG) Concentrations

Results of LG concentration analyzed with data from 29 personal samples (15 samples by cascade impactor and 14 samples by cyclone) showed no differences by job or crew operation for personal total LG concentration. Respirable LG concentrations as measured by impactor differed by crew operation, being significantly higher for fire line construction (GM = 1.88 μg/m^3) than for mop-up (0.26 μg/m^3) ($p < 0.05$). Values from three cyclones were below the MDC of 0.4 μg/m^3

for 8-hr samples collected at 1.7 L/min based on an analytical LOD of 0.3 μg LG per sample.

The GM concentration of LG in the respirable particulate as measured by cyclones was 1.05 μg /m^3. Similar to the estimates from the respirable particulate portion of the impactor samples, concentrations obtained from the cyclone samples differed by crew operation; concentrations for fire line construction (GM = 2.59 μg/m^3) being significantly higher than those for mop-up (GM = 0.28 μg/m^3) ($p < 0.05$). Mean LG concentrations as measured by cyclone, also differed by job; concentrations for Swampers (GM = 3.24 μg/m^3) being significantly higher than for Sawyers (0.21 μg/m^3) ($p < 0.05$).

Analyses of respirable LG concentrations obtained from seven area cyclone samples showed that median respirable LG concentration appeared much higher in samples obtained from the fire line (GM = 4.99 μg/m^3, GSD = 7.19 μg/m^3) than base camp (GM = 0.39 μg/m^3, GSD = 1.13 μg/m^3). However, this difference was not statistically significant. The value of one cyclone sample obtained from the fire line area at a sampling rate of 1.7 L/min for 344 min was below the MDC of 0.54 μg/m^3 for LG, based on an analytical LOD of 0.3 μg per sample.

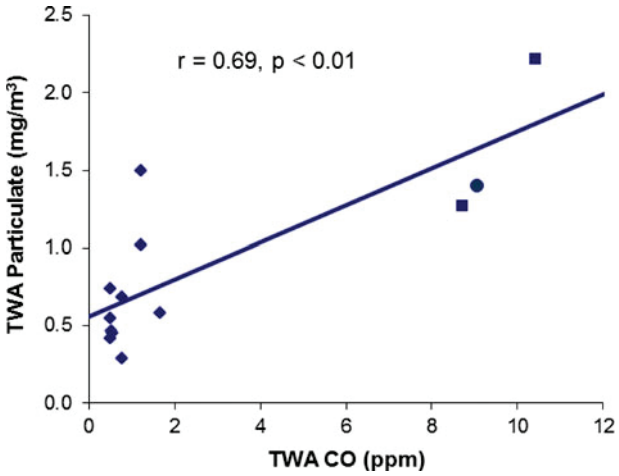


FIGURE 3. Correlation between paired real-time full-shift time-weighted average concentrations of CO and particulate (0.1 to 10 μm, as measured by personalDataRAM), stratified by job. Line worker (♦) (eight paired observations); Sawyer (■) (two paired observations); and Swamper (•) (one paired observation). (color figure available online)

Crystalline Silica Concentrations

Analyses of concentrations of respirable crystalline silica obtained from 15 personal and 7 area samples showed that neither tridymite nor cristobalite was detected in any samples. None of the area samples and only three of the 15 personal samples (14%) were found to have quartz concentrations above the MDC of 0.005 mg/m^3 for an 8-hr sample collected at 1.7 L/min based on an analytical LOD of 4 μg per sample. None of the detected values exceeded the minimum quantifiable concentration of 0.025 mg/m^3 for an 8-hr sample. Thus, no concentrations exceeded either the American Conference of Governmental Industrial Hygienists (ACGIH®) threshold limit value (TLV) of 0.025 mg/m^3 or the NIOSH REL of 0.05 mg/m^3 for crystalline silica.

Elemental Carbon/Organic Carbon Concentrations

GM concentration of organic carbon obtained from seven area samples was 0.17 mg/m^3. Although the geometric mean concentration of respirable airborne organic carbon measure at the fire line location (0.24 mg/m^3) was greater than the geometric mean concentration measured at the base camp (0.07 mg/m^3), the difference by location was not significant. All but one of the seven values for elemental carbon were below the minimal detectable concentration (MDC) of 0.01 mg/m^3 for an 8-hr sample collected at 1.7 L/min based on an analytical LOD of 6 μg elemental carbon per sample; the detectable sample value was at the MDC.

Investigation of Correlations Between CO Concentrations and Other Measured Exposure Factors

Paired analyses of RT personal air sampling measurements indicated that higher TWA CO concentrations were correlated with higher particulate concentrations when examined by mean values. However, this was not the case when values for each analyte were low (Figure 3). Moreover, we did not observe a similar relationship between CO and particulate concentrations when examined by individual data points. In multifactor analysis, TWA CO was associated with job ($p < 0.001$) and crew activity ($p < 0.01$). Specifically, Sawyers had the highest TWA CO (regression estimate: 9.0, 95% CI: 7.34–10.6 ppm), followed by Swampers (regression estimate: 5.90, 95% CI: 4.59–7.20 ppm), and Line workers (regression estimate: −0.00, 95% CI:–0.66, 0.65 ppm). Digging line produced the highest mean TWA CO concentrations (regression estimate: 6.09, 95% CI: 5.17, 7.00 ppm), followed by mop-up (regression estimate: 4.84, 95% CI: 3.57, 6.12). Additional information about investigated correlations is provided in the supplemental material section.

Exposure Questionnaire Responses

A total of 68 questionnaire responses regarding the severity of smoke exposure and the severity of dust exposure during the preceding shift were obtained from the 17 participants over the course of four days. Regarding smoke exposure, day-one responses comprised two "no" smoke exposures, 14 "mild" exposures, and one "moderate" exposure; day-two responses comprised nine "no smoke" exposures, seven "mild" exposures, and one "moderate" exposure; day-three responses comprised three "no" smoke exposures and 14 "mild" exposures; and on day four, all 17 participants reported "no" smoke exposure. No "severe" smoke exposures were reported for any of the study days.

Regarding dust exposure, day-one responses comprised one "no" dust exposure, 10 "mild" exposures, and six "moderate" exposures; day-two responses comprised two "no" dust exposures, 10 "mild" exposures, and five "moderate" exposures;

day three responses comprised one "no" dust exposure, 12 "mild" exposures, and four "moderate" exposures; and on day four there were two responses of "no" dust exposure, five responses of "mild" dust exposure, and 10 responses of "moderate" dust exposure. No "severe" dust exposures were reported at any time.

Based on questionnaire responses, the median time spent in smoke during the preceding shift was 25 minutes min (range 0, 10 hr) and the median time spent in dust during the preceding shift was four hours (range 0, 10).

Medical Survey

Exhaled Breath Carbon Monoxide

The pre-shift exhaled breath CO level was 1.8 ppm and the mean post-shift level was significantly higher at 3.2 ppm ($p < 0.001$). Paired mean post-shift exhaled breath and personal TWA CO concentrations from the same shift and crew members were moderately correlated within firefighters ($r = 0.54$) ($p < 0.001$). In univariate analysis, exhaled breath ΔCO was significantly associated with TWA CO concentrations (regression estimate: 0.31, 95% CI: 0.10, 0.50). However, this association ceased to be significant after adjusting for job and crew activity ($p < 0.10$). No other analytes were found to be associated with exhaled breath CO.

Spirometry

A total of 67 sets of individually paired pre-shift and post-shift spirometry tests were available for analysis. One individual declined to participate in one post-shift session. The mean ΔFEV_1 was a 0.045 L decline ($p = 0.08$). Cross-shift spirometry results were previously reported.[3]

One participant experienced a 16% ΔFEV_1 decline over the first day of testing. His FEV_1 returned to his baseline value which was within the normal range on the evening of the second day of testing, but he experienced a greater than 9% ΔFEV_1 decline over the third day of testing. Five other participants, two of whom were Swampers, experienced a ΔFEV_1 decline between 5% and 10% at least once during the observation period. One participant experienced declines of this magnitude on three separate days, another on two separate days, and three on only one day during the study period. These six individuals ranged in age from 24 to 36 years (mean age 31), one had a history of asthma, and most (5/6) reported a history of allergies. They reported mild or no smoke exposure over the preceding shift on the days that they experienced the ΔFEV_1 declines. However, as we note in the Discussion section this subjective and qualitative description may underestimate the true exposure these individuals experienced.

The mean cross-shift ΔFEV_1 was a decline of 0.05 L ($p = 0.08$). However, univariate analysis by distributional tertiles of age revealed an age-related trend in this change ($p < 0.01$), with mean ΔFEV_1 values for participants ages 24 and younger, ages 25 to 29, and ages 30 and older, respectively, being an increase of 0.02 L, a decline of 0.08 L, and a decline of 0.10 L. Also in univariate analysis, ΔFEV_1 decline was associated with higher concentrations of respirable LG, as measured by cyclone. There were 14 spirometry measurements that had corresponding LG concentration measurements by cyclone. Participants in the high LG exposure group had a mean ΔFEV_1 decline of 0.23 L compared to a mean decline of 0.02 L in the low LG exposure group. Larger mean ΔFEV_1 values were also observed for Swampers (0.19 L decrease) compared to sawyers (0.03 L increase) and Line workers (0.03 L decrease). These associations remained significant in multifactor analyses for LG after adjusting for age, job, and crew operation ($p < 0.05$). Unadjusted and adjusted results can be found in Table VI. Finally, larger mean values of pre-shift exhaled breath CO were associated with larger declines in FEV_1. Specifically, for every 1 unit increase in pre-shift exhaled breath CO, ΔFEV_1 value increased by 0.03 L ($p < 0.05$). This association remained significant in multifactor analyses after adjusting for smoking status, job, and crew operation. No other analytes were significantly associated with change in FEV_1.

Analyses of correlations with respirable dust are documented in the supplemental table. For total dust, the p-value was 0.77, and there were not enough observations to examine associations between peak dust and cross-shift lung function. Similarly, no other predictors, including exhaled breath ΔCO, TWA CO, time spent working on an IHC, or atopy, were significantly associated with change in FEV_1.

TABLE VI. Cross-shift Decline in FEV_1 (mean estimate in liters followed by 95% CI) Based on Univariate and Multivariate Analyses of Levoglucosan Exposure, Job, and Crew Operation

Variable	Unadjusted decline in FEV_1 (L)	Adjusted[A] decline in FEV_1 (L)
Levoglucosan[B]		
• High concentration	**0.23 (0.02, 0.44)**	**0.25 (0.02, 0.48)**
• Low concentration	0.02 (−0.10, 0.09)	0.05 (−0.05, 0.16)
Job		
• Line	0.03 (−0.04, 0.11)	0.06 (−0.03, 0.15)
• Sawyer	−0.03 (−0.22, 0.16)	0.03 (−0.17, 0.24)
• Swamper	**0.19 (0.04, 0.34)**	0.12 (−0.01, 0.30)
Crew operation		
• Fire line construction	0.00 (−0.09, 0.09)	0.01 (−0.10, 0.11)
• Mop-up	0.11 (−0.02, 0.23)	0.12 (−0.02, 0.26)
• Both[C]	0.10 (−0.02, 0.21)	1.0 (−0.02, 0.21)

Notes: Positive values indicate a cross-shift decline in mean FEV_1 value; bold values indicate significance at $p < 0.05$; negative estimates indicate a cross-shift increase in mean FEV_1 value.

[A]Age-adjusted estimates controlling for all other specified variables.

[B]As measured by cyclone

[C]Crew operations during the shift involved both fire line construction and mop-up.

DISCUSSION

We found evidence that larger declines in ΔFEV_1 were significantly associated with exposure to higher concentrations of LG in the respirable range. Measurement of LG could serve as a valuable exposure assessment tool for fine and ultrafine smoke particles from biomass burning in the presence of other confounding factors including silicates and fossil fuel/gas engine exhaust. Additional work appears needed to confirm whether respirable exposure to LG is a robust indicator of acute pulmonary function changes resulting from inhalation of wildland fire smoke.

Because there were no periods during our field study when the crews were not fighting fires, our study is not able to indicate which firefighters might have cross-shift declines in the absence of smoke exposure, and whether individual firefighters with greater cross-shift declines during non-exposure periods are at greater risk for progressive declines in lung function. Future studies could benefit from inclusion of cross-shift information during periods when participants were unexposed to provide evidence of whether cross-shift FEV_1 declines such as those we observed were caused by fire-related exposures.

However, evidence from worker populations chronically exposed to other airborne occupational hazards suggests that individuals with greater cross-shift declines during exposure periods are at potentially greater risk for long-term declines in lung function.[21–23] In addition, we are not aware of any studies indicating a positive correlation between exercise and FEV_1, but it would be helpful to know whether exercise causes an increase in FEV_1 and whether this is an important consideration or not.

TWA CO concentrations differed by job and were not strongly correlated with total particulate, respirable particulate, organic carbon, and respirable LG. These findings differ from observations made by Reinhardt and Ottmar[9] who gathered area samples (specifically, TWAs of respiratory irritants including CO, acrolein, formaldehyde, and $PM_{3.5}$) at eight wildfires and 39 prescribed fires from 1991–1995. Those authors observed a high correlation between pollutants, supporting the use of CO as a surrogate for other pollutants.[9] Although in our study higher CO readings were associated with higher particulate concentrations, and measurement of lower CO concentrations were associated with a wide range of particulate concentrations. Thus, contrary to the observations of Reinhart and Ottmar, low CO readings may not provide reliable assurance that concomitant particulate exposures are, in fact, low. Nevertheless, a small, inexpensive CO monitor could still be used instead of a bulky, expensive particle monitor to screen for conditions of high average exposures.

When RT CO and particulate concentrations were examined as TWAs, the r-values for their correlation were high. However, when examined on the basis of 1-min interval data, the correlations remained statistically significant (likely due to the large amount of data), but the r-values decreased. For example, the highest correlation indicated that only 22% of the variation in minute-by-minute particulate concentration could be explained by variation in CO concentration ($r = 0.47$). Furthermore, the positive correlation based on TWAs was driven by job, specifically Sawyers and Swampers. It is possible that exhaust from the chainsaw is contributing to the elevated levels observed for these individuals.

We therefore conclude that although CO concentration may serve as a surrogate for particulate concentration when examined by mean values, CO concentration may be less instructive when predicting associations based on individual data points. This discrepancy would need to be considered during the design phase of protocol development when outcomes (e.g., peak exposures versus averages) and analytical methods are determined.

We found significant differences in exposures by crew operation. Specifically, concentrations of RT particulate and respirable LG (as measured by both cyclone and impactor) were higher with fire line construction than mop-up operations. Note that intercomparison of RT particulate concentration measurements alone is limited by possible task-related aerosol differences in light-scattering response. Job task differences in wildland firefighter exposures have been observed previously. Reinhardt and Ottmar reported that smoke exposure was highest in direct attack operations (where a fire line is dug adjacent to an active wildfire) compared with fire line construction farther from the active fire.[9]

Observed differences in organic carbon concentration were not significant between the base camp and the fire line locations, possibly due to the small number of samples taken ($n = 7$), but the general observation of higher organic carbon concentrations near the fire line was consistent with the generally higher concentrations of airborne particle mass. The loose agreements of respirable particulate and respirable LG concentrations measured by impactor and cyclone shown in Tables III–V are also consistent with the limited power of small-sample-number statistics.

It is of note that the estimated cross-shift ΔFEV_1 associated with exposure to higher concentrations of LG increased in magnitude from an unadjusted decline of 0.11 L to an adjusted decline of 0.18 L after controlling for job and crew operation. Specifically, job-related ΔFEV_1 declines were greatest for Swampers (compared to Lead workers, Line workers, and Sawyers) and operation-related declines were greater for fire line construction (compared to mop-up). As previously noted, Swampers were exposed to the highest concentrations of LG, CO, RT particulate, and respirable particulate. Moreover, both respirable particulate and LG mean concentrations were higher for fire line construction than for mop-up. Indeed, the second largest particle concentration percent mass collected (13%) was found in the ultrafine range and nearly two-thirds of the LG collected was found in the respirable range. In addition, in our previous analysis of wood smoke aerosol[24] we observed that highly reactive ·OH radical precursors were more prevalent (per unit mass) in the ultrafine particles, possibly explaining the mechanism for this association with greater cross-shift declines in lung function.

Sample collection is a challenge in wildland fire situations. Our analyses of wildfire aerosol particles by SEM and EDX revealed three distinct types of particles—a crystal-like particle comprised primarily of titanium, iron, and aluminum silicate; a spherical carbon particle, most likely tar; and aggregates of many tar-like particles—and is supportive of previous findings of Booze et al.[13] and Sandberg and Martin.[25] Use of impactors, although requiring greater labor for sampler preparation and greater cost for the analysis of multiple substrates, does yield useful information on particle size distribution, total concentration, and respirable concentration. Use of cyclones requires evaluation of only a single filter, but yields less information.

Confirmation of the equivalency of the impactor and cyclone methods for measurement of respirable concentrations was not quantitatively possible in the current study because it was not feasible to ask firefighters to wear more than a CO monitor plus one other sampler at a time. Perhaps future success in developing miniaturized samplers will overcome this limitation by enabling the collection of paired impactor and cyclone sample sets for job and crew activities. Area sampling in the current study was the only situation in which side-by-side impactor and cyclone samples were collected. When area values of respirable particulate concentration as measured by impactor were compared to the area values of respirable particulate concentration simultaneously measured by cyclone, they were not found to be significantly different ($p = 0.71$).

Limitations of Study

Interpretation and application of our findings involves a number of limitations. Our study involved only one fire and included a relatively small number of participants ($n = 17$) and sampling days ($n = 4$), which limited the statistical power of some of our analyses and may also restrict the generalizability of our findings. Our study also lacked cross-shift data during periods in which the crews were working but not fighting a fire. Such data could have strengthened our finding that the observed ΔFEV_1 declines were caused by exposure to wildland fire smoke particles. Note, however, that a recent study by Adetona et al. [26] did not find significant changes in cross-shift lung function on burn days compared to non-burn days.

We also recognize that use of the qualitative terms "mild," "moderate," and "severe" is subjective, and responses of the subjects to our instructions were based on their individual perception of the smoke conditions. Given the fact that smoke exposures perceived by the subjects as "none" or "mild" are associated with ΔFEV_1 declines, a more quantitative measure of self-reported smoke exposure should be found. We have considered training options (such as visible sight distance or smoke comparison to a printed gray-scale card) that might provide a more common basis for subject responses, but we have not taken that step.

The focus of our study was on health concerns related to firefighter exposures to aerosols in the respirable size range. As shown in Figure 1, the mode of the airborne particle size distribution was above the respirable size range, indicating that a complete characterization of the airborne particle size distribution would require use of instrumentation capable of providing size-fractionated samples in the range greater than 30 μm and possibly as large as several hundred μm. Characterization of the larger particle range could be done, but would need to be justified on the basis of concerns for health effects in the head airways and conducting airways.

CONCLUSION

Despite limitations, results from the current study inform considerations and decision-making for improving the protection of respiratory health. Respiratory protection is not normally worn by wildland firefighters, but NIOSH announced in July 2012 that in collaboration with the Safety Equipment Institute, they will begin issuing certificates of approval for respirators for use during wildland fire-fighting operations.[27] Certification will be in compliance with NFPA 1984-2011.[28]

We conclude that wildland firefighter smoke exposures are characterized by a wide range of particle sizes and that inhalation of fine smoke particles, including associated airborne concentrations of respirable LG, is associated with acute lung function declines in some wildland firefighters. Based on these short-term findings, it appears important to address possible long-term respiratory health issues for workers involved in wildland fire fighting.

ACKNOWLEDGMENTS

The authors thank the Alpine IHC for their participation in the study. We thank the U.S. Department of the Interior, the National Park Service, and the National Interagency Fire Center for arranging for the crew's participation. The authors also thank Chuck Stanich's Incident Management Team for data collected at the Red Eagle Fire.

In addition, the authors thank the following NIOSH personnel for assistance with various aspects of protocol development, data collection at the Red Eagle Fire, data analysis, and/or manuscript preparation and review: Michael Beaty, Randy Boylstein, Robert Castellan, Kristin Cummings, Gerald Hobbs, Thomas Jefferson, Richard Kanwal, Kay Kreiss, Greg Kullman, Aleksandr Stefaniak, Brian Tift, and David Weissman.

We dedicate this article to the memory of the 19 wildland firefighters from the Granite Mountain IHC of Prescott, Arizona, who perished battling the Yarnell Hill Fire, 80 miles northwest of Phoenix, Arizona, on June 30, 2013.

The findings and conclusions in this report are those of the authors and do not necessarily represent the views of NIOSH. Mention of any company or product does not constitute endorsement by NIOSH.

REFERENCES

1. **National Interagency Fire Center:** "Total Wildland Fires and Acres (1960–2009)." Available at http://www.nifc.gov/fireInfo/fireInfo_stats_totalFires.html (accessed June 14, 2013).
2. **Betchley, C.**, **J.Q. Koenig**, **G. VanBelle**, **H. Checkoway, and T. Reinhardt**: Pulmonary function and respiratory symptoms in forest firefighters. *Am. J. Ind. Med. 31*:503–509 (1997).
3. **Gaughan, D.M.**, **J.M. Cox-Ganser**, **P.L. Enright**, **et al.:** Acute upper and lower respiratory effects in wildland firefighters. *J. Occup. Environ. Med. 50*:1019–1028 (2008).
4. **Liu, D.**, **I.B. Tager**, **J.R. Balmes**, and **R.J. Harrison:** The effect of smoke inhalation on lung function and airway responsiveness in wildland firefighters. *Am. Rev. Respir. Dis. 146*:1469–1473 (1992).
5. **Materna, B.L.**, **J.R. Jones**, **P.M. Sutton**, **N. Rothman, and R.J. Harrison**: Occupational exposures in California wildland fire fighting. *Am. Ind. Hyg. Assoc. J. 53*:69–76 (1992).
6. **Rothman, N.**, **D.P. Ford**, **Baser, M.E.**, **et al.:** Pulmonary function and respiratory symptoms in wildland firefighters. *J. Occup. Med. 33*:1163–1167 (1991).
7. **Swiston, J.R.**, **W. Davidson**, **S. Attridge**, **G.T. Li**, **M. Brauer, and van S.F. Eeden:** Wood smoke exposure induces a pulmonary and systemic inflammatory response in firefighters. *Eur. Respir. J. 32*:129–138 (2008).
8. **Hejl, A.M.**, **O. Adetona**, **D. Diaz-Sanchez**, **et al.**: Inflammatory effects of woodsmoke exposure among wildland firefighters working at prescribed burns at the Savannah River Site, SC. *J. Occup. Environ. Hyg*. *10*:173–180 (2013).
9. **Reinhardt, T.E., and R.D. Ottmar:** Baseline measurements of smoke exposure among wildland firefighters. *J. Occup. Environ. Hyg. 1*:593–606 (2004).
10. **Slaughter, J.C.**, **J.Q. Koenig, and T.E. Reinhardt:** Association between lung function and exposure to smoke among firefighters at prescribed burns. *J. Occup. Environ. Hyg. 1*:45–49 (2004).
11. **Edwards, R.**, **M. Johnson**, **K.H. Dunn, and L.P. Naeher:** Application of real-time particle sensors to help mitigate exposures of wildland firefighters. *Arch. Environ. Occup. Health 60*:40–43 (2005).
12. **Lee, S.**, **K. Baumann**, **J.J. Schauer**, **et al.:** Gaseous and particulate emissions from prescribed burning in Georgia. *Environ. Sci. Technol. 39*:9049–9056 (2005).
13. **Booze, T.F.**, **T.E. Reinhardt**, **S.J. Quiring, and R.D. Ottmar:** A screening-level assessment of the health risks of chronic smoke exposure for wildland firefighters. *J. Occup. Environ. Hyg. 1*:296–305 (2004).
14. **Jarjour N.N., and W.J. Calhoun:** Enhanced production of oxygen radicals in asthma. *J. Lab. Clin. Med. 123*:131–136 (1994).
15. **Simoneit, B.R.T.**, **J. J. Schauer**, **C.G. Nolte**, **et al.:** Levoglucosan, a tracer for cellulose in biomass burning and atmospheric particles. *Atmos. Environ. 33*:173–182 (1999).
16. **Miller, M.R.**, **J. Hankinson**, **V. Brusasco**, **et al.:** Standardisation of spirometry. *Eur. Respir. J. 26*(*2*):319–338 (2005).
17. **Hankinson J.L.**, **J.R. Odencrantz, and K.B. Fedan:** Spirometric reference values from a sample of the general U.S. population. *Am. J. Respir. Crit. Care. Med. 159*:179–187 (1999).
18. **Ghio, A.J.**, **R.M. Castellan**, **K.B. Kinsley, and J.L. Hankinson:** Changes in forced expiratory volume in one second and peak expiratory flow rates across a work shift among unexposed blue collar workers. *Am. Rev. Respir. Dis. 143*:1231–1234 (1991).
19. **Rubow, K.L.**, **V.A. Marple**, **J. Olin, and M.A. McCawley:** A personal cascade impactor: Design, evaluation and calibration. *Am. Ind. Hyg. Assoc. J. 48*:532–538 (1987).
20. **Finkelstein, M.M., and D.K. Verma:** Exposure estimation in the presence of nondetectable values: Another look. *Am. Ind. Hyg. Assoc. J. 62*(*2*):195–198 (2001).
21. **Wang, X.**, **H.X. Zhang**, **B.X. Sun**, **et al.:** Cross-shift airway responses and long-term decline in FEV_1 in cotton textile workers. *Am. J. Respir. Crit. Care. Med. 177*(*3*):316–320 (2008).
22. **Becklake, M.R.**: Relationship of acute obstructive airway change to chronic (fixed) obstruction. *Thorax 50*(*Suppl 1*):S16–21 (1995).
23. **Glindmeyer, H.W.**, **J.J. Lefant**, **R.N. Jones**, **R.J. Rando, and H. Weill:** Cotton dust and across-shift change in FEV_1 as predictors of annual change in FEV_1. *Am. J. Respir. Crit. Care Med. 149*:584–590 (1994).
24. **Leonard, S.S.**, **V. Castranova**, **B.T. Chen**, **et al.:** Particle size-dependent radical generation from wildland fire smoke. *Toxicology 236*(*1–2*):103–113 (2007).
25. **Sandberg, D.V., and R.E. Martin:** Particle Size in Slash Fire Smoke. USDA Forest Service research paper. PNW-199. Pacific Northwest Forest and Range Experiment Station, Portland Ore., 1975.
26. **Adetona O., D.B. Hall, and L.P. Naeher:** Lung function changes in wildland firefighters working at prescribed burns. *Inhal. Toxicol. 23*(*13*):835–841 (2011).
27. **Ahlers, H.:** "NIOSH Initiation of Respirator Certification of Respirators with Protections for Wildland Fire-Fighting Operations," 2012. Available at http://www.cdc.gov/niosh/npptl/resources/pressrel/letters/Manufacturers/lttr-07102012.html?s_cid=3ni7d2TW1207171200 (accessed June 14, 2013).
28. **National Fire Protection Agency:** "NFPA 1984: Standard on Respirators for Wildland Fire Fighting Operations 2011 Edition." Available at: http://www.nfpa.org/catalog/product.asp?pid=198411 (accessed June 14, 2013).

Acute Upper and Lower Respiratory Effects in Wildland Firefighters

Denise M. Gaughan, MPH
Jean M. Cox-Ganser, PhD
Paul L. Enright, MD
Robert M. Castellan, MD, MPH
Gregory R. Wagner, MD
Gerald R. Hobbs, PhD
Toni A. Bledsoe, MS
Paul D. Siegel, PhD
Kathleen Kreiss, MD
David N. Weissman, MD

Objectives: *To assess acute respiratory effects experienced by wildland firefighters.* ***Methods:*** *We studied two Interagency Hotshot Crews with questionnaires, spirometry, and measurement of albumin, eosinophilic cationic protein (ECP), and myeloperoxidase (MPO) as indicators of inflammation in sputum and nasal lavage fluid. Assessments were made preseason, postfire, and postseason.* ***Results:*** *Fifty-eight members of the two crews had at least two assessments. Mean upper and lower respiratory symptom scores were higher postfire compared to preseason ($P < 0.001$). The mean forced expiratory volume in 1 second was lower postfire compared to preseason ($P < 0.001$) and then recovered by postseason. Individual increases in sputum and nasal ECP and MPO from preseason to postfire were all significantly associated with postfire respiratory symptom scores.* ***Conclusions:*** *Wildland firefighting was associated with upper and lower respiratory symptoms and reduced forced expiratory volume in 1 second. Within individuals, symptoms were associated with increased ECP and MPO in sputum and nasal lavage fluid. The long-term respiratory health impact of wildland firefighting, especially over multiple fire seasons, remains an important concern.* (J Occup Environ Med. *2008;50:1019–1028*)

Municipal and wildland firefighters have an increased prevalence of respiratory problems.[1,2] The chief inhalation hazards associated with wildland firefighting have been identified as carbon monoxide (CO), aldehydes, and respirable particulate matter.[3] Much research has documented deleterious effects of smoke exposure in municipal firefighters.[4–7] Those results, however, may not be generalizable to wildland firefighters, given differences in smoke composition, generally longer duration of fires fought by wildland firefighters, and the fact that respiratory personal protective equipment is routinely, though not always, worn by municipal firefighters but is not generally worn by or even recommended for wildland firefighters.[8]

The Federal government employs about 15,000 seasonal and permanent wildland firefighters each year.[9] Many additional wildland firefighters are employed by State and private agencies. There are four types of wildland firefighter crews: engine crew, hand crew, helicopter crew, and smokejumpers. Type 1 Interagency Hotshot Crews (IHCs) are elite 20-member hand crews that construct fire lines using hand tools during the most dangerous phases of fire suppression.

At a 1997 conference on wildland firefighter health and safety, attendees acknowledged that respiratory problems were common in wildland firefighters and accounted for 30% to 50% of visits to fire incident medical aid stations.[10] Studies examining respiratory symptoms and pulmonary function in wildland firefighters have

From the National Institute for Occupational Safety and Health (NIOSH) (Ms Gaughan, Dr Cox-Ganser, Dr Castellan, Dr Wagner, Ms Bledsoe, Dr Siegel, Dr Kreiss and Dr Weissman), Morgantown, WVa.; University of Arizona (Dr Enright), Tucson, Ariz.; West Virginia University (Dr Hobbs), Morgantown, WVa.

Address correspondence to: Denise M. Gaughan, MPH, NIOSH MS-H2800, 1095 Willowdale Road, Morgantown, WV 26505; E-mail: dug5@cdc.gov.

DOI: 10.1097/JOM.0b013e3181754161

found increases in symptoms, airways hyperresponsiveness, and declines in lung function cross-shift and cross-season.[11–16]

These previously observed increases in subjective symptoms and declines in objective measures of lung function suggest that wildland firefighting is associated with upper and lower airways inflammation and raise concern about potential risk of long-term respiratory effects, including asthma, chronic obstructive pulmonary disease (COPD), and upper airways conditions such as sinusitis. To our knowledge, examination of induced sputum or nasal lavage fluid for objective measures of eosinophilic and neutrophilic inflammation during wildland firefighting has not previously been done.

The question addressed by the present study was whether wildland firefighting is associated with acute and sub-chronic respiratory effects. To address this question, we serially assessed symptoms, spirometry, and markers of inflammation in sputum and nasal lavage fluid in members of two type 1 IHCs of wildland firefighters.

Materials and Methods

Study Population

From 2004 through 2006, we attempted to collect medical and exposure data preseason (in May), in a wildfire setting, and postseason (in October, a minimum of 2 weeks postfire exposure) on all members of the only two type 1 IHCs employed by the National Park Service. We studied the Alpine IHC of Rocky Mountain National Park for 7 days while fighting the Boundary Fire (Fox, AK, July 2004), a very large and intense wildfire, for 7 days while working the Tuolumne Grove Fire (Yosemite National Park, CA, October 2005), a less intense prescribed burn, and for 6 days while fighting the Red Eagle Fire (Glacier National Park, MT, August 2006), a large wildfire. We studied the Arrowhead IHC of Sequoia and Kings Canyon National Parks for 3 days while fighting the South Sundance Fire Complex (Sundance, WY, July 2005), a smaller wildfire that was nearly completely contained during our testing. Preseason participation was 100% for both crews in both years, but crew turnover within seasons and the demobilization of the Alpine IHC shortly after the late-season fire in California resulted in incomplete data for some preseason participants on each crew. Also, a fire-related death of an Arrowhead crew member in the 2004 season led to a decision to cancel studies of this crew for the remainder of that season.

The study protocol was approved by the National Institute for Occupational Safety and Health (NIOSH) Human Subjects Review Board and informed consent was obtained from each research participant.

Questionnaire

A preseason questionnaire, a modification of the standardized American Thoracic Society (ATS) Adult Respiratory Questionnaire,[17] ascertained: lifetime chronic respiratory conditions, history of tobacco use, history of symptoms over the past week, volunteer firefighter status, and lifetime occupational history. A separate postfire/postseason questionnaire ascertained: exposures, new diagnoses, work histories, and changes in symptoms since the last interview. A validated symptom scale, with Likert scoring where 0 = none, 1 = trivial, 2 = mild, 3 = moderate, and 4 = severe for upper and lower airways symptoms, was used to derive overall symptom scores by summing the responses to questions on 19 symptoms.[18] Symptoms ascertained included cough, wheeze, sputum production, shortness of breath or chest tightness, and shortness of breath while walking, as well as various eye, nose, and throat symptoms. At each wildfire studied, participants were also asked daily to rate the severity (none, mild, moderate, or severe) of his or her smoke exposure for the preceding shift.

Spirometry

Spirometry was conducted preseason, daily cross-shift during each studied wildfire, at the conclusion of each studied wildfire, and postseason. Technicians who had completed a NIOSH-approved spirometry course followed ATS guidelines using an ultrasonic flow spirometer (EasyOne Diagnostic Spirometry System 2001, ndd Medical Technologies, Zurich, Switzerland).

We used equations for predicted values and lower limits of normal (LLN) derived from National Health and Nutrition Examination Survey (III) data.[19] We defined obstruction as a ratio of the forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC) $<$ LLN with FEV_1 $<$ LLN; borderline obstruction as an FEV_1/FVC ratio $<$ LLN with normal FEV_1 and normal FVC; and restriction as a normal FEV_1/FVC ratio with FVC $<$ LLN. We used several criteria to define FEV_1 changes in an individual as potentially significant: two criteria, a decline of 12% or greater[20] and a decline of 8% or greater,[21] for cross-season decline; and one criterion of 10% or greater for cross-shift FEV_1 decline.[22]

We followed ATS procedure by requesting medications and asking participants to abstain from these medications for 1 hour before performing spirometry at the preseason, postfire, and postseason assessments. Nevertheless, we did not ask a participant to abstain from his or her medications during cross-shift testing at a wildfire, as we felt this may have jeopardized his or her safety.

Induced Sputum and Nasal Lavage Analyses

Whole induced sputum was collected using a well-validated technique that minimizes salivary contamination of the sample.[23] Nasal lavage was collected using normal saline as previously described.[24]

Due to the challenges of handling specimens in the setting of active forest fires, we used a simplified sample processing and analytical approach modified from Metso et al.[25] In this approach, cells within the sample are lysed and total intracellular and extracellular myeloperoxidase (MPO) and eosinophilic cationic protein (ECP) are assessed, providing measures of neutrophilic and eosinophilic inflammation, respectively. Albumin was also examined as a marker of inflammatory-associated transudation. After collection, samples were frozen on dry ice in the field and held at −80°C until analyzed. In the laboratory, samples were thawed, volumes measured, and mucus liquefied by addition of two volumes 1% dithiothreitol (Sputolysin Stat-Pack; Caldon Biotech) to one volume of sample. In addition, the protease inhibitor phenylmethanesulfonyl fluoride (Sigma Chemical Co) was added to a final concentration of 1 mM and proteolytic inhibitor cocktail (Cat. # P-8340, Sigma Chemical Co) was added to a final concentration of 0.05%. After shaking for 15 minutes at room temperature, aliquots were removed and complete cell lysis facilitated by addition of an equal volume of 0.4% hexadecyltrimethylammonium bromide, followed by vigorous shaking for 1 hour at room temperature.[26] The samples were centrifuged (800 × g for 10 minutes) and supernatant fluids stored frozen in aliquots at −80°C until analyzed for albumin, ECP, and MPO.

Human albumin was quantified using enzyme-linked immunosorbent assay (ELISA) (Cat. # E80-129, Bethyl Laboratories, Inc., Montgomery, TX), following manufacturer's instructions. Assays were performed using reagents included in the ELISA kit, as well as MaxiSorp 96-well ELISA plates (Nunc A/S, Denmark), and TMB Microwell Peroxidase Substrate (KPL Inc, Gaithersburg, MD). ECP was measured in duplicate by fluoroimmunoassay (Pharmacia CAP System; Phadia, Uppsala, Sweden) as per the manufacturer's instructions. MPO was measured by ELISA in duplicate and at several sample dilutions for extrapolation from the MPO ELISA standard curve, as instructed by the manufacturer (Assay Designs Inc, Ann Arbor, MI). For our analyses, we used the resulting concentrations (reflecting intracellular plus extracellular content) of each analyte in sputum and nasal lavage fluid[24] and total recovered amounts of albumin, ECP, and MPO. Total recovered amounts were calculated by multiplying the analyte concentrations by the recovered volumes.

Exhaled Breath Carbon Monoxide

Exhaled breath CO was collected daily cross-shift on each participant during each studied wildfire using a breath CO monitor according to the manufacturer's instructions (Micro 4 Smokerlyzer, Bedfont Scientific, Medford, NJ).

Statistical Methods

We restricted our analyses to data from: 1) all three assessments (ie, preseason, at fire, and postseason) from the first complete fire season of data for participants with all three assessments during at least one fire season ($n = 32$); and 2) the preseason or fire assessment and one other assessment from the first season for which a preseason or fire and only one other assessment was done for other participants ($n = 26$). Data from participants with only a preseason survey ($n = 11$) were excluded from analyses of health effects.

Cross-season predictors of several outcome variables (FEV_1, FVC, upper and lower respiratory symptom scores, and sputum and nasal lavage fluid albumin, ECP, MPO, and volume) were examined using the SAS MIXED procedure for repeated measures with a first-order autoregressive correlation structure.[27] In multifactor models, FEV_1 values were adjusted for age, sex, height, and race/ethnicity. Sputum and nasal lavage fluid results were log-transformed for inclusion in the models. The following time-varying predictor variables were examined: cumulative months spent fighting fires (throughout career), days spent fighting fires (current season), fire assignment, asthma, allergies, upper respiratory infections, upper and lower respiratory symptom scores, and smoking status. Similar models were examined to assess preseason to postfire differences and postfire to postseason differences in outcome variables. To investigate the influence of the preseason values of each of the outcome variables on subsequent postfire and postseason values of the same variable, we ran models where the outcome variables were restricted to postfire or postseason observations.

We also examined postfire associations between respiratory symptom scores and change in inflammatory markers from preseason within individuals using ordinary least squares techniques. These models were adjusted for preseason respiratory symptom scores.

Cross-shift mean changes in FEV_1 and exhaled breath CO at a fire were investigated using paired difference *t* tests. We moreover examined associations between individual participants' mean cross-shift change in FEV_1 and several predictor variables (age, gender, height, race/ethnicity, asthma, allergies, fire assignment, self-reported smoke exposure rating, and postshift exhaled breath CO) using multiple regression models comparable to those detailed above.

Results

Characteristics for the entire group of preseason participants ($n = 69$) and the group of participants included in the health effects analyses ($n = 58$) are detailed in Table 1. Based on these characteristics, the group analyzed was very similar to the entire population of these two crews. They had a median age of 26 years, had similar firefighting experience, and were comprised primarily of White, non-Hispanic males. Ap-

TABLE 1
Characteristics of Interagency Hotshot Crew (IHC) Members at Preseason Assessment

Variable	Participants at Preseason Testing *n* = 69	Participants Included in Analyses *n* = 58
Median age, yr	26 (22, 33)*	26 (22, 33)*
Median time spent as a firefighter, mo	1 (1, 85)	1 (1, 85)
Male *n* (%)	61, 88%	52, 90%
White, non-Hispanic *n* (%)	64, 93%	55, 95%
Current smoker *n* (%)	5, 7%	3, 5%
Former smoker *n* (%)	17, 25%	15, 26%
Allergies (ever) *n* (%)	24, 35%	20, 34%
Asthma (ever) *n* (%)	13, 19%	10, 17%
Median forced expiratory volume in 1 s (FEV_1) % predicted	101 (87, 116)	102 (87, 116)
Median forced vital capacity (FVC) % predicted	102 (89, 116)	102 (89, 118)
Median FEV_1/FVC (%)	83 (73, 88)	83 (73, 89)

*Tenth, 90th percentiles.

proximately 5% were current smokers and about 26% were former smokers. Nearly 35% reported having allergies and about 17% reported having been diagnosed with asthma. Median pulmonary function values were about 100% of predicted.

At the preseason evaluation, 13 participants reported ever having had physician-diagnosed asthma (Table 2). Three participants reported initial asthma diagnosis in adulthood after becoming a firefighter; all three reported current asthma and current asthma medication, but had normal spirometry. Of the 10 participants reporting an asthma diagnosis in childhood, two reported current asthma. One of these two, a current smoker, had abnormal (obstructive) spirometry and was taking asthma medication. None of the others reporting childhood asthma had abnormal spirometry. Among participants with no reported history of respiratory disease, four (three never smokers; one smoker) had borderline obstruction and one (a never smoker) had mild restriction at the preseason assessment.

At the postseason evaluation, firefighters reported an average of 16 fire assignments over the season. Crew assignments between preseason assessment and the studied fire averaged nine fires: firefighting averaged 4 days and shift length averaged 14 hours. Crew assignments between the studied fire and the postseason assessment averaged six fires: firefighting averaged 4 days and shift length averaged 14 hours.

Questionnaire

Upper and lower respiratory symptom scores were both higher postfire compared to preseason and postseason (Table 3). The mean upper respiratory symptom score was 5.0 preseason, compared to 14.1 postfire ($P < 0.001$) and 8.6 postseason ($P < 0.05$). The postseason score was also significantly lower than the postfire score ($P < 0.001$). The mean lower respiratory symptom score was 1.7 preseason, compared to 4.1 postfire, ($P < 0.001$) and 2.5 postseason ($P = 0.27$). The postseason score was significantly lower than the postfire score ($P < 0.05$).

TABLE 2
Preseason Characteristics of the 13 Participants who Reported Ever Having Asthma

Current Asthma	Age at Asthma Diagnosis (Yrs)	Smoking History	Forced Expiratory Volume in 1 Second (FEV_1) (% Predicted)	Forced Vital Capacity (FVC) (% Predicted)	FEV_1/ FVC (%)	Current Medication for Asthma
Yes	5	Yes	73*	98	61*	Yes
Yes	6	No	115	118	82	No
Yes	23	Yes	102	102	83	Yes
Yes	30	No	117	119	82	Yes
Yes	32	No	111	103	87	Yes
No	5	No	116	123	81	—
No	8	Yes	93	91	85	—
No	9	Yes	103	105	81	—
No	10	No	98	99	81	—
No	11	Yes	95	92	85	—
No	11	No	118	113	86	—
No	12	No	88	86	85	—
No	12	No	99	100	86	—

*Spirometry value abnormal.
–, not applicable.

TABLE 3
Unadjusted Mean Values for Symptoms, Spirometry, and Inflammatory Marker Concentrations

	Preseason		Postfire		Postseason		*P**		
	Score or Value	*n*	Score or Value	*n*	Score or Value	*n*	Preseason to Postfire	Preseason to Postseason	Postfire to Postseason
Symptoms									
Upper respiratory symptoms	5.0	56	14.1	50	8.6	42	$P < 0.001$	$P < 0.05$	$P < 0.001$
Lower respiratory symptoms	1.7	56	4.1	50	2.5	42	$P < 0.001$	NS	$P < 0.05$
Spirometry									
FEV_1 (L)	4.57	56	4.35	50	4.54	42	$P < 0.001$	NS	$P < 0.001$
FVC (L)	5.58	56	5.53	50	5.62	42	NS	NS	NS
Sputum									
ECP (μg/L)	1457	56	1537	50	1128	42	NS	NS	NS
MPO (ng/mL)	10,457	56	6464	50	8075	42	NS	NS	NS
Albumin (μg/mL)	177	56	233	50	134	42	NS	NS	NS
Volume (mL)	4.3	56	5.8	50	4.9	42	$P < 0.05$	NS	NS
Nasal Lavage Fluid									
ECP (μg/L)	154	56	651	50	584	42	$P < 0.01$	$P < 0.05$	NS
MPO (ng/mL)	1468	56	3642	50	8745	42	NS	NS	NS
Albumin (μg/mL)	106	56	48	50	88	42	$P < 0.05$	NS	$P < 0.05$
Volume (mL)	6.4	56	6.4	50	5.8	42	NS	$P < 0.01$	$P < 0.01$

**P*-values are from univariate models in which sputum and nasal lavage values were log-transformed.

In multifactor analyses, greater cumulative time spent fighting fires (throughout career) was significantly associated with higher upper respiratory score at each time point ($P < 0.05$) after adjusting for significant associations of preseason respiratory symptom score and with recent upper respiratory infection. Days spent fighting fires (current season), history of asthma, allergies, age, and smoking status were not significantly associated with respiratory symptom scores.

Spirometry

Univariate analysis showed a mean FEV_1 decline of 224 mL ($P < 0.001$) from preseason to postfire, followed by an increase of 190 mL from postfire to postseason ($P < 0.001$) (Table 3). The postseason mean FEV_1 was not statistically different from the preseason mean FEV_1 ($P = 0.60$). Mean FVC values did not change significantly over these same three time points.

One participant's FEV_1 fell 12% across the season. The next largest cross-season decline was 8%, observed in three participants. All four had lung function that remained within the predicted normal range at both preseason and postseason assessments. None reported having been diagnosed with asthma; three were former smokers. Their median age was 23.

The overall mean cross-shift change in FEV_1 was a 30 mL decline ($P = 0.12$). However, cross-shift change varied by fire incident: an 80 mL mean decline at the wildfire in Alaska ($P < 0.001$); a 64 mL mean decline at the prescribed fire in California ($P = 0.12$); a 6 mL mean decline at the wildfire in Wyoming ($P = 0.99$); and a 40 mL mean decline at the wildfire in Montana ($P = 0.08$). Figure 1 details the results from the wildfire in Alaska, the fire associated with the greatest cross-shift changes in FEV_1.

Four of the 19 participants at the Alaska fire each experienced a single cross-shift FEV_1 decline greater than 10% (range: 10% to 11%). All four had normal lung function at both preseason and postseason assessments. One reported having been diagnosed with asthma (resolved); two were former smokers. Their median age was 27. No other cross-shift declines of that magnitude were observed for any participants at the other three fires.

In multifactor analysis, after adjusting for a significant association between an individual's preseason FEV_1 and that individual's subsequent FEV_1 values, lower FEV_1 values were associated with greater upper respiratory symptom scores ($P < 0.05$), with higher sputum ECP recovered values ($P < 0.05$), and with higher sputum MPO recovered values ($P < 0.01$). Similar associations were observed when we examined concentration values of these inflammatory markers. Cumulative time spent fighting fires (throughout career), days spent fighting fires (that season), allergies, asthma, upper respiratory infection in the week preceding testing, and smoking status were not significantly associated with FEV_1 at any time point.

In multifactor analysis, cross-shift FEV_1 change was not significantly associated with age, gender, height, race/ethnicity, asthma, allergies, fire assignment, postshift exhaled breath CO, or self-reported smoke exposure rating.

Induced Sputum and Nasal Lavage Analyses

Induced Sputum. Mean sputum concentrations (reflecting intracellular

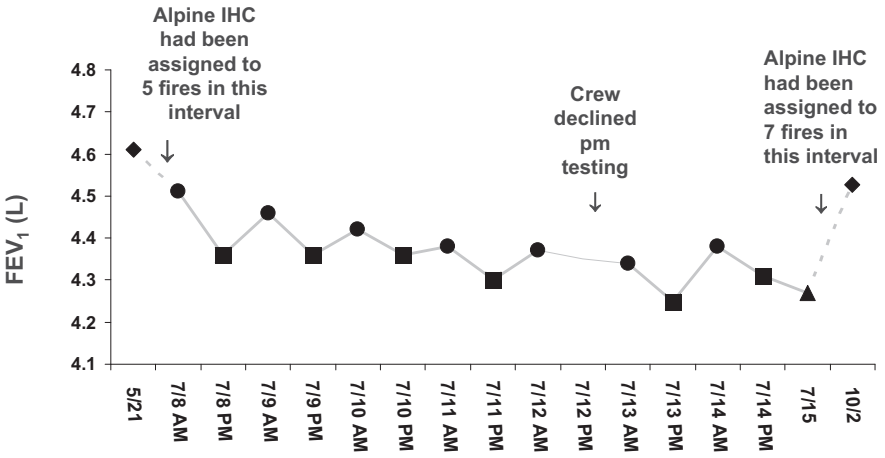


Fig. 1. Unadjusted mean FEV_1 values relating to the Boundary Fire, Fox, AK (n = 19 participants). IHC: Interagency Hotshot Crew. Preseason and postseason testing (◆); preshift testing during fire (●); postshift testing during fire (■); postfire testing (▲). Statistically significant declines were observed from mean preseason FEV_1 to mean preshift FEV_1 on first day of fire ($P < 0.05$), from mean preseason FEV_1 to mean postfire FEV_1 ($P < 0.05$), and in mean cross-shift FEV_1 ($P < 0.001$). No statistically significant difference was observed preseason to postseason.

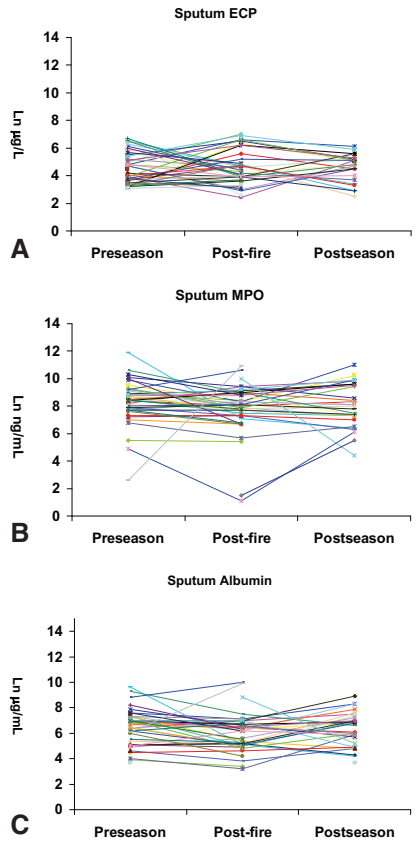


Fig. 2. ECP (A), MPO (B), and albumin (C) concentrations in sputum (n = 56 participants). Measurements reflect both intracellular and extracellular ECP and MPO (see Methods). ECP, eosinophil cationic protein; MPO, myeloperoxidase.

plus extracellular content) of albumin, ECP, and MPO were not significantly different at preseason, postfire, or postseason (Table 3 and Figs. 2A–C). There was marked variability, both within individuals and between surveys, in all of these measures (Figs. 2A–C). Mean sputum volume was significantly increased postfire compared to preseason ($P < 0.05$).

In multifactor analyses of inflammatory markers in sputum, higher MPO concentrations were associated with higher scores for both upper respiratory symptoms ($P < 0.01$) and lower respiratory symptoms ($P < 0.01$); higher ECP concentrations were associated with higher scores for both upper respiratory symptoms ($P < 0.05$) and lower respiratory symptoms ($P < 0.05$); and albumin concentrations were not significantly associated with any of the examined factors. Each model was adjusted for significant association between preseason and subsequent values of the respective outcome variable. Comparable associations were observed in models examining total recovered amount of each of these three inflammatory markers.

Within individuals, postfire respiratory symptom scores were significantly associated with preseason to postfire differences in total recovered amount of albumin, ECP, or MPO in sputum (Table 4). The greater the increase in sputum ECP from preseason to postfire, the higher the postfire scores for upper respiratory symptoms ($P < 0.01$) and lower respiratory symptoms ($P < 0.001$). Greater increases in sputum MPO from preseason to postfire were also associated with higher scores for postfire upper respiratory symptoms ($P < 0.001$) and lower respiratory symptoms ($P < 0.001$). In contrast to other parameters measured in sputum, differences in sputum albumin were inversely related to respiratory symptoms: the greater the increase in albumin from preseason to postfire, the lower the postfire scores for upper respiratory symptoms ($P < 0.01$) and lower respiratory symptoms ($P < 0.001$). Similar associations were observed when we examined concentration values of these three inflammatory markers.

Nasal Lavage. Compared with sputum values, there was marked variability in nasal lavage concentrations (reflecting intracellular plus extracellular content) of ECP and MPO (Table 3 and Figs. 3A–C). Still, mean ECP concentration in nasal lavage fluid increased significantly from preseason to postfire ($P < 0.01$) and from preseason to postseason ($P < 0.05$). In contrast, mean albumin concentration in nasal lavage fluid decreased significantly from preseason to postfire ($P < 0.05$) and from preseason to postseason ($P < 0.05$). Mean MPO concentration did not significantly change over the three time points.

In multifactor analyses of inflammatory markers in nasal lavage fluid, higher ECP concentrations were significantly associated with higher lower respiratory symptom scores ($P < 0.05$); MPO and albumin concentrations were not significantly associated with any of the examined factors. The ECP and MPO models were adjusted for significant associations between preseason and subsequent values of ECP and MPO,

TABLE 4
Within-Individual Associations Between Changes in Inflammatory Markers and Postfire Respiratory Symptom Scores

Preseason to Postfire Change in Total Recovered Amount*	*P*	
	Postfire Upper Respiratory Symptom Score	Postfire Lower Respiratory Symptom Score
Sputum		
ECP (increase)	$P < 0.01$	$P < 0.001$
MPO (increase)	$P < 0.001$	$P < 0.001$
Albumin (decrease)	$P < 0.01$	$P < 0.001$
Nasal Lavage Fluid		
ECP (increase)	$P < 0.01$	$P < 0.001$
MPO (increase)	NS	$P < 0.05$
Albumin (decrease)	NS	NS

*Parenthetical "increase"/"decrease" indicates direction of association.

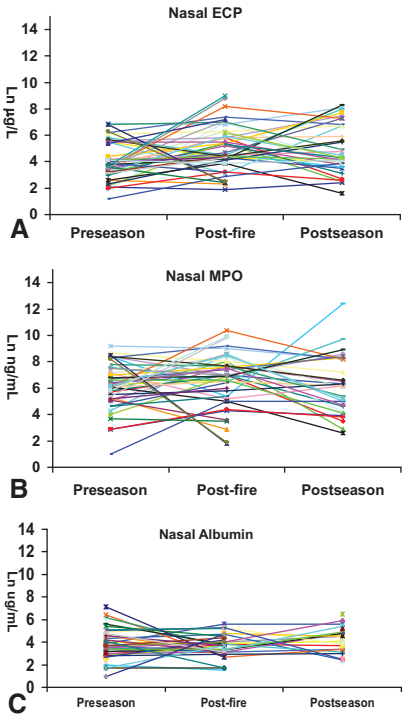


Fig. 3. ECP (A), MPO (B), and albumin (C) concentrations in nasal lavage fluids (n = 56 participants). Measurements reflect both intracellular and extracellular ECP and MPO (see Methods). ECP, eosinophil cationic protein; MPO, myeloperoxidase. Mean nasal ECP concentration was significantly lower preseason compared to postfire ($P < 0.01$) and postseason ($P < 0.05$). Mean nasal albumin was significantly lower postfire compared to preseason ($P < 0.05$) and postseason ($P < 0.05$).

respectively. The MPO model was furthermore adjusted for a significant association between MPO concentration and older age. Cumulative time spent fighting fires (throughout career), days spent fighting fires (that season), having an upper respiratory infection in the week preceding testing, allergies, asthma, and smoking status were not significantly associated with sputum or nasal fluid inflammatory markers or volume.

Within individuals, postfire respiratory symptom scores were significantly associated with preseason to postfire differences in nasal lavage fluid ECP and MPO (Table 4). The significance of these associations was essentially the same regardless of whether ECP and MPO values were expressed as concentrations or total recovered amounts. The larger the increase in ECP from preseason to postfire, the higher the postfire score for upper respiratory symptoms ($P < 0.01$). Also, the larger the increase in ECP ($P < 0.001$) or MPO ($P < 0.05$) from preseason to postfire, the higher the postfire score for lower respiratory symptoms. Changes in albumin from preseason to postfire were not significantly associated with postfire respiratory symptom scores.

Exhaled Breath Carbon Monoxide

The mean preshift exhaled breath CO level was 2.7 parts per million (ppm) and the mean postshift level was 3.7 ppm ($P < 0.001$). No individual CO levels exceeded 16 ppm.

Discussion

We observed significantly increased respiratory symptom scores postfire compared to preseason. This finding is consistent with observations made by Rothman et al,[15] who observed a significant increase in eye irritation, nose irritation, cough, phlegm, and wheezing from preseason to late-season among 52 wildland firefighters, with strong associations noted for recent firefighting activity. In our study, the increased scores for lower respiratory symptoms observed postfire returned to near preseason levels during the postseason. Upper respiratory symptom scores remained significantly elevated at postseason compared to preseason, although scores were significantly lower at postseason compared to postfire. These observations suggest substantial recovery from respiratory tract effects of firefighting by the time of our postseason assessment. Nevertheless, the finding in multifactor analyses that cumulative time spent fighting fires over a career was significantly associated with increased upper (but not lower) respiratory symptoms suggests that wildfire-associated exposure may produce a more sustained rhinitis/sinusitis. Betchley et al[11] observed no significant increase in symptoms cross-season in their study of 53 wildland firefighters, but their postseason testing was done well over a month later in the season than ours and may have allowed for more complete recovery. However, previous NIOSH investigators who made postseason assessments earlier in the calendar year than ours also found no cross-season increase in symptoms.[13]

Corresponding to our symptoms findings, we observed a statistically significant reduction in mean FEV_1 postfire compared to preseason. Likewise, we observed recovery of FEV_1 from postfire to postseason.

Similarly, a previous study of type I Hotshot crews found no significant mean cross-season FEV_1 decline.[13] These findings contrast with those from somewhat larger studies by Liu et al[12] and Betchley et al,[11] who did observe significant mean cross-season FEV_1 declines among wildland firefighters.

We observed an overall mean cross-shift decline in FEV_1 during firefighting that was not statistically significant. However, the magnitude and statistical significance varied by individual fire, and the mean cross-shift decline at the Alaska fire was highly statistically significant. Although reasons for cross-shift differences by fire are uncertain, the fire intensity and the extended shifts worked during the Alaska fire may have contributed to the greater cross-shift decline observed for that fire. Betchley et al,[11] Slaughter et al,[16] and previous NIOSH investigators[13] also observed statistically significant mean cross-shift FEV_1 declines among wildland firefighters during fires.

Only one participant in our study experienced a cross-season decline in FEV_1 as much as 12%, a criterion recommended by an ATS/European Respiratory Society Task Force for defining statistically significant FEV_1 change for an individual.[20] A total of four of the 33 (12%) participants with cross-season measurements experienced a cross-season decline in FEV_1 of at least 8%, an alternative criterion for defining significant FEV_1 change for an individual over a 6- to 12-month period.[21]

A different four participants experienced a single cross-shift decline in FEV_1 of 10% or greater, a criterion for defining a statistically significant cross-shift drop in FEV_1.[22] Notably, all four of the significant cross-shift changes occurred at the Alaska fire. Although our study cannot indicate whether individual firefighters with greater cross-shift and cross-season declines are at greater risk for progressive declines in lung function over subsequent years, evidence from worker populations chronically exposed to other occupational agents with acute respiratory effects would suggest that they are at greater risk.[28,29]

We predicted that respiratory symptoms or changes in lung function associated with wildland firefighting would be associated with airways inflammation. Inflammation of the lower airways is found in both asthma and COPD and plays an important role in the pathogenesis of these disorders.[30] COPD has been associated with neutrophilic inflammation and the neutrophil-related enzyme MPO in sputum.[31] Allergic asthma is commonly associated with increases in eosinophils and ECP in sputum, though some work-related asthma has been found to be predominantly mediated by neutrophils.[32,33] Wildland firefighting is stressful; psychological stress has been associated with exacerbation of eosinophilic inflammatory changes in sputum of asthmatics.[34] Wildland firefighting is also physically demanding; intense physical exertion has been associated with neutrophilic inflammatory changes in sputum.[35] Eosinophilic and neutrophilic inflammation in upper airway conditions, including allergic and nonallergic rhinitis, have been associated with increased inflammatory markers in nasal lavage fluid.[36–38]

We found that assessments of population mean concentrations of ECP and MPO were somewhat informative for nasal lavage fluid but were not informative for sputum. Nevertheless, within-person analyses of airways inflammation in wildland firefighters indicated that greater preseason to postfire increases in sputum ECP and MPO were significantly associated with higher postfire respiratory symptom scores. Similarly, greater preseason to postfire increases in ECP and MPO in nasal lavage fluid were significantly associated with higher postfire respiratory symptom scores. These results suggest that symptoms reflect induction of airways inflammation by wildfire-related exposures.

In contrast to results of the within-person analyses of sputum ECP and MPO, within-person analyses of sputum albumin indicated that greater preseason to postfire increases in sputum albumin were significantly associated with lower postfire respiratory symptom scores (Table 4). Also, in contrast to mean nasal lavage fluid ECP, mean nasal lavage albumin was reduced postfire. These findings seem paradoxical, as increased airway albumin is commonly employed as a marker of transudation. During inflammation, transudation can be mediated both pharmacologically by mediators such as histamine and pathologically by loss of integrity of the endothelial and/or epithelial barriers. It is possible that compensatory mechanisms such as development of tolerance to repeated insult and/or increased albumin clearance (eg, through digestion by inflammatory proteases in the airways) could have contributed to the decreased postfire albumin levels we observed in nasal lavage fluid.

Typically, sputum is processed for cellular analysis by separating cells and cell-free supernatant.[39] However, given the need to treat all sputum and nasal lavage fluid samples uniformly in difficult wildfire settings, we did not obtain cell counts. Rather, we lysed cells in the liquefied samples and expressed ECP and MPO as concentration and as total amount in recovered sputum and nasal lavage fluid, representing the combined content of the intra- and extra-cellular compartments.[25] Although not the usual approach to evaluation of neutrophilic and eosinophilic inflammation, this method was feasible to perform in this particular study and has been suggested as an option for sputum analysis.[40]

An important limitation of our study is the relatively small number of participants, which may have limited our ability to detect some associations. Although, we did observe some temporal associations of symptoms and pulmonary function results

with firefighting activities, the generally qualitative and self-reported nature of exposure characterization in our study may have limited our ability to have identified statistically significant symptom and pulmonary effects related to firefighting exposures (eg, time spent fighting fires, fire assignment, self-reported smoke exposure rating, and postshift exhaled breath CO). Exhaled breath CO was the one objectively measured and quantitative, though indirect, measurement of exposure that we analyzed. Exhaled breath CO has been shown to be elevated in people with COPD.[41] Exhaled breath CO showed a significant cross-shift increase, offering objective evidence for exposure to the products of combustion during the firefighting shifts. Nevertheless, exhaled breath CO was not found to be a significant determinant of cross-shift FEV_1 decline, perhaps because exhaled breath CO is not a reliable surrogate for the irritant smoke particulate and gas exposures that likely cause airways effects in firefighters.

Our study lacked cross-shift data during periods in which the crews were not fighting fires, which would have enabled comparison of the magnitude of fire-associated cross-shift FEV_1 declines with the magnitude of cross-shift declines, if any, that these same participants may experience when not subjected to firefighting exposures. Having cross-shift data during nonfire periods may have strengthened the inference that the cross-shift declines we observed were caused by fire-related exposures. However, ruling out physical exhaustion as a cause of our cross-shift findings would have required that the participants be subjected to the same physical exertion and exhaustion during the unexposed periods of study as during the firefighting periods.

We collected data from the Alpine IHC at three fires, each lasting 6 to 7 days in duration, and data from the Arrowhead IHC at only one fire lasting 3 days. Thus, both crews were not equally represented in the analyses, and we studied only one of the crews while fighting a largely uncontained wildfire—the Alaska fire—at which we observed the largest cross-shift FEV_1 declines.

Our findings are limited by crew attrition over the course of the wildfire season. It is possible that more susceptible participants might have been more likely to have left the crew midseason and been lost to follow-up. If so, we may have underestimated the apparent effect of firefighting on cross-season change in FEV_1. Some evidence suggests that this may have occurred. Participants who quit the crew during the season ($n = 5$) had a higher mean score for lower respiratory symptoms at preseason than all other participants ($n = 64$) (3.8 vs 2.6; $P = 0.05$). The five who quit also had a slightly lower mean preseason percent predicted FEV_1 (98%) compared to participants who remained (101%), although this difference was not statistically significant.

In summary, we observed cross-shift reductions in lung function among type 1 IHC members while firefighting. We also observed statistically significant differences in mean lung function and respiratory symptom scores at the postfire assessment compared to the preseason assessment. At the individual level, increased ECP and MPO in sputum and nasal lavage fluid were associated with higher postfire respiratory symptom scores. Although we observed evidence of recovery from most of the short-term effects by the end of the firefighting season, they raise the possibility that wildland firefighters may be at increased risk for development of chronic lung and upper airways disease. This possibility is additionally supported by our finding that upper respiratory symptom scores were related to cumulative time spent fighting wildfires over a career. More studies are warranted to investigate potential long-term adverse respiratory effects of firefighting among wildland firefighters. In the meantime, the Federal Interagency Wildland Firefighter Medical Qualification Standards Program was created in 2001 to monitor the health of wildland firefighters employed by the federal government and engaged in arduous duties. Firefighters initially undergo a comprehensive medical examination including spirometry, followed by periodic examinations (depending on employment status). Program information can be found at http://www.nifc.gov/medical_standards/index.htm.

Acknowledgments

The authors thank the Alpine IHC and Arrowhead IHC for their participation in the study and the Bonneville IHC for serving as an alternate crew. We also thank the U.S. Department of the Interior, the National Park Service, and the National Interagency Fire Center for arranging for the crews' participation. We also thank Steve Hart's Incident Management Team for data collection at the Boundary Fire, Don Angell's Incident Management Team for data collection at the South Sundance Fire Complex, Mike Beasley, Fire Use Manager, Yosemite National Park, for data collection at the Tuolumne Grove Fire, and Chuck Stanich's Incident Management Team for data collected at the Red Eagle Fire. Finally, the authors thank the following NIOSH personnel for data collection and processing: Michael Beaty, Lisa Benaise, Nicole Edwards, Kathleen Fedan, Diana Freeland, Monica Graziani, Amber Harton, Thomas Jefferson, Brandon Law, Jennifer Mosser, Richard Kanwal, Margaret Kitt, Christopher McManus, Chris Piacitelli, Nancy Sahakian, Elizabeth Shogren, David Spainhour, James Taylor, Brian Tift, Sandra White and Daniel Yereb. The findings and conclusions in this report are those of the authors and do not necessarily represent the views of NIOSH. Mention of any company or product does not constitute endorsement by NIOSH.

References

1. Musk AW, Smith TJ, Peters JM, McLaughlin E. Pulmonary function in firefighters: acute changes in ventilatory capacity and their correlates. *Br J Ind Med*. 1979;36:29–34.
2. Guidotti TL. Human factors in firefighting: ergonomic-, cardiopulmonary-, and psychogenic stress-related issues. *Int Arch Occup Environ Health*. 1992;64:1–12.
3. Materna BL, Jones JR, Sutton PM, Rothman N, Harrison RJ. Occupational

exposures in California wildland firefighting. *Am Ind Hyg Assoc J*. 1992;53:69–76.
4. Bergstrom CE, Eklund A, Skold M, Tornling G. Bronchoalveolar lavage findings in firefighters. *Am J Ind Med*. 1997; 32:332–336.
5. Burgess JL, Nanson CJ, Bolstad-Johnson DM, et al. Adverse respiratory effects following overhaul in firefighters. *J Occup Environ Med*. 2001;43:467–473.
6. Chia KS, Jeyaratnam J, Chan TB, Lim TK. Airway responsiveness of firefighters after smoke exposure. *Br J Ind Med*. 1990;47:524–527.
7. Scannell CH, Balmes JR. Pulmonary effects of firefighting. *Occup Med*. 1995; 10:789–801.
8. Federal Fire and Aviation Leadership Council. *Interagency Standards for Fire and Aviation Operations*. Boise, ID: National Interagency Fire Center; 2007:pp. 07–10. Publication No. NFES 2724. Available at: http://www.nifc.gov/red_book/2007/Chapter07.pdf.
9. Wildland Fire Leadership Council. *2007 Budget Justification*. Washington DC; 2007. Available at: http://www.fireplan.gov/resources/documents/NFP2007_budget_justification.pdf.
10. Sharkey B, ed. *Health Hazards of Smoke: Recommendations of the April 1997 Consensus Conference*. Tech Rep., 9751-2836-MTDC. Missoula, MT: U.S. Department of Agriculture, Forest Service, Missoula Technology and Development Center; 1997:84.
11. Betchley C, Koenig JQ, VanBelle G, Checkoway H, Reinhardt T. Pulmonary function and respiratory symptoms in forest firefighters. *Am J Ind Med.* 1997; 31:503–509.
12. Liu D, Tager IB, Balmes JR, Harrison RJ. The effect of smoke inhalation on lung function and airway responsiveness in wildland firefighters. *Am Rev Respir Dis*. 1992;146:1469–1473.
13. NIOSH. *Health Hazard Evaluation Report: U.S. Department of the Interior, National Park Service, Southern California*. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health; 1991. NIOSH HETA Report No. 91-152-2140, NTIS No. PB92-133347.
14. NIOSH. *Health Hazard Evaluation Report: U.S. Department of the Interior, National Park Service, Yosemite National Park, California*. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health; 1994. NIOSH HETA Report No. 90-0365-2415, NTIS No. PB95-242541.
15. Rothman N, Ford DP, Baser ME, et al. Pulmonary function and respiratory symptoms in wildland firefighters. *J Occup Med*. 1991;33:1163–1167.
16. Slaughter JC, Koenig JQ, Reinhardt TE. Association between lung function and exposure to smoke among firefighters at prescribed burns. *J Occup Environ Hyg*. 2004;1:45–49.
17. Ferris BG. Epidemiology standardization project. *Am Rev Respir Dis*. 1978;108:1–113.
18. Wasserfallen JB, Gold K, Schulman KA, Baranuik JN. Development and validation of a rhinoconjunctivitis and asthma symptom score for use as an outcome measure in clinical trials. *J Allergy Clin Immunol*. 1997;100:16–22.
19. Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. *Am J Respir Crit Care Med*. 1999;159:179–187.
20. Pellegrino R, Viegi G, Brusasco V, et al. Interpretive strategies for lung function tests. *Eur Respir J*. 2005;26:948–968.
21. Wang ML, Petsonk EL. Repeated measures of FEV_1 over six to twelve months: what change is abnormal? *J Occup Environ Med*. 2004;46:591–595.
22. Ghio AJ, Castellan RM, Kinsley KB, Hankinson JL. Changes in forced expiratory volume in 1 second and peak expiratory flow rates across a work shift among unexposed blue collar workers. *Am Rev Respir Dis*. 1991;143:1231–1234.
23. Fahy JV, Boushey HA, Lazarus SC, et al. Safety and reproducibility of sputum induction in asthmatic subjects in a multicenter study. *Am J Respir Crit Care Med*. 2001;163:1470–1475.
24. Lignell U, Meklin T, Putus T, et al. Microbial exposure, symptoms and inflammatory mediators in nasal lavage fluid of kitchen and clerical personnel in schools. *Int J Occup Med Environ Health*. 2005;18:139–150.
25. Metso T, Rytila P, Peterson C, Haahtela T. Granulocyte markers in induced sputum in patients with respiratory disorders and healthy persons obtained by two sputum-processing methods. *Respir Med*. 2001;95:48–55.
26. Moshfegh A, Hallden G, Lundahl J. Methods for simultaneous quantitative analysis of eosinophil and neutrophil adhesion and transmigration. *Scand J Immunol*. 1999;50:262–269.
27. SAS Institute Inc. *SAS 9.1.3*. Cary, NC: SAS Institute Inc.; 2000–2004.
28. Becklake MR. Relationship of acute obstructive airway change to chronic (fixed) obstruction. *Thorax*. 1995;50(Suppl 1): S16–S21.
29. Glindmeyer HW, Lefant JJ, Jones RN, Rando RJ, Weill H. Cotton dust and across-shift change in FEV_1 as predictors of annual change in FEV_1. *Am J Respir Crit Care Med*. 1994;149:584–590.
30. Keatings VM, Barnes PJ. Granulocyte activation markers in induced sputum: comparison between chronic obstructive pulmonary disease, asthma, and normal subjects. *Am J Respir Crit Care Med*. 1997;155:449–453.
31. Yamamoto C, Yoneda T, Yoshikawa M, et al. Airway inflammation in COPD assessed by sputum levels of interleukin-8. *Chest*. 1997;112:505–510.
32. Maghni K, Lemiere C, Ghezzo H, Yuquan W, Malo JL. Airway inflammation after cessation of exposure to agents causing occupational asthma. *Am J Respir Crit Care Med*. 2004;169:367–372.
33. Park H, Jung K, Kim H, Nahm D, Kang K. Neutrophil activation following TDI bronchial challenges to the airway secretion from subjects with TDI-induced asthma. *Clin Exp Allergy*. 1999;29:1395–1401.
34. Liu LY, Coe CL, Swenson CA, Kelly EA, Kita H, Busse WW. School examinations enhance airway inflammation to antigen challenge. *Am J Respir Crit Care Med*. 2002;165:1062–1067.
35. Bonsignore MR, Morici G, Riccobono L, et al. Airway inflammation in nonasthmatic amateur runners. *Am J Physiol Lung Cell Mol Physiol*. 2001;281:L668–L676.
36. Noah TL, Henderson FW, Henry MM, Peden DB, Devlin RB. Nasal lavage cytokines in normal, allergic, and asthmatic school-age children. *Am J Respir Crit Care Med*. 1995;152:1290–1296.
37. Svensson C, Andersson M, Persson CG, Venge P, Alkner U, Pipkorn U. Albumin, bradykinins, and eosinophil cationic protein on the nasal mucosal surface in patients with hay fever during natural allergen exposure. *J Allergy Clin Immunol*. 1990;85:828–833.
38. Woodin MA, Hauser R, Liu Y, et al. Molecular markers of acute upper airway inflammation in workers exposed to fuel-oil ash. *Am J Respir Crit Care Med*. 1998;158:182–187.
39. Pizzichini E, Pizzichini MM, Efthimiadis A, et al. Indices of airway inflammation in induced sputum: reproducibility and validity of cell and fluid-phase measurements. *Am J Respir Crit Care Med*. 1996;154:308–317.
40. Kelly MM, Keatings B, Leigh R, et al. Analysis of fluid-phase mediators. *Eur Respir J*. 2002;(Suppl 37):24s–39s.
41. Barnes PJ, Chowdhury B, Kharitonov SA, et al. Pulmonary biomarkers in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 2006;174:6–14.

Taylor & Francis
Taylor & Francis Group

Journal of Toxicology and Environmental Health, Part A
Current Issues

ISSN: 1528-7394 (Print) 1087-2620 (Online) Journal homepage: https://www.tandfonline.com/loi/uteh20

Wildland Smoke Exposure Values and Exhaled Breath Indicators in Firefighters

Ana Isabel Miranda , Vera Martins , Pedro Cascão , Jorge Humberto Amorim , Joana Valente , Carlos Borrego , António Jorge Ferreira , Carlos Robalo Cordeiro , Domingos Xavier Viegas & Roger Ottmar

To cite this article: Ana Isabel Miranda , Vera Martins , Pedro Cascão , Jorge Humberto Amorim , Joana Valente , Carlos Borrego , António Jorge Ferreira , Carlos Robalo Cordeiro , Domingos Xavier Viegas & Roger Ottmar (2012) Wildland Smoke Exposure Values and Exhaled Breath Indicators in Firefighters, Journal of Toxicology and Environmental Health, Part A, 75:13-15, 831-843, DOI: 10.1080/15287394.2012.690686

To link to this article: https://doi.org/10.1080/15287394.2012.690686

Journal of Toxicology and Environmental Health, Part A, 75:831–843, 2012

ISSN: 1528-7394 print / 1087-2620 online
DOI: 10.1080/15287394.2012.690686

Taylor & Francis
Taylor & Francis Group

WILDLAND SMOKE EXPOSURE VALUES AND EXHALED BREATH INDICATORS IN FIREFIGHTERS

Ana Isabel Miranda[1], Vera Martins[1], Pedro Cascão[1], Jorge Humberto Amorim[1], Joana Valente[1], Carlos Borrego[1], António Jorge Ferreira[2], Carlos Robalo Cordeiro[2], Domingos Xavier Viegas[3], Roger Ottmar[4]

[1]CESAM & Department of Environment and Planning, University of Aveiro, Aveiro, Portugal
[2]Centre of Pulmonology of Coimbra University Medical School, Coimbra, Portugal
[3]Association for the Development of Industrial Aerodynamics, University of Coimbra, Coimbra, Portugal
[4]Pacific Wildland Fire Sciences Laboratory, U.S. Forest Service Pacific Northwest, Research Station, Seattle, Washington, USA

Smoke from forest fires contains significant amounts of gaseous and particulate pollutants. Firefighters exposed to wildland fire smoke can suffer from several acute and chronic adverse health effects. Consequently, exposure data are of vital importance for the establishment of cause/effect relationships between exposure to smoke and firefighter health effects. The aims of this study were to (1) characterize the relationship between wildland smoke exposure and medical parameters and (2) identify health effects pertinent to wildland forest fire smoke exposure. In this study, firefighter exposure levels of carbon monoxide (CO), nitrogen dioxide (NO_2), and volatile organic compounds (VOC) were measured in wildfires during three fire seasons in Portugal. Personal monitoring devices were used to measure exposure. Firefighters were also tested for exhaled nitric oxide (eNO) and CO before and after their firefighting activities. Data indicated that exposure levels during firefighting activities were beyond limits recommended by the Occupational Exposure Standard (OES) values. Medical tests conducted on the firefighters also indicated a considerable effect on measured medical parameters, with a significant increase in CO and decrease in NO in exhaled air of majority of the firefighters.

Wildland firefighters are exposed to many hazards, including burns, heat stress, tripping and falling hazards, accidents with hand and power tools, being struck by falling rocks and trees, and exposure to air toxics due to smoke inhalation. Many experienced firefighting personnel consider the air toxics to be only an inconvenience that occasionally produces acute eye and respiratory irritation, nausea, and headache (Reinhardt and Ottmar 2000). Other investigations express concern regarding chronic health impacts, especially cancer, from years of exposure (Austin et al. 2001; Golka and Weistenhofer 2008) or when large-scale fires occur in terrain and atmospheric conditions that force firefighters to work for many days in smoky conditions (Reinhardt et al. 2000).

Smoke from forest fires contains significant amounts of pollutants such as carbon monoxide (CO), particulate matter (PM), nitro-

The authors acknowledge the financial support of the Portuguese Ministry of Science, Technology and Higher Education, through the Foundation for Science and Technology (FCT), for the PhD grants of V. Martins (SFRH/BD/39799/2007) and J. Valente (SFRH/BD/22687/2005), and the postdoctoral grant of J. H. Amorim (SFRH/BPD/48121/2008). Also, FCT is acknowledged for the funding of the national research project FUMEXP (FCOMP-01-0124-FEDER-007023) through the POCI2010 program and the FEDER fund. We are also thankful to all firefighters involved in project FUMEXP.

Address correspondence to Ana Isabel Miranda, CESAM & Department of Environment and Planning, University of Aveiro, 3810-193 Aveiro, Portugal. E-mail: miranda@ua.pt

gen dioxide (NO_2), volatile organic compounds (VOC), and other chemical compounds (Golka and Weistenhofer 2008). Carbon monoxide is a colorless, tasteless, odorless, and nonirritating gas formed when carbon in fuel is not burned completely, and it produces lethality. Carbon monoxide is present in all fire environments and has been described as one of the most common and serious acute hazards for firefighters (Austin et al. 1997; Treitman et al. 1980). Nitrogen dioxide is an oxidant gas that is produced by combustion processes. Both clinical data and results from the exposure of animals under controlled conditions demonstrated that NO_2 is a powerful pulmonary toxin, inducing both tissue necrosis and chronic inflammation (Morrow 1984; Sandstrom et al. 1990). Particulate matter (PM) is abundantly produced during wildland fire, is highly visible, affects ambient air quality, and exerts various adverse health effects depending on its size and chemical composition.

Inhalation is the predominant route of exposure during forest fires, and when particles are in the ambient air, there is a significant likelihood that firefighters will inhale them. It is difficult to determine the adverse health effects attributed to PM in smoke, as the damaging properties of the particles depend not only on the chemical and toxic characteristics but also on their size, shape, and density (Dost 1991; Naeher et al. 2007; Schwela 2001).

Currently there is a growing awareness that smoke produced during forest fires exposes individuals and populations to hazardous concentrations of air pollutants. However, the current state of knowledge regarding the potential health impacts on firefighting personnel is limited, in particular within Europe. The most extensive measurements of smoke exposure among wildland firefighters were conducted in the United States and Australia (Materna et al. 1993; McMahon and Bush 1992; Reinhardt and Ottmar 2000; 2004; Reinhardt et al. 2000; Reisen and Brown 2009). From these field studies it was concluded that firefighters may be exposed to significant levels of CO and respiratory irritants, including formaldehyde, acrolein, and respirable particles (Reinhardt and Ottmar 2000; 2004). Consequently, adverse health effects occur that are acute and instantaneous, and include eye and respiratory irritation, shortness of breath, headaches, dizziness and nausea lasting for hours, and mild impairment of lung function for hours to days (Reinhardt et al. 2000). In addition, chronic health effects such as impaired respiratory function, increased risk of cancer, and cardiovascular disease may be produced by these pollutants (Golka and Weistenhofer 2008; Rothman et al. 1991). Special concern is raised by exposure to respirable particles and potentially toxic compounds adsorbed to them, including polycyclic aromatic hydrocarbons (PAH) and semivolatile organic compounds, some of which may be carcinogenic (Austin et al. 2001; IARC 2010; Le Masters et al. 2006; Youakim 2006), as well as aldehydes, compounds that are classified as probable human carcinogens. The overall evaluation by the International Agency for Research on Cancer (IARC) is that occupational exposure as a firefighter is possibly carcinogenic to humans (IARC 2010b). There are a number of factors that affect the impact of smoke on health, including concentration of air pollutants within the breathing zone of the firefighter, exposure duration, exertion levels, and individual susceptibility such as preexisting lung or heart diseases (Reisen and Brown 2009).

There is a considerable lack of data on firefighters exposed to smoke in Europe. These data are needed for the establishment of cause/effect relationships between exposure to smoke and firefighter health effects. The composition of smoke depends on the type of vegetation consumed, the efficiency of combustion, fuel moisture content, temperature of the fire, and wind conditions (Reisen and Brown 2009). Consequently, exposure results from the U.S. and Australian experiments may not be applicable to European wildland firefighters due to differences in vegetation, fire conditions, and firefighting operations. Further, a major factor influencing exposure is the type of work activities that firefighters carry out and their position relative to the fire during those activities. Therefore it is crucial to (1) assess exposure

at the individual level in Europe, (2) determine whether this exposure might result in health damage, and (3) identify the primary factors influencing the exposure of wildland firefighters. Miranda et al. (2005) presented the first smoke measurements in experimental fires in Europe. Passive sampling devices were used to monitor NO_2 exposure levels. Measurements showed high exposure values affecting firefighters during experimental burns. Recently, Miranda et al. (2010) used portable "in continuum" measuring devices to monitor a group of firefighters' individual exposures to toxic gases and particles during experimental field fires. Measured levels were high, exceeding the Occupational Exposure Standard (OES) limits, in particular for peak limit thresholds. The aims of this study were to (1) further characterize the relationship between wildland smoke exposure and medical parameters during real firefighting situations, and (2) identify health effects pertinent to wildland forest fire smoke exposure.

METHODOLOGY AND EQUIPMENT

Four fire brigades (3 volunteer and 1 professional) were contacted and 40 firefighters were selected to be involved in this study. The fire brigades were from different central districts in Portugal, including Leiria, Coimbra, and Aveiro. More than one fire brigade and district were selected to increase the chances of measuring exposure of firefighters to smoke during a wildland fire. The study was conducted during the 2008, 2009, and 2010 fire seasons (May–October).

Smoke Exposure

Taking into account the available human resources, firefighter age, type of work, years of experience as a firefighter, respiratory diseases, and smoking habits, 10 firefighters from 4 brigades were chosen to be monitored for their individual exposure to smoke emitted during wildfires. From this group of firefighters, seven were volunteers and three were professional. The 10 firefighters were monitored during each fire season. The number of firefighters selected for the study was limited by the number of personal monitoring devices. The firefighters were equipped with a personal device for CO monitoring, and another for VOC and NO_2. The selection of monitoring equipment was based on toughness, weight, possibility of continuous data acquisition, and ease of operation. Table 1 summarizes the instruments specifications. More information regarding the monitoring devices is found in Miranda et al. (2010). Figure 1 shows firefighters with the exposure monitoring equipment and using a respiratory bandana.

The 10 selected firefighters were instructed on how to use the equipment and how to record basic fire data information, including beginning and end of firefighter exposure period (date; hours), fire location (district; municipality, submunicipality), type of fire, and dimension of fire (small fire [less than 1 ha] or large fire [more than 100 ha]). When the selected firefighters were sent to a wildland fire during the 2008, 2009, and 2010 fire seasons, the subjects carried the exposure monitoring equipment. When returning the individuals downloaded the exposure data and filled out the fire occurrence form (fire report).

TABLE 1. Characteristics of the Exposure Equipment

			Characteristics	
Pollutant	Type of data	Equipment	Range	Resolution
VOC	Continuous measurement: 5 s interval	GasAlertMicro 5 PID from BW Technologies	0–1000 ppm	1 ppm
NO_2			0–99.9 ppm	0.1 ppm
CO	Continuous measurement: 5 s interval	GasAlertextreme from BW Technologies	0–1000 ppm	1 ppm

FIGURE 1. Firefighters with the exposure monitoring equipment (color figure available online).

Table 2 presents the available information on exposure duration, geographic location, and area burned for all the monitored fire occurrences (52 in total). Data presented on location and area burned were provided by the fire brigade reports or by the national fire inventory (Portuguese National Forest Authority 2010) developed by the National Forest Authority. For some wildfires the data were not available (n.a.). The data available for 2010 were limited, resulting in only one occurrence where data was collected.

During the 2008, 2009, and 2010 fire seasons, the firefighters were involved with 11, 13, and 28 wildland fire operations. The burned areas were generally smaller than 1 ha. The wildfires of July 30, 2008, August 1, 2008, and July 26, 2010, were the exceptions, with 80, 3, and 5 ha, respectively. These fire seasons were mild and the amount of burned area was small in central Portugal. The exposure periods were always shorter than 8 h, except on July 26, 2010, for firefighter 23, with an exposure period of 11 h. With the exception of this particular situation, in general these periods varied between 15 min (firefighter 7, 01/09/2008) and 7.5 h (firefighter 23, 27/07/2010).

The vegetation burned by wildfires is characterized by resinous (*Pinus pinaster, Pinus pinea*), decidous (*Quercus spp., Castanca sativa*), and eucalyptus species (*Eucalyptus* spp.), and shrubs, namely, *Erica umbellata*, *Erica australis*, and *Chamaespartium tridentatum*.

Medical Tests

The studied sample initially encompassed 38 healthy firefighters. During the 3 years of study, 3 firefighters left their corporations and the final tally of sample individuals was 35, with a mean age of 29.92 yr (standard deviation of 7.065) and a median height of 173 cm (standard deviation of 6.2 cm). The median of years spent in the firefighting force was 9.7, with a standard deviation of 5.6 yr. All firefighters were healthy and were clinically examined by a team of pulmonologists to determine respiratory and general health status. None had previous history of respiratory pathology.

The respiratory function of the 35 firefighters sample was evaluated prior to any exposure, during April 2008, and at the end of the 2010 fire season. Data were collected using the calibrated MicroMedical Spirometer, model MicroLab ML3500. Evaluation was completed following standard procedures and international norms (ATS/ERS 2005). The following spirometry parameters were measured:

- Forced expiratory volume in 1 s (FEV1).
- Forced vital capacity (FVC).
- Ratio FEV1/FVC (Tiffeneau Index, Tiff.).
- Peak expiratory flow (PEF).
- Flow at 50% of FVC (F50).
- Flow at 75% of FVC (F25).
- Midexpiratory flow rate (MEF).

Firefighters were examined, before and after fire-fighting, regarding their NO (eNO), CO, and percent carboxyhemoglobin (COHb) in the exhaled breath for the 2009 and 2010 fire seasons. In case of a wildland fire occurrence, one or two elements of the medical team were contacted by phone by the fire department when firefighters were to be deployed to an incident. All measurements were collected during the first 1.5 h following the work shift. All measurements were acquired in a smoke-free environment,

TABLE 2. Wildland Fires Characteristics and Exposure Durations for 2008, 2009, and 2010 Fire Seasons

Firefighter	Date	Firefighter exposure period			Location (region)	Burnt area (ha)	
		Beginning	End	Duration		Forest	Shrubs
3	01/08/2008	00:42	01:15	0:33	Coimbra	1.5	1.5
4	16/07/2008	17:35	22:12	4:37	Coimbra	0	0.08
	02/08/2008	15:49	17:51	2:02	Coimbra	0	0.2
5	17/08/2008	12:54	13:53	0:59	Coimbra	0.5	0
	04/10/2008	14:59	17:37	2:38	Coimbra	0	0.02
6	30/07/2008	17:49	20:32	2:43	Coimbra	80	0
	19/08/2008	17:08	17:57	0:49	Coimbra	0	0.02
	23/08/2008	17:15	17:32	0:17	Coimbra	0	0.02
7	31/07/2008	20:32	21:50	1:18	Coimbra	0.1	0
	01/09/2008	15:07	15:22	0:15	Coimbra	0	0.01
9	18/07/2008	16:13	19:22	3:09	Aveiro	1	0
11	05/08/2009	08:59	10:12	1:13	Leiria	n.a.	n.a.
14	28/07/2009	15:43	17:13	1:30	Coimbra	0.2	0
	01/09/2009	21:33	01:41	4:08	Coimbra	0	n.a
	05/09/2009	12:20	18:51	6:31	Coimbra	0.6	0
15	15/07/2009	15:26	16:38	1:12	Coimbra	n.a.	n.a.
	17/09/2009	10:23	10:45	0:22	Coimbra	n.a.	n.a.
17	08/09/2009	15:45	17:17	1:28	Coimbra	0.5	n.a.
	12/09/2009	17:32	18:13	0:41	Coimbra	0	0.015
	20/09/2009	10:55	11:25	0:30	Coimbra	n.a.	n.a.
18	13/08/2009	16:09	16:35	0:26	Aveiro	0	0.03
	15/08/2009	17:10	17:44	0:34	Aveiro	n.a.	n.a.
	27/09/2009	14:47	15:06	0:19	Aveiro	0	0.015
	30/09/2009	06:58	10:51	3:53	Aveiro	0	0.5
22	07/08/2010	14:05	17:10	3:05	Leiria	n.a.	n.a.
23	26/07/2010	13:00	24:00	11:00	Coimbra	n.a.	n.a.
	27/07/2010	16:30	24:00	7:30	Coimbra	n.a.	n.a.
	04/08/2010	13:54	17:43	3:49	Coimbra	n.a.	n.a.
	05/08/2010	17:20	19:40	1:40	Coimbra	n.a.	n.a.
	09/08/2010	14:53	16:13	1:20	Coimbra	n.a.	n.a
	10/08/2010	17:19	18:30	1:11	Coimbra	n.a	n.a
	11/08/2010	17:34	20:35	3:01	Coimbra	n.a	n.a
	30/08/2010	16:37	20:25	3:48	Coimbra	n.a.	n.a.
	03/10/2010	03:49	09:00	5:10	Coimbra	n.a.	n.a.
24	24/07/2010	16:47	20:00	3:13	Coimbra	n.a.	n.a.
	26/07/2010	17:25	24:15	5:50	Coimbra	n.a.	n.a.
	07/08/2010	22:57	23:47	0:50	Coimbra	n.a.	n.a.
	11/08/2010	17:34	20:35	3:01	Coimbra	n.a.	n.a.
25	26/07/2010	18:42	20:42	2:00	Coimbra	5	0
	17/09/2010	22:32	23:28	0:56	Coimbra	n.a.	n.a.
28	27/07/2010	14:44	18:30	3:46	Aveiro	n.a.	n.a.
	28/07/2010	12:48	15:48	3:00	Aveiro	n.a.	n.a.
	29/07/2010	15:25	23:24	7:59	Aveiro	n.a.	n.a.
	03/08/2010	13:26	14:44	1:18	Aveiro	n.a.	n.a.
	04/08/2010	14:36	23:56	9:20	Aveiro	n.a.	n.a.
29	25/05/2010	11:04	18:04	7:00	Aveiro	n.a.	n.a.
	31/05/2010	15:53	17:52	1:59	Aveiro	n.a.	n.a.
	07/06/2010	10:40	12:57	2:17	Aveiro	n.a.	n.a.
	10/06/2010	13:34	15:14	1:40	Aveiro	n.a.	n.a.
30	19/07/2010	15:33	16:08	0:35	Aveiro	n.a.	n.a.
	21/07/2010	17:55	23:35	5:40	Aveiro	n.a.	n.a.
	26/07/2010	11:25	23:59	12:34	Aveiro	n.a.	n.a.

Note. References 1 to 10 correspond to fire season 2008 firefighters, 11 to 20 to 2009, and >20 to 2010; n.a., not available.

away from the forest fire area. The eNO was measured using Nioxmino equipment from AEROCRINE. In 2008 the MICRO CO/Smokecheck from Micromedical was used to measure alveolar CO. This equipment allows monitoring four classes of values. In 2009 and 2010 wildfires the Micro CO (Micromedical Viasys) was used instead of the previous equipment and therefore quantitative measures of CO were possible, as well as measurement of COHb.

RESULTS AND DISCUSSION

Exposure Results

Exposure results were compared to Occupational Exposure Standards (OES) defined for different air pollutants. According to the American Conference of Governmental Industrial Hygienists (ACGHI), OES are presented as: (i) threshold limit value (TLV) of the time-weighted average (TWA); (ii) TLV of the short-term exposure limit (STEL); and (iii) peak limit. The TWA is calculated over a normal 8-h working day and a 5-d working week. The TLV-STEL corresponds to a 15-min TWA exposure that should not be exceeded at any time during a workday, even if the 8-h TWA is under the TLV. The TLV-STEL is the higher concentration to which it is believed that workers may be exposed continuously for a short period of time without suffering effects. In Portugal, OES values for occupational activities are established by Occupational Health and Safety (OHS) regulations through the Portuguese Regulation NP 1796:2007. In the case of CO and NO_2, TLV-TWA values are established by the NP 1796:2007. Table 3 presents the OES values for the different air pollutants analyzed under this study.

For CO TLV-STEL and peak limit, the exposure limits set by the Australian Safety and Compensation Council (1995) were considered. Due to the lack of a NO_2 peak limit in the Portuguese OHS regulation, a value of 20 ppm was considered. This value is proposed by the National Institute for Occupational Safety and Health (NIOSH), taking into consideration recommendations derived from acute inhalation toxicity data (Patty 1963), which indicate this limit value as immediately dangerous to life or health (IDHL). Table 4 shows the TWA and the number of exceedances of the peak limit (and the maximal value), and indicates whether the STEL was fulfilled or not, for firefighters exposed in the wildfires of 2008, 2009, and 2010. It is worth mentioning that the TWA values were calculated based on exposure time and not on 8-h periods. In bold are the situations in which the limit values are exceeded or criteria not fulfilled. Exposure data are missing (n.d.) for some of the firefighters due to technical issues.

Although most firefighters use a bandana for respiratory protection, the protection offered by this filtering device is limited. According to Reh et al. (1994) the pore size of this type of bandanas is approximately 200 μm × 200 μm, roughly 500- to 2000-fold larger than the smaller smoke particles (0.100–0.400 μm); consequently, gases and fine particulate matter pass through the fabric.

There were several exceedances to the TWA, STEL, and peak values for CO. The CO peak limit concentration was exceeded 40% of the time for the 52 reported occurrences and

TABLE 3. OES Limit Values for Different Air Pollutants Contained in Biomass Burning Smoke

Air pollutant	TLV-TWA	Reference	TLV-STEL	Reference	Peak limit	Reference
CO	25 ppm	NP 1796:2007	200 ppm	Australian legislation	400 ppm	Australian legislation
NO_2	3 ppm		5 ppm	NP 1796:2007	20 ppm	NIOSH
VOC	n.a.		n.a.	n.a.	n.a.	n.a.

Note. For some VOC these values are not available (n.a.) in national or international regulations.

TABLE 4. TWA, Number of Peak (n), Peak Values, and TLV-STEL Fulfillment for CO, NO_2, and VOC for Firefighters Exposed to Smoke During Wildland Fires

Firefighter	Date	Parameter	CO (ppm)	NO_2 (ppm)	VOC (ppm)
3	01/08/2008	TWA	5.8	1.9	2.3
		n (Peak value)	0 (143)	0 (3)	5
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
4	16/07/2008	TWA	11.6	0.05	1.2
		n (Peak value)	**3 (544)**	0 (4)	25
		Fulfilment of TLV-STEL criteria	**No**	Yes	n.a.
	02/08/2008	TWA	22.8	0.17	0.9
		n (Peak value)	**6 (684)**	0 (6)	21
		Fulfilment of TLV-STEL criteria	**No**	Yes	n.a.
5	17/08/2008	TWA	12.6	1	0.8
		n (Peak value)	0 (367)	0 (3)	34
		Fulfilment of TLV-STEL criteria	**No**	Yes	n.a.
	04/10/2008	TWA	**30.5**	0.7	1.2
		n (Peak value)	**2 (422)**	0 (5)	20
		Fulfilment of TLV-STEL criteria	**No**	Yes	n.a.
6	30/07/2008	TWA	8.1	n.d.	n.d.
		n (Peak value)	0 (155)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	19/08/2008	TWA	**53.4**	n.d.	0.8
		n (Peak value)	**1 (410)**	n.d.	12
		Fulfilment of TLV-STEL criteria	**No**	n.d.	n.a.
	23/08/2008	TWA	1.8	n.d.	n.d.
		n (Peak value)	0 (93)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
7	31/07/2008	TWA	2.8	1.1	0.04
		n (Peak value)	0 (128)	0 (5)	5
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
	01/09/2008	TWA	8.8	n.d.	n.d.
		n (Peak value)	0 (78)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
9	18/07/2008	TWA	n.d.	2.5	0.2
		n (Peak value)	n.d.	0 (7)	11
		Fulfilment of TLV-STEL criteria	n.d.	Yes	n.a.
11	05/08/2009	TWA	12.6	n.d.	n.d.
		n (Peak value)	0 (170)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
14	28/07/2009	TWA	7.9	0.1	0.2
		n (Peak value)	**1 (413)**	0 (0.9)	42
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
	01/09/2009	TWA	3.1	0.1	n.d.
		n (Peak value)	0 (89)	0 (3.7)	n.d.
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
	05/09/2009	TWA	4.6	0.4	n.d.
		n (Peak value)	0 (64)	0 (7)	n.d.
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
15	15/07/2009	TWA	8.4	0.06	n.d.
		n (Peak value)	0 (179)	0 (1.8)	n.d.
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
	17/09/2009	TWA	1.4	0.1	n.d.
		n (Peak value)	0 (72)	0 (2.8)	n.d.
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
17	08/09/2009	TWA	23.9	1.1	1.4
		n (Peak value)	**7 (597)**	0 (9)	14
		Fulfilment of TLV-STEL criteria	**No**	Yes	n.a.
	12/09/2009	TWA	2.8	n.d.	n.d.
		n (Peak value)	0 (62)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	20/09/2009	TWA	5.1	n.d.	n.d.
		n (Peak value)	**1 (893)**	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.

(*Continued*)

TABLE 4. (Continued)

Firefighter	Date	Parameter	CO (ppm)	NO_2 (ppm)	VOC (ppm)
18	13/08/2009	TWA	23.8	n.d.	n.d.
		n (Peak value)	**1 (405)**	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	**No**	n.d.	n.a.
	15/08/2009	TWA	13.7	n.d.	n.d.
		n (Peak value)	0 (182)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	27/09/2009	TWA	7.4	n.d.	n.d.
		n (Peak value)	0 (182)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	30/09/2009	TWA	4.3	n.d.	n.d.
		n (Peak value)	0 (201)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
22	07/08/2010	TWA	**25**	0.01	1.1
		n (Peak value)	0 (253)	0 (2.5)	6
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
23	26/07/2010	TWA	**34**	0.03	1.7
		n (Peak value)	**15 (817)**	0 (1.8)	12
		Fulfilment of TLV-STEL criteria	**No**	Yes	n.a.
	27/07/2010	TWA	11	n.d.	n.d.
		n (Peak value)	**10 (1,000)**	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	04/08/2010	TWA	17	n.d.	n.d.
		n (Peak value)	**1 (704)**	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	05/08/2010	TWA	23	0.01	0.15
		n (Peak value)	**8 (708)**	0 (2.9)	4
		Fulfilment of TLV-STEL criteria	**No**	Yes	n.a.
	09/08/2010	TWA	19	n.d.	n.d.
		n (Peak value)	0 (241)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.a.	n.a.
	10/08/2010	TWA	**38**	1.1	2.1
		n (Peak value)	**4 (671)**	0 (2.3)	6
		Fulfilment of TLV-STEL criteria	**No**	Yes	n.a.
	11/08/2010	TWA	17	0.05	0.2
		n (Peak value)	0 (283)	0 (1.6)	8
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a
	30/08/2010	TWA	13	0.01	1.8
		n (Peak value)	0 (276)	0 (1.5)	15
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
	03/10/2010	TWA	23	n.d.	n.d.
		n (Peak value)	**16 (1,000)**	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
24	24/07/2010	TWA	11	n.d.	n.d.
		n (Peak value)	0 (108)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	26/07/2010	TWA	11	n.d.	n.d.
		n (Peak value)	**7 (991)**	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	07/08/2010	TWA	24	0.1	0.2
		n (Peak value)	0 (173)	(0.9)	40
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
	11/08/2010	TWA	5	n.d.	n.d.
		n (Peak value)	0 (153)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
25	26/07/2010	TWA	1	0.04	0.16
		n (Peak value)	0 (30)	2	2
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
	17/09/2010	TWA	18	0.24	1.1
		n (Peak value)	0 (214)	0 (5)	5
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.

(*Continued*)

TABLE 4. (Continued)

Firefighter	Date	Parameter	CO (ppm)	NO_2 (ppm)	VOC (ppm)
28	27/07/2010	TWA	21	n.d.	n.d.
		n (Peak value)	0 (294)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	28/07/2010	TWA	11	n.d.	n.d.
		n (Peak value)	0 (174)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	29/07/2010	TWA	11	n.d.	n.d.
		n (Peak value)	**6 (1,000)**	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	03/08/2010	TWA	21	n.d.	n.d.
		n (Peak value)	**6 (599)**	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	04/08/2010	TWA	15	n.d.	n.d.
		n (Peak value)	0 (341)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
29	25/05/2010	TWA	**29**	n.d.	n.d.
		n (Peak value)	**11 (1,000)**	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	**No**	n.d.	n.a.
	31/05/2010	TWA	17	n.d.	n.d.
		n (Peak value)	**1 (446)**	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	07/06/2010	TWA	7	n.d.	n.d.
		n (Peak value)	0 (111)	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
	10/06/2010	TWA	16	n.d.	n.d.
		n (Peak value)	**3 (443)**	n.d.	n.d.
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.
30	19/07/2010	TWA	16	0.3	0.5
		n (Peak value)	**2 (578)**	0.9	5
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
	21/07/2010	TWA	19	0.01	0.6
		n (Peak value)	0 (360)	5.1	64
		Fulfilment of TLV-STEL criteria	Yes	Yes	n.a.
	26/07/2010	TWA	9	n.d.	0.1
		n (Peak value)	0 (364)	n.d.	25
		Fulfilment of TLV-STEL criteria	Yes	n.d.	n.a.

Note. References 1 to 10 correspond to fire season 2008 firefighters, 11 to 20 to 2009, and >20 to 2010; n, number of exceedances to the peak limit; n.a., not applicable - there is no limit value to compare; n.d., no data; in bold, limit values are exceeded or criteria not fulfilled.

for 67% of them more than once. The highest CO peak limit observed was 1,000 ppm. The STEL also exceeded the TLV for nearly 19% of the monitored situations. The exceedence of the STEL is in agreement with studies of Reinhardt and Ottmar (2004), Reisen and Brown (2009), and De Vos et al. (2009).

No exceedances of the TWA, STEL, or peak limits for NO_2 were noted.

There is no national or international legislation that sets TLV-TWA, TLV-STEL, or peak limits for the total VOC but only for the specific compounds. Thus, it was not possible to compare the monitored concentrations with any limit value.

As an example of the time evolution of the monitored concentrations, Figure 2 shows the CO levels for a specific firefighter when combating a specific fire occurrence. The OES limit values defined by the National and International regulations are also indicated (according to Table 3) for a better understanding of the attained exposure values. Data represented in Figure 2 show that the acquired instantaneous concentration values were often high. The CO peak limit was

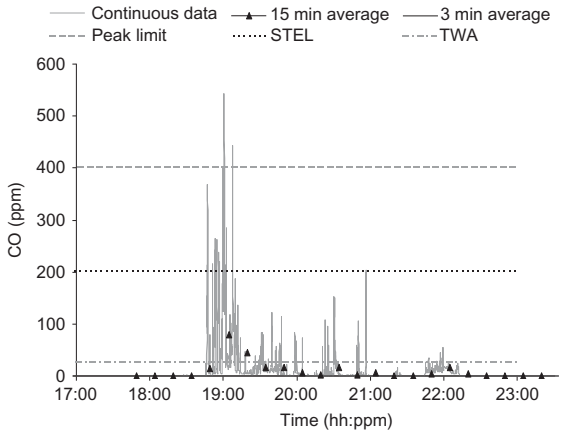


FIGURE 2. Measured CO concentrations for a specific firefighter during a wildfire.

exceeded three times. Similar results were also obtained from other wildfires and for field experiments (Miranda et al. 2010).

Data show the magnitude of the exposure peaks that occurred during regular firefighting operations. De Vos et al. (2009) found that a healthy individual starts experiencing mild headaches after 2–3 h of exposure to CO at 232,840 $\mu g/m^3$ (200 ppm). At 465,680 $\mu g/m^3$ (400 ppm) exposure, the individual experiences nausea, headache, and dizziness after 1 or 2 h. With an exposure concentration of 931,360 $\mu g/m^3$ (800 ppm) or higher, confusion, ataxia, coma, and seizures might develop. At high work levels, such as in the case of firefighting, these symptoms may be expected to appear at lower exposure levels (De Vos et al. 2009). Therefore, knowledge of CO concentration peaks to which firefighters are exposed is crucial.

The presented exposure concentration results are not directly proportional to the area burned. For instance, firefighter 6 was involved in the July 30, 2008, fire that burned 80 ha of forest. His measured exposure values were low; for CO the TWA was 8.1 ppm with a peak exposure of 155 ppm. In contrast, firefighter 4 worked in the August 2, 2008, fire within a smaller burned area (0.2 ha) of mainly shrub. His CO exposure values were higher (the TWA reached 22.8 ppm) and peak and STEL exceedances were recorded. The exposure period for both firefighters was similar, 2h43 and 2h02, respectively. Exposure values depend especially on firefighter position relating to the fire line and task in the crew. Sometimes in small forest fires firefighters are more exposed than in larger fires, because usually the strategy for large fires is to stay back and contain them rather than making a direct attack, as is the usual strategy for smaller fires.

Medical Parameters Results

The respiratory function of the 35 firefighters in April 2008 and at the end of the 2010 fire season was compared and results are presented in Table 5. Data demonstrated a statistically significant decrease of four spirometric parameters (FEV1, F25, F50, and MEF), indicating that firefighters experienced a reduction of respiratory function between the two evaluations. As an example of the eNO measurements before and after smoke exposures, Figure 3 illustrates the changes in the eNO, CO, and COHb values measured just before and after the exposure to smoke in 2010 wildland fires.

There was a significant decrease of eNO between the values measured before and after the exposure to smoke. This result is somewhat unexpected since a decrease on eNO indicates a reduction in airways inflammation. However, this may indicate an effect similar to exposure to cigarette smoke. Indeed, in current smokers Malinovschi et al. (2006) observed a decrease on eNO, probably related to the inhibition of nitric oxide synthetase. In previous studies regarding eNO and smoking habit, peak concentrations were significantly reduced in smokers compared to nonsmokers (Kharitonov et al. 1995), with a significant relationship between the measured values and amount of cigarette consumption. These findings suggest that smoke may inhibit the enzyme NO synthase. Endogenous NO plays an important role protecting the respiratory tract against infection, and counteracting bronchoconstriction, vasoconstriction, and platelet aggregation. This effect may contribute to increased risks of chronic respiratory and cardiovascular disease

TABLE 5. Statistical Comparison Between Spirometric Parameters Obtained in 2008 (Before the Fire Season) and 2010 (After the Fire Season)

		Average	*n*	Standard deviation	*p*
Pair 1	FEV1	103.49	35	12, 349	.028*
	FEV1 2010	101.83	35	10, 063	
Pair 2	CVF	101.26	35	12, 802	ns
	CVF 2010	100.83	35	11, 369	
Pair 3	PEF	96.09	35	13, 744	ns
	PEF 2010	97.57	35	13, 349	
Pair 4	Tiff	105.57	35	8, 624	ns
	Tiff 2010	104.34	35	6, 743	
Pair 5	F50	101.71	35	23, 130	.028+
	F50 2010	96.83	35	20, 024	
Pair 6	F25	93.80	35	35, 192	.005*
	F25 2010	82.11	35	22, 418	
Pair 7	MEF	95.74	35	24, 888	.009*
	MEF 2010	89.77	35	18, 130	

Note. Student's *t*-test for paired data. Asterisk indicates Wilcoxon test.

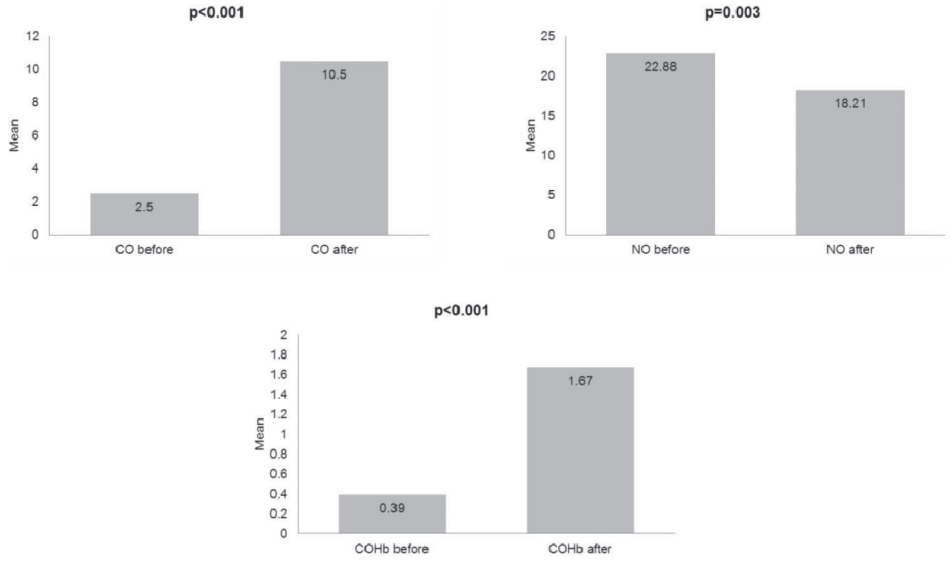


FIGURE 3. Exhaled NO (ppb), CO and COHb (ppm) values measured just before and after exposure to smoke in 2010 wildland fires.

in cigarette smokers. Whether similar findings on eNO represent a similar risk after inhalation of different types of smoke is a matter that needs further investigation.

There was a marked increase of exhaled CO and COHb after exposure to smoke, indicating that O_2 delivery to the body organs and tissues is significantly diminished after smoke exposure, as seen in Figure 3.

CONCLUSIONS

The amount and characteristics of noxious occupational exposures of forest firefighters are not widely recognized, as attention historically focused on risks of urban firefighting. This study indicates that forest firefighting exposed firefighters to very high concentrations of CO, and also high concentrations of NO_2 and VOC, with potential harmful effects on health, even

in wildfires with small burn areas. A particular concern is the peak and short-term exposure to CO. When comparing experimental fires based on Miranda et al. (2010) results to wildland fires data, on average the results present the same order of magnitude for each pollutant analyzed, but higher values were observed in experimental fires. It is not easy to establish a direct relationship between smoke exposure and health respiratory indicators, but results point to an increase of exhaled CO when a higher exposure to CO occurs and a decrease of exhaled NO when the exposure to NO_2 is higher.

REFERENCES

Austin, C. C., Ecobichon, D. J., Dussault, G., and Tirado, C. 1997. Carbon monoxide and water vapor contamination of compressed breathing air for firefighters and divers. *J. Toxicol. Environ. Health A* 52: 403–23.

Austin, C. C., Wang, D., Ecobichon, D. J., and Dussault, G. 2001. Characterization of volatile organic compounds in smoke at experimental fires. *J. Toxicol. Environ. Health A* 63: 191–206.

De Vos, A. J. B. M, Reisen, F., Cook, A., Devine, B., and Weinstein, P. 2009. Respiratory irritants in Australian bushfire smoke: Air toxics sampling in a smoke chamber and during prescribed burns. *Arch. Environ. Contam. Toxicol.* 56:380–88.

Dost, F. N. 1991. Acute toxicology of components of vegetation smoke. *Rev. Environ. Contam. Toxicol.* 119: 1–46.

Golka, K., and Weistenhofer, W. 2008. Fire fighters, combustion products and urothelial cancer. *J. Toxicol. Environ. Health B* 11: 32–44.

International Agency for Research on Cancer. 2010a. Some non-heterocyclic polycyclic aromatic hydrocarbons and some related exposures. *IARC Monogr. Eval. Carcinogen. Risks Hum*. 92. Lyon, France: IARC.

International Agency for Research on Cancer. 2010b. Painting, firefighting, and shiftwork. *IARC Monogr. Eval. Carcinogen. Risks Hum*. 98. Lyon, France: IARC.

Kharitonov, S. A., Robbins, R. A., Yates, D., Keatings, V., and Barnes, P. J. 1995. Acute and chronic effects of cigarette smoking on exhaled nitric oxide. *Am. J. Respir. Crit. Care Med*. 152: 609–12.

LeMasters, G. K., Genaidy, A. M., Succop, P., Deddens, J., Sobeih, T., Barriera-Viruet, H., Dunning, K., and Lockey, J. 2006. Cancer risk among firefighters: A review and meta-analysis of 32 studies. *J. Occup. Environ. Med*. 48: 1189–202.

Malinovschi, A., Janson, C., Holmkvist, T., Norback, D., Merilainen, P., and Hogman, N. 2006. Effect of smoking on exhaled nitric oxide and flow independent nitric oxide exchange parameters. *Eur. Respir. J.* 28: 339–45.

McMahon, C. K., and Bush, P. B. 1992. Forest worker exposure to airborne herbicide residues in smoke from prescribed fires in the southern United States. *Am. Ind. Hyg. Assoc. J.* 53: 265–72.

Materna, B. L., Koshland, C. P., and Harrison, R. J. 1993. Carbon monoxide exposure in wildland firefighting: A comparison of monitoring methods. *Appl. Occup. Environ. Hyg.* 8: 479–87.

Miranda, A. I., Ferreira, J., Valente, J., Santos, P., Amorim, J. H., and Borrego, C. 2005. Smoke measurements during Gestosa 2002 experimental field fires. *Int. J. Wildland Fires* 14: 107–16.

Miranda, A. I., Martins, V., Cascão, P., Amorim, J. H., Valente, J., Tavares, R., Borrego, C., Tchepel, O., Ferreira, A. J., Cordeiro, C. R., Viegas, D. X., Ribeiro, L. M., and Pita, L. P. 2010. Monitoring of firefighters exposure to smoke during fire experiments in Portugal. *Environ. Int*. 36: 736–45.

Morrow, P. E. 1984. Toxicological data on NO_x: An overview. *J. Toxicol. Environ. Health* 13: 205–27.

Naeher, L. P., Brauer, M., Lipsett, M., Zelikoff, J. T., Simpson, C. D., Koenig, J. Q., and Smith, K. R. 2007. Woodsmoke health effects: A review. *Inhal. Toxicol.* 19: 67–106.

Patty, F. A., ed. 1993. Industrial hygiene and toxicology, 2nd rev. ed., vol. II, *Toxicology*,

919–23. New York, NY: Interscience Publishers.

Portuguese National Forest Authority. 2010. Annual report on burnt areas and occurrences (2006–2010). http://www.afn.min-agricultura.pt/portal/dudf/estatisticas/estatistica-sgif

Reh, C. M., Letts, D., and Deitchman, S. 1994. Health hazard evaluation report. U.S. Department of the Interior National Park Service, Yosemite National Park, California, National Institute of Occupational Health and Safety (NIOSH). Available at http://www.cdc.gov/niosh/hhe/reports/pdfs/1990-0365-2415.pdf

Reinhardt, T. E., and Ottmar, R. D. 2000. *Smoke exposure at Western wildfires*. USDA Forest Service Pacific Northwest Research Station Research Paper. Availale at http://www.fs.fed.us/pnw/pubs/pnw_rp525.pdf

Reinhardt, T. E., and Ottmar, R. D. 2004. Baseline measurements of smoke exposure among wildland firefighters. *J. Occup. Environ. Hyg.* 9: 593–606.

Reinhardt, T.E., Ottmar, R. D., and Hanneman, A. 2000. Smoke exposure among firefighters at prescribed burns in the Pacific Northwest. USDA Forest Service Pacific Northwest Research Station Research Paper 526, pp. U1–45. Available at http://www.fs.fed.us/pnw/pubs/pnw_rp526.pdf

Reisen, F., and Brown, S. K. 2009. Australian firefighters' exposure levels to air toxics during bushfire burn of autumn 2005 and 2006. *Environ. Int.* 35: 342–52.

Rothman, N., Ford, D. P., Baser, M. E., Hansen, J. A., O'Toole, T., Tockman, M. S., and Strickland, P. T. 1991. Pulmonary function and respiratory symptoms in wildland firefighters. *J. Occup. Med.* 33: 1163–69.

Sandstrom, T., Andersson, M. C., Kolmodin-Headman, B., Stjernberg, N., and Angstrom, T. 1990. Bronchoalveolar mastotytosis and lymphocytosis after nitrogen dioxide exposure in man: a time kinetic study. *Eur. Respir. J.* 3:138–43.

Schwela, D. 2001. Fire disasters: The WHO-UNEP-WMO health guidelines for vegetation fire events. *Ann. Burns Fire Disasters* 13: 178–79.

Treitman, R. D., Burgess, W. A., and Gold, A. 1980. Air contaminants encountered by firefighters. *Am. Ind. Hyg. Assoc. J.* 41:796–802.

Youakim, S. 2006. Risk of cancer among firefighters: A quantitative review of selected malignancies. *Arch. Environ. Occup. Health* 61: 223–31.

Environmental Research 173 (2019) 462–468

Contents lists available at ScienceDirect

Environmental Research

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

Wildland firefighter smoke exposure and risk of lung cancer and cardiovascular disease mortality

Kathleen M. Navarro[a], Michael T. Kleinman[b], Chris E. Mackay[c], Timothy E. Reinhardt[d], John R. Balmes[e], George A. Broyles[f], Roger D. Ottmar[g], Luke P. Naher[h], Joseph W. Domitrovich[i,*]

[a] USDA Forest Service, Pacific Southwest Region, Fire and Aviation Management, Clovis, CA, USA
[b] Center for Occupational and Environmental Health, University of California, Irvine, CA, USA
[c] Intertox, Seattle, WA, USA
[d] AMEC Foster Wheeler Environment & Infrastructure, Inc., Seattle, WA, USA
[e] Division of Environmental Health Sciences, School of Public Health, University of California, Berkeley, Berkeley, CA, USA
[f] USDA Forest Service, National Technology and Development Program, Boise, ID, USA
[g] USDA Forest Service, Pacific Northwest Research Station, Seattle, WA, USA
[h] Department of Environmental Health Science, College of Public Health, University of Georgia, Athens, GA, USA
[i] USDA Forest Service, National Technology and Development Program, Missoula, MT, USA

ARTICLE INFO

Keywords:
Wildland fire
Firefighters
Smoke
Particulate matter
Risk assessment

ABSTRACT

Wildland firefighters are exposed to wood smoke, which contains hazardous air pollutants, by suppressing thousands of wildfires across the U. S. each year. We estimated the relative risk of lung cancer and cardiovascular disease mortality from existing $PM_{2.5}$ exposure-response relationships using measured PM_4 concentrations from smoke and breathing rates from wildland firefighter field studies across different exposure scenarios. To estimate the relative risk of lung cancer (LC) and cardiovascular disease (CVD) mortality from exposure to $PM_{2.5}$ from smoke, we used an existing exposure-response (ER) relationship. We estimated the daily dose of wildfire smoke $PM_{2.5}$ from measured concentrations of PM_4, estimated wildland firefighter breathing rates, daily shift duration (hours per day) and frequency of exposure (fire days per year and career duration). Firefighters who worked 49 days per year were exposed to a daily dose of PM_4 that ranged from 0.15 mg to 0.74 mg for a 5- and 25-year career, respectively. The daily dose for firefighters working 98 days per year of PM_4 ranged from 0.30 mg to 1.49 mg. Across all exposure scenarios (49 and 98 fire days per year) and career durations (5–25 years), we estimated that wildland firefighters were at an increased risk of LC (8 percent to 43 percent) and CVD (16 percent to 30 percent) mortality. This unique approach assessed long term health risks for wildland firefighters and demonstrated that wildland firefighters have an increased risk of lung cancer and cardiovascular disease mortality.

1. Introduction

Wildland firefighters suppress thousands of wildfires each year that burn across millions of acres in the U.S. (NIFC, 2017a). During 2017, more than 8.8 million acres burned and more than 26,000 wildland firefighters worked to suppress fire during the height of the summer wildfire season (NIFC, 2017a; NIFC, 2017b). Large forest fires in the western U.S. are nearly five times as frequent annually as they were in 1990 (NIFC, 2017a). These fires burn more land area and last much longer than in the past. The wildfire season is also much longer, as exemplified by the Thomas Fire near Santa Barbara, which became (on December 22, 2017) California's largest wildfire in modern history. The wildfire season in California typically ends in October, when the autumn rains begin (InciWeb the Incident Info, 2018).

Wildland firefighters are exposed to inhalation health hazards including hazardous air pollutants from the combustion of vegetative live and dead biomass (smoke) and the breathing of soil dust, while working long work shifts with no respiratory protection (Broyles, 2013; Naeher et al., 2007). Along with exposure to smoke on the fire line, wildland firefighters may be exposed to smoke at incident command posts (ICP) situated near the wildfire to support suppression operations (McNamara et al., 2012). Wildland firefighters conducting prescribed fires (intentionally ignited, low-intensity fires used for land management) are also exposed to smoke (Ryan et al., 2013). Wildfire smoke is a complex

* Corresponding author. National Technology and Development Program, US Forest Service, USDA, 5785 Highway 10 W, Missoula, MT 59808, USA.
E-mail address: joseph.domitrovich@usda.gov (J.W. Domitrovich).

https://doi.org/10.1016/j.envres.2019.03.060
Received 12 November 2018; Received in revised form 18 March 2019; Accepted 24 March 2019
Available online 26 March 2019
0013-9351/

mixture of gas and particle-phase air contaminants, including acrolein, benzene, carbon dioxide, carbon monoxide, formaldehyde, polycyclic aromatic hydrocarbons, and fine and respirable particulate matter (PM with aerodynamic diameters ≤ 2.5 μm or ≤ 4 μm, respectively), which can contain amorphous carbon or soot (Naeher et al., 2007; Adetona et al., 2016). Soil disturbance from several work activities including fire line construction, mop-up, and open vehicle transportation, also exposes firefighters to mineral contaminants such as crystalline silica (Broyles, 2013).

Previous health studies of wildland firefighters examined acute health effects of smoke exposure across individual shifts and entire fire seasons. Liu et al., 1992, found that 63 wildland firefighters in California had significant declines of individual lung function (FVC, FEV_1, and FEF_{25-75}) and an increase in airway responsiveness post-season when compared with their pre-season baseline values (Liu et al., 1992). When examining cross-shift changes in lung function, Gaughan et al., 2014a, b, reported that wildland firefighters had a significant decline in lung function associated with high exposure to levoglucosan (a tracer for smoke from wood or vegetation combustion) (Gaughan et al., 2014a). Additionally, Adetona et al. (2017), Hejl et al. (2013), and Swiston et al. (2008), reported increased levels of biomarkers of systemic inflammation in firefighters after wildland fires and prescribed burns (Adetona et al., 2017; Hejl et al., 2013; Swiston et al., 2008). Booze et al. (2004), conducted a health risk assessment to characterize the risk of cancer and non-cancer health effects in wildland firefighters (Booze et al., 2004). The study concluded that there were elevated risks of developing cancer, primarily from exposure to benzene and formaldehyde, as well as non-cancer health effects from exposure to $PM_{3.5}$ and acrolein. Recently, Semmens et al. (2016), conducted the first long-term health survey of wildland firefighters that examined the association between the duration of wildland firefighters' careers and self-reported health outcomes (Semmens et al., 2016). The survey reported significant associations between the number of years worked as a wildland firefighter and history of ever diagnosis of two cardiovascular measures - hypertension and/or heart arrhythmia, as well as the need for knee surgery.

The U.S. Environmental Protection Agency regulates fine particulate matter ($PM_{2.5}$) because there is robust epidemiologic evidence of associations between short-term exposures to $PM_{2.5}$ and cardiopulmonary mortality, as well as increased risk of acute cardiovascular outcomes, including myocardial infarction, stroke, and arrhythmias (Atkinson et al., 2015). Along with the epidemiological evidence, experimental evidence from both animal and human studies supports the associations between exposure to $PM_{2.5}$ and cardiovascular outcomes (Brook et al., 2017). Risk of lung cancer is also associated with exposure to ambient $PM_{2.5}$ (Hamra et al., 2014). The recently reported results of a large U.S. cohort study of older individuals (nearly 19 million Medicare beneficiaries) showed increased risks of both cardiovascular mortality and lung cancer with increased $PM_{2.5}$ exposure (Pun et al., 2017).

In our study, we conducted an analysis to examine long-term health impacts for wildland firefighters. Our objective was to estimate relative risk of lung cancer and cardiovascular disease mortality from existing PM exposure-response relationships using a measured PM concentration from smoke and breathing rates from wildland firefighter field studies across different exposure scenarios.

2. Methods

Wildfire Smoke Exposure-Response Relationship. To estimate the relative risk of lung cancer (LC) and cardiovascular disease (CVD) mortality from exposure to $PM_{2.5}$ from smoke, we used the exposure-response (ER) relationships developed by Pope III et al. (2011); Pope et al., 2011. Briefly, they conducted a cohort study analysis from the American Cancer Society (ACS) Cancer Prevention Study II, which included 1.2 million adults, to examine the shape of the exposure-response relationships of $PM_{2.5}$ from ambient air pollution and cigarette smoke with lung cancer and cardiovascular (including ischemic heart disease and cardiopulmonary) mortality. They used the ACS data to estimate relative risks (RR) of LC and/or CVD mortality by increments of cigarette smoking and combined it with selected studies that reported RR from ambient air pollution and second-hand tobacco smoke exposure (estimated daily dose of $PM_{2.5}$) to quantify the exposure-response relationship using a power function with the form $[(RR = 1 + \alpha(dose)^{\beta})]$. For lung cancer, the fitted function reported by Pope III et al. (2011), was $[RR = 1 + 0.3195\ (dose)^{0.7433}]$ and for CVD the fitted function was $[RR = 1 + 0.2685\ (dose)^{0.2730}]$. We used these equations with to calculate disease risk for wildland firefighters.

Estimation of Daily Dose of $PM_{2.5}$. We estimated the daily dose of wildfire smoke $PM_{2.5}$ from measured concentrations of PM_4 (particulate matter with a median diameter of 4 μm), estimated wildland firefighter breathing rates, daily shift duration, and frequency of exposure. We estimated the daily dose across different frequency of exposure scenarios to examine varied days spent on wildfires each year and career length. We used equation (1) to estimate the lifetime daily dose of $PM_{2.5}$ from wildfire smoke for wildland firefighters. The occupational exposure data collected from wildland smoke was characterized based on an aerodynamic ratio of less than 4 μm. However, since pulmonary transport increases with a reduction in particulate size, the use of a relation based on $PM_{2.5}$ should be a close approximation for data based on PM_4 and at the very least, be more conservative. This allowed us to use the Pope III et al. (2011), ER curves. Data from combustion studies have demonstrated that the particle size of combustion-generated particles are on the order of 300 nm (Kleeman et al., 1999; McMeeking et al., 2005). Thus, even though conventional occupational PM samples collect particles with an aerodynamic cut size of 3.5–4 μm, most PM from wildfire smoke exposure is composed of submicron particles, much smaller than $PM_{2.5}$. McMeeking et al. (2005), used an optical particle counter and a differential mobility analyzer to examine the size distribution of particles from wildfire smoke measured during an aerosol study in Yosemite National Park (McMeeking et al., 2005). That study concluded that mass median aerodynamic particle diameter (MMAD) was about 300 nm and volume geometric mean diameters ranged from about 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. Kleeman et al. (1999), measured (under laboratory conditions) the particle sizes of smoke aerosol from several different types of wood and reported that particles ranged from about 90 to about 300 nm in MMAD and that smoke from conventional cigarettes ranged from 300 to 400 nm (Kleeman et al., 1999). Lastly, Leonard et al., 2007, collected aerodynamically size-selected aerosol samples at a wildfire in Alaska to examine particle size and reported that approximately 78 percent of the total mass concentration was from collected particles with a mean diameter of 2.4 μm (Leonard et al., 2007).

$$\begin{aligned} Daily\ dose\ PM_4(mg) &= Exposure\ Concentration\left(\frac{mg}{m^3}\right) \\ &\times Daily\ Shift\ Duration\left(\frac{hrs}{shift}\right) \\ &\times Breathing\ Rate\left(\frac{L}{min}\right) \times F \times CF \end{aligned}$$

$$\text{CF}-\text{Conversion Factors}\left(\frac{60\ min}{hr}\right) \text{and} \left(\frac{m^3}{1000\ liters}\right)$$

$$\text{F}-\text{Frequency of exposure} = \left(\frac{shift\ days\ per\ year}{365\ days\ per\ year} \times \frac{years\ of\ firefighting\ career}{45\ years}\right) \quad (1)$$

From 2009 to 2012, the U. S. Department of Agriculture, Forest Service (USFS), National Technology and Development Program (NTDP), conducted an extensive field study that collected breathing zone measurements of occupational exposure to carbon monoxide, PM_4, and crystalline silica at wildfire incidents across the U.S., including Alaska (Broyles, 2013). From 2010 to 2011, a NTDP trained field research team measured wildland firefighter exposure to PM_4 across 80 wildland firefighters on different fire crew types performing various

suppression tasks on wildfires. The research team consisted of wildland firefighters trained by NTDP researchers to collect exposure data and perform direct observation methodologies. The ability of these trained individuals to function amid wildfire suppression activities enabled direct observation of research subjects throughout their respective work shifts without compromising safety or performance.

The research team directly observed each subject for the duration of the work shift, from the moment the subject was equipped with data recording devices before breakfast (typically at 6:00 a.m.) until the conclusion of the work shift. During sample collection, NTDP field observers also recorded information on work shift duration and used the average hours worked, which we used to calculate the daily dose of PM_4 Capturing the shift duration for wildland firefighters was important because individuals working on wildfires typically work more than an 8-h day.

The research team collected PM_4 through methods that are generally consistent with National Institute of Occupational Safety and Health (NIOSH) Method 0600, with analysis of crystalline silica content using NIOSH Method 7500 (NIOSH, 1998; NIOSH, 2003). The NTDP research team attached pre-weighed, 37-mm diameter polyvinyl chloride (PVC) filters with 1 μm pore size in 3-piece cassettes to BGI SCC 1.062 Triplex cyclone, constructed of aluminum to minimize wall losses from electrostatic effects. SKC AirChek pumps drew air through the cyclone and cassette at a target flow rate of just over 1 L per minute. A commercial laboratory accredited by the American Industrial Hygiene Association in their Industrial Hygiene Laboratory Accreditation Program (RJ Lee Group, Monroeville, PA) obtained and later analyzed the pre-weighed filters. After sampling, all filters were capped and transported under chain of custody to the laboratory, accompanied by field blanks prepared each day. Throughout the sampling campaign, we collected 20–100% frequency of field blanks of total samples collected each day. Lab results indicated that there was not net blank mass to subtract from the net sample mass. In addition, field replicates were collected to identify any potential equipment or data collection errors. Sampling pumps were calibrated on site with a primary standard frictionless piston dry calibrator (BIOS DC-Lite) before sampling, and checked again after sampling, using the cyclone calibration adapter provided by the manufacturer. Sampling pumps were placed inside each firefighter's gear pack and cassettes were placed on the shoulder straps of each firefighter's gear pack, as close to the breathing zone as possible. Additionally, the research team placed active sampling equipment at a central location at ICPs or spike camps (satellite camps closer to the wildfire) at most wildfire events to examine off-fire-line exposures.

Shift exposure concentrations were calculated for each firefighter as a time weighted average (TWA) that included exposure on the fire line and at the ICPs or spike camps, and while traveling to and from the wildfire. If ICP or spike camp data showed that the firefighters were exposed even when they were not on the fire line, this was included in the shift average. Summary statistics were calculated in the R System for Statistical Computing (Version 2.13.2), using the package *sand* Version 1.5 which allowed for nonparametric analyses for data with a large number of non-detect concentration values (Frome and Frome; Frome, 2005).

The data from personal PM_4 air samples collected on firefighters during typical firefighting activities would contain some contribution from soil sources that may include crystalline silica, such as dust from hiking on trails and constructed fire line. This could bias our estimates of PM_4 daily dose and could impact the calculated smoke-related health risks. To better estimate the amount of $PM_{2.5}$ from smoke, we subtracted out the respirable crystalline silica, which we used as a surrogate for the soil dust contribution to the measured PM_4 concentrations, yielding our best estimate of the exposure due only to smoke. Crystalline silica samples were analyzed using NIOSH 7500 method which uses x-ray powder diffraction).

We calculated wildland firefighter breathing rates (BR; L air inhaled per minute) from field-measured heart rates while firefighters performed wildland fire operations. A trained NTDP field research team following the protocols described above for smoke data collection collected heart rate measurements during the fire season (May through September) in the western U.S. from 2013 to 2015. The morning of each trial, two to three subjects from a fire crew were fitted with a Hidalgo Equivital™ Physiological Monitor (Equivital, UK) to record heart rate (HR). The NTDP field research team directly observed each subject for the duration of his or her work shift and collected minute-by-minute observations and information on each subject's job task and the physiologic response for each specific task performed. Breathing rate was calculated using HR across the main job tasks performed by wildland firefighters using regression equations developed by Valli et al., 2013) to estimate BR from HR. BR was first calculated for each individual job task and then averaged across all tasks generally performed by a firefighter each day (Valli et al., 2013).

The number of days spent on wildfire assignments per fire season can vary greatly from year to year, and we found no good data source for this information. Additionally, wildland firefighter career length is not well reported in the literature. For this reason, we developed a few different scenarios based on various frequencies of exposure to calculate daily dose of PM_4 using varied days spent on wildfire assignments and career duration. These scenarios were based on the expert opinion of the research team and conversations with wildland firefighters during field studies. According to the Interagency Standards for Fire and Fire Aviation Operations, the standard wildfire assignment is 14 days long; which we used as a guide to estimate days spent on fire assignments (NIFC, 2017c). Additionally, we used the common goal of working 1000 h of overtime for wildland firefighters per fire season to estimate the number of 14-day assignments completed. For wildland firefighters, we estimated a “firefighter long season” to be 98 days spent on fire assignments (equivalent to seven 14-day assignments) and a “firefighter short season” to be 49 days spent on fire assignments (equivalent to three and a half 14-day assignments). We calculated frequency of exposure using 5, 10, 15, 20, and 25 years for wildland firefighter career duration. Lastly, we adjusted career duration by 45 years, which is the average working career of an individual, according to the Occupational Safety and Health Administration (OSHA, 2016).

3. Results

Table 1 presents the parameters we used to calculate daily dose of PM_4 for the two exposure scenarios: (1) firefighter long season and (2) firefighter short season. Based on field study observations, firefighters worked an average of 13.6 h per shift. The mean concentrations of PM_4 and crystalline silica measured on wildland firefighters was $0.53\ mg\ m^{-3}$ and $0.026\ mg\ m^{-3}$, respectively. After adjusting the measured shift average concentration of PM_4 for dust exposure (crystalline silica), wildland firefighters were exposed to a mean concentration $0.51\ mg\ m^{-3}$ of PM_4 due to smoke exposure, with the 95th percentile shift exposure of $0.64\ mg\ m^{-3}$.

Based on measured HR, we calculated the firefighter breathing rate to be 24-L min^{-1} while conducting suppression operations on the fire line. For firefighters, we used measured HR data collected while they performed common work tasks during fire suppression operations, including: hiking, fire line construction, holding, mop-up, lighting, operating a pump, and standing by to receive work assignments (staging). Firefighters can perform fire line construction near the fire's edge (direct) or at a distance (indirect). Fire line construction involves clearing vegetation (often with a chain saw) and digging or scraping down to mineral soil with hand tools to create a break in fuels to stop the spread of a fire. Holding requires firefighters to walk along the active fire to ensure that it has not crossed the fire line. After the fire has burned through an area, firefighters will mop-up by digging out or applying water to extinguish any smoldering material. Another suppression tactic includes lighting, which involves using torches filled with a 3:2

Table 1
Parameters used to Calculate Daily Dose of PM_4 for Each Exposure Scenario and Relative Risk for Lung Cancer and Cardiovascular Disease.

Exposure Scenario	Shift Duration (hours)	Shift Exposure (mg/m^3)		Breathing Rate (LPM)	Fire Days (Days/Year)	Career Duration	PM_4 Daily Dose (mg)	Lung Cancer	CVD
		Mean	95th Percentile				Mean[a] (95th PCTL)	RR[b]	RR[b]
Firefighter Short Season	13.6	0.51	0.64	24	49	5	0.15 (0.19)	1.08 (1.09)	1.16 (1.17)
						10	0.30 (0.37)	1.13 (1.15)	1.19 (1.21)
						15	0.45 (0.56)	1.18 (1.21)	1.22 (1.23)
						20	0.60 (0.75)	1.22 (1.26)	1.23 (1.25)
						25	0.74 (0.93)	1.26 (1.30)	1.25 (1.26)
Firefighter Long Season					98	5	0.30 (0.37)	1.13 (1.15)	1.19 (1.21)
						10	0.60 (0.75)	1.22 (1.26)	1.23 (1.25)
						15	0.89 (1.12)	1.29 (1.35)	1.26 (1.28)
						20	1.19 (1.50)	1.36 (1.43)	1.28 (1.30)
						25	1.49 (1.87)	1.43 (1.51)	1.30 (1.32)

[a] Daily dose was calculated using the mean and 95th percentile shift exposure concentration.
[b] Relative Risk was calculated using the mean and 95th percentile PM_4 Daily Dose.

diesel/unleaded gasoline mixture to burn any unburned fuel to create a fuel break. Pump operators manage gas or diesel pumps that provide water to firefighters.

For wildland firefighters, as frequency of exposure, career duration and days on fire assignment each year (fire days), increased, the daily dose of PM_4 also increased (Table 1). Even though exposure concentration, shift duration, and breathing rate remained static for each exposure scenario, the frequency of exposure increased which resulted in an increase of daily dose of PM4 across exposure scenarios. Firefighters who worked a short fire season (49 days) were exposed to a daily dose of PM_4 that ranged from 0.15 mg for a 5-year career to 0.74 mg for a 25-year career, respectively. Daily dose of PM_4 ranged from 0.30 mg to 1.49 mg for firefighters who worked 5–25 years, respectively, for a long fire season (98 days).

Across all exposure scenarios and career durations, the calculated relative risk for LC and CVD was greater than 1, indicating an increased risk of mortality from LC and CVD from smoke exposure (Fig. 1). Risk of LC ranged from 1.08 to 1.26 for short-season firefighters and 1.13 to 1.43 for long-season firefighters across 5- to 25-year careers. Risk of CVD ranged from 1.16 to 1.25 for short-season firefighters and 1.19 to 1.30 for long-season firefighters across careers that spanned 5–25 years. For both firefighter exposure scenarios, the risk of LC steadily rose as career length increased, while the risk of CVD increased sharply for firefighters with 5- to 15-year careers and increased slightly over 20- and 25-year careers.

4. Discussion

Our study objectives were to estimate lifetime risk of lung cancer and cardiovascular mortality from exposure to PM_4 from smoke. Our analysis used measured PM_4 concentration from smoke and estimated breathing rates that we collected as part of extensive field studies of wildland firefighters in the U.S. Using the $PM_{2.5}$ exposure-response relationships developed by Pope III et al. (2011), we estimated that wildland firefighters had an increased risk of LC and CVD mortality, with RRs greater than 1 across all exposure scenarios and career durations.

The measured mean shift TWA PM_4 exposures (after correcting for soil-derived respirable particulate contribution using the measured concentrations of quartz) from the NTDP field study used for this analysis were similar (Mean: 0.51 $mg\,m^{-3}$; 95th percentile 0.64 $mg\,m^{-3}$) to previous firefighter PM2.5 exposure concentrations measured at prescribed burns and similar to or within the range of concentrations reported by others for wildfire incidents (Adetona et al., 2011; Materna et al., 1992; Reinhardt and Ottmar, 2004). After excluding soil dust contribution using the measured concentrations of quartz (averaging 4% of the mass), the remaining PM_4 that we measured is expected to be mostly of smal aerodynamic size (averaging

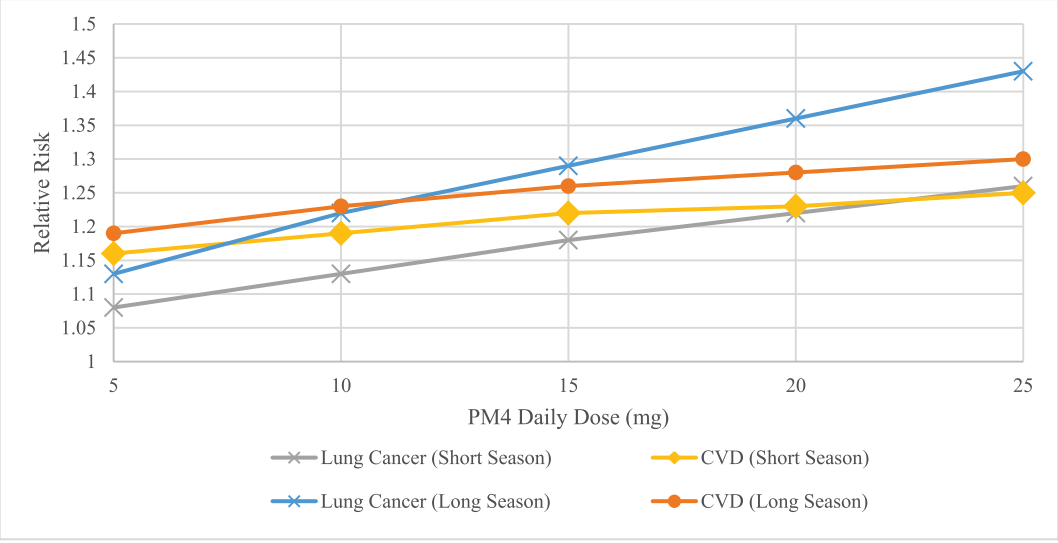


Fig. 1. Relative risk of lung cancer and cardiovascular disease across career length.

approximately 300 nm in diameter), as reported by others (McMeeking et al., 2005; Chakrabarty et al., 2006). Leonard et al. (2007) reported 36% of their mass concentration had an MMAD at 4.2 μm or above, and 64% of the total mass concentration was from collected particles with a mean diameter of 2.4 μm or below (Leonard et al., 2007). Those authors did not grease their collection substrates and noted that their long sampling times may bias their results due to particle bounce. McMahon and Bush reported that operating a Dorr-Oliver nylon cyclone at $4\,L\,min^{-1}$ to measure an MMAD of PM2.3 only captured 88% of the weight concentration of PM3.5 in open burning experiments with forest biomass in a greenhouse, but it is not clear that these tests would be representative of actual exposures in ambient conditions affecting particle removal and agglomeration rates at an actual wildfire (McMahon and Bush, 1992). Reh et al., 1994, measured respirable particulate matter on six firefighters over 2 days in Yosemite National Park and reported concentrations ranging from 0.60 to $1.7\,mg\,m^{-3}$ (Reh et al., 1994). Although occupational health studies generally report personal concentrations as geometric means, we were able to use the geometric mean and geometric standard deviation to calculate arithmetic mean values to compare with our mean shift concentrations. Booze et al. (2004), used $PM_{3.5}$ concentrations measured on the firefighters by Reinhardt and Ottmar (2000) at western wildfires to calculate non-cancer hazard indices (Booze et al., 2004; Reinhardt and Ottmar, 2000). Reinhardt and Ottmar reported mean concentrations of $PM_{3.5}$ of $0.79\,mg\,m^{-3}$ over the entire work shift and $1.13\,mg\,m^{-3}$ for fire line only exposure. Gaughan et al. (2014a,b), reported mean concentration of respirable particles collected by cyclones to be 0.77 and $0.80\,mg\,m^{-3}$ for firefighters performing fire line construction and mop-up, respectively (Gaughan et al., 2014a). Our lower measured mean shift TWA exposures could have resulted in an underestimation of risk, however, our estimate is based on a larger sample size of 80 samples at project wildfires.

Our measured heart rates and estimated breathing rate were lower than one would expect for most individuals presumably performing reasonably heavy labor and were also lower than previous studies measuring breathing rate of wildland firefighters in the field and laboratory over shorter periods of time. However, firefighters are expected to work at consistent exertion levels for the duration of a shift and they are conditioned to be able to perform under the conditions of the job. Cuddy et al. (2015), reported that, based on measured heart rate, wildland firefighters do not experience high levels of cardiovascular strain (Cuddy et al., 2015). By comparison, our estimated breathing rate was comparable to those measured for trained athletes performing exercise at a relatively mild level (e.g., walking on a treadmill at $10.8\,km\,h^{-1}$) (Sracic, 2016). Brotherhood et al. (1997), measured breathing rates of four crews constructing fire line for 7-min periods at various work rates and reported mean breathing rates from 41 to $100\,l\,min^{-1}$ (Brotherhood et al., 1997). In the laboratory, eight male wildland firefighters had a mean breathing rate of $48.4\,l\,min^{-1}$ while performing endurance exercises (45–60 min) three times per week to test for physiological differences from various types of personal protective clothing (Carballo-Leyenda et al., 2017). Past estimates of inhalation doses during wildland firefighting used breathing rates that ranged from 21.6 to $60\,l\,min^{-1}$. Booze et al. (2004), used breathing rates of 40 and $60\,l\,min^{-1}$ to calculate inhalation doses for their screening-level health risk assessment. When calculating inhalation doses for exposure to radionuclides, Viner et al. (2018), used a breathing rate of $21.6\,l\,min^{-1}$ for industrial workers to estimate firefighter cumulative dose. The estimated breathing rate we used was lower than previously reported measurements, it led to underestimation for daily dose and our overall risk calculation.

For this analysis, we developed wildland firefighter exposure scenarios that accounted for days spent working on wildfires and career duration. Although we based these scenarios on the expert opinion of the research team and conversations with wildland firefighters during field studies, they are similar to past wildland firefighter studies. Semmens et al. (2016), surveyed 545 wildland firefighters (499 completed surveys) about occupational history and self-reported health outcomes and reported that 35 percent of survey respondents had careers that lasted less than 10 years, 33 percent had careers of 10–19 years, and 32 percent had careers of more than 20 years and a mean career duration of 17 years (Semmens et al., 2016). The number of days spent firefighting that we selected for our analysis were within the range of days used by Booze et al. (2004), for Type I wildland fire crews (67 days). However, they were much higher than Type II crews (10 days). The Type I data came from the Northwest Regional Coordinating Center for 1990 to 1994, and the Type II data came from a database from the Okanogan National Forest, which may not have been representative of wildland firefighters across the U.S. The shift durations that NTDP recorded and used to calculate daily dose were similar to past exposure assessments of wildland firefighters. Gaughan et al. (2014a,b), reported a mean shift duration of 12 h and Reinhardt and Ottmar (2000) reported a mean shift duration of 10.4 h (range: 0–24 h) (Gaughan et al., 2014a; Reinhardt and Ottmar, 2000). The varying career durations that we used for our risk calculations seemed reasonable and representative of a wildland firefighter's career and were comparable to previous studies of wildland firefighters.

In the context of the $PM_{2.5}$ exposure-response (ER) curves of Pope III et al. (2011), the daily dose of PM_4 that we calculated for wildland firefighters was in the range of the daily dose of $PM_{2.5}$ from exposure to ambient air pollution and secondhand cigarette smoke. Even after we adjusted the daily dose of PM for firefighters with the frequency of exposure to account for fire days and career length. Through the use of the Pope III et al. (2011) ER curves, we observed that the risk for lung cancer mortality increased nearly linearly with exposures over time and is more strongly influenced by exposure duration than is the risk of death from CVD. However, the risk of cardiovascular mortality rises steeply for doses in the range we estimated for firefighter exposures but flattens out at higher doses. Short-season firefighters that had 5- to 10-year careers and long-season firefighters with a 5-year career had daily doses of PM_4 in the ambient air pollution range. Short-season firefighters with a 15- to 25-year career and long-season firefighters with a 15-year career had daily doses of PM_4 in the secondhand cigarette smoke range; while long-season firefighters with 20- to 25-year careers had higher doses, these doses were still lower than daily doses of $PM_{2.5}$ for active cigarette smokers. Additionally, our calculated range of daily dose of PM_4 for wildland firefighters was similar to the mass intake (equivalent metric to daily dose) of $PM_{2.5}$ from the 2013 Rim Fire in California (Navarro et al., 2016). In Tuolumne County (where the Rim Fire occurred), which was most impacted by smoke, mass intake of $PM_{2.5}$ averaged 0.49 mg per day with a maximum of 1.31 mg per day across the most active 10-day fire period. This comparison demonstrates that a wildland firefighter's inhalation dose of smoke can be similar to communities that are highly impacted by wildfires. However, a wildland firefighter is exposed to these concentrations of smoke for longer periods and not just for a span of a few days or weeks during a single wildfire event.

Although mortality has not been well studied in wildland firefighters, there is evidence of excess mortality among structural firefighters. Baris et al., 2001, observed statistically significant excess risks of ischemic heart disease (SMR = 1.09) among a retrospective cohort of 7789 Philadelphia firefighters (Baris et al., 2001). Daniels et al. (2014, 2015), observed statistically significant positive associations between fire-hours and both leukemia and lung cancer mortality; the relationship between lung cancer and cumulative exposure was nearly linear (Daniels et al., 2014, 2015). Lastly, female firefighters in Florida had increased risk of death from atherosclerotic heart disease when compared with the general Florida population (SMR = 3.85; 95% CI: 1.66 to 7.58) (Ma et al., 2005).

Previous health assessments of wildland firefighters found evidence of arterial stiffness and inflammation after exposure to smoke, which are important to the development of cardiovascular disease (Adetona

et al., 2017; Navarro et al., 2016). Gaughan et al. (2014b), reported that arterial stiffness (measured as mean augmentation index percent) was higher for firefighters from a Type 1 interagency hotshot crew (IHC) exposed to wildfire smoke 4 days before testing when compared with a different Type 1 IHC crew that was not exposed to smoke (Gaughan et al., 2014b). Additionally, the researchers stated that mean augmentation index percent was higher in firefighters with a higher oxidative stress score, which was positively associated with higher levoglucosan concentrations (an indicator of wood smoke). A recent study of wildland firefighters performing holding and lighting at prescribed burns reported that firefighters performing lighting had significantly higher increases in three pro-inflammatory mediators (interleukin-8, C-reactive protein, and serum amyloid A) across their work shifts when compared with firefighters performing holding (Adetona et al., 2017). The researchers did not find an association between estimated $PM_{2.5}$ dose and biomarkers and noted that the biomarker difference in lighters and holders could have been due to exposure to the combustion of the diesel/gasoline mixture used by lighters to ignite prescribed fires. Although we based our analysis on exposure to PM_4 from smoke, the firefighters measured for the NTDP field study did perform lighting duties on wildfires and were also exposed to the combustion of the diesel/gasoline mixture.

Past health assessments of wildland firefighters recommended the need to conduct long-term studies. However, given the seasonal employment and relatively short-term tenures of many wildland firefighters, such studies are difficult to conduct. Semmens et al., 2016, examined long-term health impacts, but only surveyed currently employed wildland firefighters (Semmens et al., 2016). Future long-term health assessments should also target retired wildland firefighters to examine health status later in life. Our analysis provided a unique approach to assess long-term mortality risks for two specific diseases using calculated inhalation doses of PM_4 from smoke that we estimated from measurements collected during extensive field studies. However, there are some limitations to consider when interpreting our results. First, because we used the Pope III et al. (2011), curves to estimate RR for specific disease outcomes, we were unable to provide any confidence values in the calculated RR values. Additionally, because the ER curves have a steep relationship at lower exposures, this approach did not lend itself to back-calculating a threshold exposure for an elevated RR. Second, we assumed PM_4 from most fires (after correcting for coarse-mode contribution using the measured concentrations of quartz) to be equivalent in size, toxicity, and carcinogenicity to $PM_{2.5}$ from tobacco smoke and ambient air pollution, so that we could use the ER curves from Pope III et al. (2011), to estimate RR. Next, we based the long-term risks associated with exposures over a working life on assumptions about exposure concentration, exposure frequency and duration to determine a daily dose. Over an individual firefighter's career, he or she will serve in many tasks and activities that may influence these assumptions. Thus, a weighted average of exposures over the course of a career, possibly by job title, could potentially provide a more realistic measure of exposure for a wildland firefighter. Additionally, our current data did not account for any smoke exposure from prescribed fires and we were unable to use our current data sources to estimate risk for individuals working in ICPs as part of an incident management team. In future analyses, one should adjust the daily doses by the demographics and other characteristics that may impact exposure or any underlying disease risk. Also, our analysis only considered the size and concentration of PM across exposures to wood smoke, ambient air pollution, and cigarette smoke, and we did not address any of the differences in chemical composition of the PM from these sources. In future analyses, characterizing the toxicity and health risk of the different chemical components of wildfire smoke would be ideal.

Our study demonstrated that wildland firefighters are at an increased risk for the development of lung cancer and cardiovascular disease. This risk increases with an increase in career duration and days spent on wildfire incidents each fire season. Over the last 25 years, there have been recommendations to reduce exposure to smoke by minimizing mop-up where appropriate on a fire line and rotating firefighters in and out of heavy smoke situations throughout a work shift, develop a medical surveillance program and occupational exposure limits specific to wildfires, and increase wildland firefighter training on the hazards of smoke. Currently, it is unclear if these recommendations would reduce exposure to smoke enough to reduce health risks. We believe that firefighters should reduce exposure to smoke in any way possible. Exposure to wildland smoke underlies virtually every aspect of risk management and must be addressed effectively in order to assure risk management decisions are sound and safe. It is essential that sound smoke exposure mitigation strategies be developed, implemented, and enforced.

Declaration of competing financial interests (CFI)

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

Acknowledgements

This work was funded by the Joint Fire Science Program (Project ID: 13-1-02-14).

References

Adetona, O., Dunn, K., Hall, D.B., Achtemeier, G., Stock, A., Naeher, L.P., 2011. Personal PM(2.5) exposure among wildland firefighters working at prescribed forest burns in Southeastern United States. J. Occup. Environ. Hyg. 8, 503–511. https://doi.org/10.1080/15459624.2011.595257.

Adetona, O., Reinhardt, T.E., Domitrovich, J., et al., 2016. Review of the health effects of wildland fire smoke on wildland firefighters and the public. Inhal. Toxicol. 28, 95–139. https://doi.org/10.3109/08958378.2016.1145771.

Adetona, A.M., Adetona, O., Gogal, R.M., et al., 2017. Impact of work task-related acute occupational smoke exposures on select proinflammatory immune parameters in wildland firefighters. J. Occup. Environ. Med. 59, 679–690. https://doi.org/10.1097/JOM.0000000000001053.

Atkinson, R.W., Mills, I.C., Walton, H.A., et al., 2015. Fine particle components and health–a systematic review and meta-analysis of epidemiological time series studies of daily mortality and hospital admissions. J. Expo. Sci. Environ. Epidemiol. 25, 208–214. https://doi.org/10.1038/jes.2014.63.

Baris, D., Garrity, T.J., Telles, J.L., et al., 2001. Cohort mortality study of Philadelphia firefighters. Am. J. Ind. Med. 39, 463–476.

Booze, T.F., Reinhardt, T.E., Quiring, S.J., et al., 2004. A screening-level assessment of the health risks of chronic smoke exposure for wildland firefighters. J. Occup. Environ. Hyg. 1, 296–305. https://doi.org/10.1080/15459620490442500.

Brook, R.D., Newby, D.E., Rajagopalan, S., 2017. Air pollution and cardiometabolic disease: an update and call for clinical trials. Am. J. Hypertens. 31, 1–10. https://doi.org/10.1093/ajh/hpx109.

Brotherhood, J.R., Budd, G.M., Hendrie, A.L., et al., 1997. Project aquarius 3. Effects of work rate on the productivity, energy expenditure, and physiological responses of men building fireline with a rakehoe in dry eucalypt forest. Int. J. Wildland Fire 7, 87–98. https://doi.org/10.1071/wf9970087.

Broyles, G., 2013. Wildland Firefighter Smoke Exposure. US Forest Service. http://www.fs.fed.us/t-d/pubs/pdfpubs/pdf13511803/pdf13511803dpi100.pdf, Accessed date: 18 April 2016.

Carballo-Leyenda, B., Villa, J.G., López-Satué, J., et al., 2017. Impact of different personal protective clothing on wildland firefighters' physiological strain. Front. Physiol. 8. https://doi.org/10.3389/fphys.2017.00618.

Chakrabarty, R.K., Moosmüller, H., Garro, M.A., Arnott, W.P., Walker, J., Susott, R.A., Babbitt, R.E., Wold, C.E., Lincoln, E.N., Hao, W.M., 2006. Emissions from the laboratory combustion of wildland fuels: particle morphology and size. J. Geophys. Res. 111. https://doi.org/10.1029/2005JD006659.

Cuddy, J.S., Sol, J.A., Hailes, W.S., et al., 2015. Work patterns dictate energy demands and thermal strain during wildland firefighting. Wilderness Environ. Med. 26, 221–226. https://doi.org/10.1016/j.wem.2014.12.010.

Daniels, R.D., Kubale, T.L., Yiin, J.H., et al., 2014. Mortality and cancer incidence in a pooled cohort of US firefighters from San Francisco, Chicago and Philadelphia (1950-2009). Occup. Environ. Med. 71, 388–397. https://doi.org/10.1136/oemed-2013-101662.

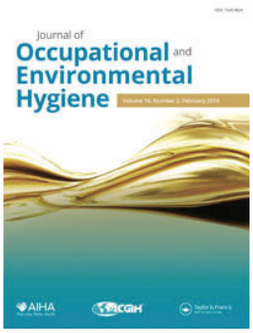
Daniels, R.D., Bertke, S., Dahm, M.M., et al., 2015. Exposure-response relationships for select cancer and non-cancer health outcomes in a cohort of U.S. firefighters from San Francisco, Chicago and Philadelphia (1950-2009). Occup. Environ. Med. 72, 699–706. https://doi.org/10.1136/oemed-2014-102671.

Frome, E.L., 2005. Statistical Methods and Software for the Analysis of Occupational Exposure Data with Non-detectable Values. ORNL.

Frome EL, Frome DP. Sand - Statistical Analysis of Non-detect Data.

Gaughan, D.M., Piacitelli, C.A., Chen, B.T., et al., 2014a. Exposures and cross-shift lung

function declines in wildland firefighters. J. Occup. Environ. Hyg. 11, 591–603. https://doi.org/10.1080/15459624.2014.895372.
Gaughan, D.M., Siegel, P.D., Hughes, M.D., et al., 2014b. Arterial stiffness, oxidative stress, and smoke exposure in wildland firefighters. Am. J. Ind. Med. 57, 748–756. https://doi.org/10.1002/ajim.22331.
Hamra, G.B., Guha, N., Cohen, A., et al., 2014. Outdoor particulate matter exposure and lung cancer: a systematic review and meta-analysis. Environ. Health Perspect. 122, 906–911. https://doi.org/10.1289/ehp.1408092.
Hejl, A.M., Adetona, O., Diaz-Sanchez, D., et al., 2013. Inflammatory effects of woodsmoke exposure among wildland firefighters working at prescribed burns at the Savannah River Site, SC. J. Occup. Environ. Hyg. 10, 173–180. https://doi.org/10.1080/15459624.2012.760064.
InciWeb the Incident Information System: Thomas Fire. . https://inciweb.nwcg.gov/incident/5670/, Accessed date: 12 February 2018.
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. Environ. Sci. Technol. 33, 3516–3523. https://doi.org/10.1021/es981277q.
Leonard, S.S., Castranova, V., Chen, B.T., et al., 2007. Particle size-dependent radical generation from wildland fire smoke. Toxicology 236, 103–113. https://doi.org/10.1016/j.tox.2007.04.008.
Liu, D., Tager, I.B., Balmes, J.R., et al., 1992. The effect of smoke inhalation on lung function and airway responsiveness in wildland fire fighters. Am. Rev. Respir. Dis. 146, 1469–1473. https://doi.org/10.1164/ajrccm/146.6.1469.
Ma, F., Fleming, L.E., Lee, D.J., et al., 2005. Mortality in Florida professional firefighters, 1972 to 1999. Am. J. Ind. Med. 47, 509–517. https://doi.org/10.1002/ajim.20160.
Materna, B.L., Jones, J.R., Sutton, P.M., Rothman, N., Harrison, R.J., 1992. Occupational exposures in California wildland fire fighting. Am. Ind. Hyg. Assoc. J. 53, 69–76. https://doi.org/10.1080/15298669291359311.
McMahon, C.K., Bush, P.B., 1992. Forest worker exposure to airborne herbicide residues in smoke from prescribed fires in the southern United States. Am. Ind. Hyg. Assoc. J. 53, 265–272. https://doi.org/10.1080/15298669291359636.
McMeeking, G.R., Kreidenweis, S.M., Carrico, C.M., et al., 2005. Observations of smoke-influenced aerosol during the yosemite aerosol characterization study: size distributions and chemical composition. J. Geophys Res-Atmospheres 110 < Go to ISI > ://WOS:000229211500005.
McNamara, M.L., Semmens, E.O., Gaskill, S., et al., 2012. Base camp personnel exposure to particulate matter during wildland fire suppression activities. J. Occup. Environ. Hyg. 9, 149–156. https://doi.org/10.1080/15459624.2011.652934.
Naeher, L.P., Brauer, M., Lipsett, M., et al., 2007. Woodsmoke health effects: a review. Inhal. Toxicol. 19, 67–106. https://doi.org/10.1080/08958370600985875.
Navarro, K.M., Cisneros, R., O'Neill, S.M., et al., 2016. Air-quality impacts and intake fraction of PM2.5 during the 2013 Rim megafire. Environ. Sci. Technol. 50, 11965–11973. https://doi.org/10.1021/acs.est.6b02252.
NIFC, 2017a. National Interagency Fire Center Statistics. Natl. Fire News Year–Date Fires Acres. https://www.nifc.gov/fireInfo/fireInfo_stats_totalFires.html, Accessed date: 13 November 2017.
NIFC, 2017b. National Interagency Coordination Center Incident Management Situation Report Monday. September 4, 2017.
NIFC, 2017c. Interagency Standards for Fire and Fire Aviation Operations. Boise, ID. https://www.nifc.gov/policies/pol_intgncy_guides.html.
NIOSH, 1998. Particulates not otherwise regulated. Respirable. Method 0600. https://www.cdc.gov/niosh/docs/2003-154/pdfs/0600.pdf, Accessed date: 26 February 2018.
NIOSH, 2003. Silica, Crystalline, by XRD (Filter Redeposition). Method 7500. https://www.cdc.gov/niosh/docs/2003-154/pdfs/7500.pdf, Accessed date: 26 February 2018.
OSHA, 2016. Occupational Exposure to Respirable Crystalline Silica. https://www.federalregister.gov/documents/2016/03/25/2016-04800/occupational-exposure-to-respirable-crystalline-silica, Accessed date: 19 December 2017.
Pope III, C.A., Burnett, R.T., Turner, M.C., et al., 2011. Lung cancer and cardiovascular disease mortality associated with ambient air pollution and cigarette smoke: shape of the exposure–response relationships. Environ. Health Perspect. 119, 1616.
Pun, V.C., Kazemiparkouhi, F., Manjourides, J., et al., 2017. Long-term PM2.5 exposure and respiratory, cancer, and cardiovascular mortality in older US adults. Am. J. Epidemiol. 186, 961–969. https://doi.org/10.1093/aje/kwx166.
Reh, C.M., Letts, D., Scott, Deitchman, 1994. NIOSH Health Hazard Evaluation Report - HETA 90-0365-2415. US Department of Interior, National Park Service, Yosemite National Park, California. http://www.cdc.gov/niosh/hhe/reports/pdfs/1990-0365-2415.pdf, Accessed date: 20 April 2016.
Reinhardt, T.J., Ottmar, R.D., 2000. Smoke Exposure at Western Wildfires. U.S. Department of Agriculture, Forest Service, Pacific Northwest Research Station, Portland, OR.
Reinhardt, T.E., Ottmar, R.D., 2004. Baseline measurements of smoke exposure among wildland firefighters. J. Occup. Environ. Hyg. 1, 593–606. https://doi.org/10.1080/15459620490490101.
Ryan, K.C., Knapp, E.E., Varner, J.M., 2013. Prescribed fire in North American forests and woodlands: history, current practice, and challenges. Front. Ecol. Environ. 11, e15–24. https://doi.org/10.1890/120329.
Semmens, E.O., Domitrovich, J., Conway, K., et al., 2016. A cross-sectional survey of occupational history as a wildland firefighter and health. Am. J. Ind. Med. 59, 330–335. https://doi.org/10.1002/ajim.22566.
Sracic, M.K., 2016. Modeled regional airway deposition of inhaled particles in athletes at exertion. J. Aerosol Sci. 99, 54–63. https://doi.org/10.1016/j.jaerosci.2015.12.007.
Swiston, J.R., Davidson, W., Attridge, S., et al., 2008. Wood smoke exposure induces a pulmonary and systemic inflammatory response in firefighters. Eur. Respir. J. 32, 129–138. https://doi.org/10.1183/09031936.00097707.
Valli, G., Internullo, M., Ferrazza, A.M., et al., 2013. Minute ventilation and heart rate relationship for estimation of the ventilatory compensation point at high altitude: a pilot study. Extreme Physiol. Med. 2, 7. https://doi.org/10.1186/2046-7648-2-7.
Viner, B.J., Jannik, T., Hepworth, A., et al., 2018. Predicted cumulative dose to firefighters and the offsite public from natural and anthropogenic radionuclides in smoke from wildland fires at the Savannah River Site, South Carolina USA. J. Environ. Radioact. 182, 1–11. https://doi.org/10.1016/j.jenvrad.2017.10.017.



Taylor & Francis
Taylor & Francis Group

Journal of Occupational and Environmental Hygiene

ISSN: 1545-9624 (Print) 1545-9632 (Online) Journal homepage: https://www.tandfonline.com/loi/uoeh20

Factors affecting smoke and crystalline silica exposure among wildland firefighters

Timothy E. Reinhardt & George Broyles

To cite this article: Timothy E. Reinhardt & George Broyles (2019) Factors affecting smoke and crystalline silica exposure among wildland firefighters, Journal of Occupational and Environmental Hygiene, 16:2, 151-164, DOI: 10.1080/15459624.2018.1540873

To link to this article: https://doi.org/10.1080/15459624.2018.1540873

Accepted author version posted online: 08 Nov 2018.
Published online: 11 Mar 2019.

JOURNAL OF OCCUPATIONAL AND ENVIRONMENTAL HYGIENE
2019, VOL. 16, NO. 2, 151–164
https://doi.org/10.1080/15459624.2018.1540873

Taylor & Francis
Taylor & Francis Group

Factors affecting smoke and crystalline silica exposure among wildland firefighters

Timothy E. Reinhardt[a] and George Broyles[b]

[a]Wood Environment & Infrastructure Solutions, Inc., Seattle, Washington; [b]USDA Forest Service, Technology & Development, Boise, Idaho

ABSTRACT

Smoke exposure data among U.S. wildland firefighters for carbon monoxide, respirable particulate, and respirable crystalline silica are presented from a field surveillance program between 2009 and 2012. Models to predict fireline-average exposure to each inhalation hazard were developed and fit to the available data. The models identify factors to consider when defining similar exposure groups and designing future data collection. Task-based rather than shift-average data collection is important because the work activity representing the majority of fireline time, the position up- or downwind of the fire, and the proportion of time this combination represented were significant factors in the model for carbon monoxide, and all but wind position were significant for respirable particulate matter. The wind position versus the fire was not important for respirable quartz exposure. The crew type was an important factor in each model.

KEYWORDS

Carbon monoxide; firefighter; prescribed burn; quartz; silica; smoke; wildfire

Introduction

Exposure to smoke and other inhalation hazards among wildland firefighters is a concern to federal, state and local firefighting agencies, and contractors providing wildland fire management services.[1] Wildland firefighters have a different work environment than structural firefighters, who are principally engaged in fire suppression in the built environment, where fires consume diverse manmade materials in addition to wood-derived building materials. Wildland firefighters (subsequently, "firefighters") manage fire consuming primarily cellulosic fuels in the natural landscape. Along with occupational exposures to many physical hazards, firefighters are exposed to inhalation hazards from wildland fire smoke and from soil-derived dust. The smoke exposure can clearly cause short-term adverse health effects, and may contribute to chronic adverse health effects.[2–4] The soil composition can introduce asbestiform mineral exposures in certain geographies, and respirable crystalline silica should be considered a widely applicable soil dust hazard.[5,6] However, the long-term consequences for firefighters have not been addressed by adequate epidemiology despite substantial evidence of adverse effects among populations chronically exposed to fine particles from a variety of sources, including wildfire smoke.[7,8] Certain situational factors and tasks have been associated with higher smoke exposures, but we are not aware of a quantitative evaluation of the significance of these potential factors.[9,10] Multiple occupational exposure assessments among wildland firefighters have contributed to our understanding of inhalation hazards, but many are limited by issues such as: (1) small sample sizes that fail to capture infrequent but high exposure events; (2) challenges with industrial hygiene measurements by investigators without logistical experience at wildfires; (3) limited range of targeted compounds; and (4) a narrow geographical scope.[6,7,11,12] At the request of the National Wildfire Coordinating Group (NWCG) the United States Department of Agriculture - Forest Service (Forest Service) undertook a project in 2009 to fill some of these gaps with measurements of occupational smoke exposure across the U.S. by fire-qualified agency personnel who were engaged in a work production study by the San Dimas Technology & Development Center (TDC).[13] This project began data collection for carbon monoxide (CO) in 2009 among firefighters participating in the TDC work-production rate study. In 2010, at the request of the

CONTACT Timothy E. Reinhardt tim.reinhardt@woodplc.com Wood Environment & Infrastructure Solutions, Inc., 600 University Street, Suite 600, Seattle, 98101 WA.

Color versions of one or more of the figures in the article can be found online at www.tandfonline.com/uoeh.

NWCG Risk Management Committee, the study was expanded to estimate exposures at command and logistical centers for wildfires (called "fire camps"), and exposure to respirable particulate was added. In 2012, the study focused field data collection among fireline management personnel, because most long-time firefighters move into management roles, where their exposures may differ from the typical firefighter, whose average career duration has been estimated to be in the range of 7–25 years.[14]

This article summarizes the overall results of the project and presents our initial analysis of the importance of various factors for the fireline-average exposure results for the wildland firefighters. The exposures of fire managers were lower and summarized separately from firefighters. Fire camp results were generally at or below method detection limits and are not summarized here.

Methods

Exposure measurements

Fires were selected for the study by convenience across regions of the U.S., according to the seasonal patterns of fire activity in the continental U.S. The monitoring team personnel were experienced wildland firefighters very familiar with incident operations and logistical difficulties. On each day of exposure monitoring, 2–3 firefighters were typically monitored from within each of two crews, which ranged in size from 2–20 firefighters. The crews were often assigned to different areas of the fire, typically resulting in 4–6 firefighters per day from among two crews. At four prescribed burns (planned fire ignitions to achieve land management objectives), 6–9 firefighters were monitored from single 20-person crews. The personal breathing zone measurements of exposure to CO were made using CO dosimeters (Altair Pro Fire, MSA Safety Inc., Cranberry Township, PA) consistent with the National Institute for Occupational Safety and Health (NIOSH) Method 6604.[15] Beginning in 2010, data were also collected for exposure to respirable particulate matter (PM4) with a mass median aerodynamic diameter of 4 micrometers (μm), with analysis of crystalline silica content via NIOSH Method 7500.[16] Because long shifts were expected, a relatively low sampling flowrate was desirable to minimize overloading filters. Personal sampling pumps were used with the BGI SCC1.062 (Triplex) cyclone at 1.05 L/min (Mesa Laboratories, Inc., Butler, NJ) for consistency with 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®).[17]

All filter samples from the firefighters began upon arrival at the fireline, and ended at the end of the fireline activity, thus they are "fireline" -duration averages that omit time in the shift spent at briefings in fire camp, or traveling to or from the firefighting location in a vehicle or aircraft. The CO dosimeters recorded 1-min averages from the beginning of each shift until the crew departed from the fireline or ended duty at a remote spike camp. CO dosimeter responses were checked at the end of each shift with a second-source calibration gas traceable to National Institute of Standards and Technology Standard Reference Materials, and the data downloaded to field computers before the instruments were reset. The highest of the 5-min rolling-average CO exposures were calculated from the 1-min data for each firefighter to arrive at the highest brief exposure, for comparison to a Short-Term Exposure Limit (STEL). Similarly, the highest of their rolling 8-hr CO exposures was obtained for comparison to time-weighted average (TWA) occupational exposure limits (OELs), and their fireline- and shift-average CO exposures. For data modeling, we substituted half the detection limit if the period-integrated results were less than the reported detection limit.[18] Data reported in Table 2 used the reported detection limits because nonparametric methods were applied in that summary.

Explanatory variable measurements

The field teams collected detailed data for many possible explanatory variables, but due to missing observations, the data analysis only considered the following:

- fire type (prescribed burn, initial attack, project wildfire, prescribed natural fire). Prescribed fire includes preplanned-ignition prescribed burns, and naturally-ignited wildfires which are monitored rather than suppressed in areas where they achieve land management objectives. Results at both types of prescribed fire are summarized here.
- resource type. These included fire managers and fire crews, categorized as Type I (most-trained/qualified); and Type II (less-trained/qualified) 20-person "hand crews", of which the Type I crews were usually Interagency Hotshot Crews (IHC); some of the Type II crews were additionally qualified as Initial Attack (IA) crews, and some Type II crews were designated "Fuels" crews that

commonly perform fire management tasks through prescribed burning operations. The six wildland fire engine types and their associated 3–5-person crews were lumped into a single "engine" crew type, and the two-person bulldozer or tractor-plow crews were lumped into "dozer (I/II/III)" crews. A glossary of wildland fire suppression terms is maintained online by the National Wildfire Coordinating Group at https://www.nwcg.gov/glossary/a-z.

- work activity (recorded at the start of each shift and then when changes were observed).
- a condensed list of natural vegetation fuel models based on the widely-used 13 National Forest Fire Laboratory fuel categories developed by Albini and Anderson.[19] This was simplified by combining the flashy-burning fuels grass, grass/brush, and grass/timber into a single category "light"; combining the intermediate-duration burning fuels brush, grass/brush/timber into a single category "moderate"; and combining the sustained-burning brush/timber, timber, and slash into a single category "heavy"; and
- area of the country, as represented by U.S. Forest Service Region (1–9).

Modeling of exposure by these factors was performed on the log of each exposure metric. The exposure metrics evaluated included:

- the 5-min maximum CO exposure (representing STEL exposure situations);
- fireline-average CO exposure;
- fireline-average PM4 exposure; and
- fireline-average respirable crystalline silica (quartz) exposure.

Explanatory factors for shift-average exposures were not modeled because they include non-fireline time within the work shift, which depends on logistical aspects such as transport availability, travel distance and available travel modes (driving or hiking) between the fire and the daily operations base. Shift-average exposures are known to be reduced by the percentage of time spent off the firelines.[9]

For the firefighters, the data structure of the observational data set was considered and generalized linear mixed-effects models (GLMMs, also known as hierarchical or "multilevel") were developed and fit to the observed exposures.[20] In the GLMMs, the individual "firefighter" was not used in a repeated measures approach because most were unique, with fewer than 30% appearing in more than one shift and only a handful having more than two replicate shifts.

Firefighters are almost always assigned to tasks as a crew, working within line of sight much of the time, so the crew was a key clustering factor. Each day presents a unique assignment, landscape, set of environmental conditions and overall fire behavior, acting as a random factor influencing the exposure for each crew. We combined the day and crew name variables into a "daycrew" clustering factor.

An array of the within-day observations of work activity, standardized fuel composition (fuel model), wind position relative to the fire, windspeed (in mph), slope position relative to the fire, slope (%), and other variables identified above was created with a start and stop time, and net duration of each period within each firefighter's daily fireline time. Depending on each firefighter's within-day changes in activity, location, or simply observational opportunities, the number of unique observation periods varied between one and 18 periods per firefighter per day. The percentage of fireline time represented by a specific combination of potential explanatory variables (ultimately limited to work activity and wind position due to missing observations scattered within the data) was summed across each firefighter's shift, representing a weighting factor for how much of the fireline time the combination represented for each firefighter. The percentage of fireline time represented by a given activity and wind position was included as a continuous variable using grand mean centering to reduce collinearity effects.[20]

Two-level GLMMs were developed for the CO exposure, testing fixed effects of explanatory variables after grouping by the daycrew factor.[21] A null model using the daycrew factor was developed, and the significance of the GLMMs were compared against each other and the null model using likelihood-ratio tests. We developed only a random-intercept model, keeping the same average slope for each fixed effect across all instances of "daycrew" to arrive at a generally-applicable model for U.S. firefighters. All data analysis was performed in the R System for Statistical Computing (versions 3.4.1–3.5.1, R Core Team, Vienna, Austria).[22] The R packages NLME and LME4 were used for most of the data analyses.[23,24] The R package lmerTest was used for model simplification.[25]

Parameter estimates for a given model used restricted maximum likelihood estimates. Model comparisons by likelihood ratio tests were performed after refitting the models using maximum likelihood estimates. Graphics were produced in the package

Table 1. Workday duration data by fire type.

Fire Type and Personnel	n	Shift Duration (±Std. Dev.) (Hours)	Fireline Duration (±Std. Dev.) (Hours)
Prescribed Burns (crews)	83	10.5 (±2.7)	6.1 (±2.7)
Initial Attack (crews)	50[A]	12.4 (±3.6)	4.4 (±2.4)
Project Wildfires (crews)	417	13.6 (±1.5)	10.1 (±2.1)
Project Wildfires (managers)	31	14.5 (±2.2)	9.2 (±3.3)
Prescribed natural fires (crews)	83	13.6 (±2.2)	10.2 (±2.1)

Note: [A]There were 50 initial attack firefighters, but six personnel among three crews had either a 4- or a 2-incident day.

Lattice.[26] A similar modelling effort was undertaken for PM4 and respirable quartz exposure. Because generally one PM4/quartz sample was obtained per crew-day, there was insufficient replication within crew days, the GLMMs for these parameters used fire name as the best available random factor for clustering analogous to the "daycrew" used for CO.

Results

Exposure monitoring from 2009–2011 was completed for 83 firefighters' shifts at prescribed burns, 83 shifts at prescribed natural fires, 50 shifts on days with initial attack deployments (within which 60 total events occurred), and 417 firefighters' shifts at multi-day project wildfires. In 2012, data were collected from 31 additional shifts of line supervisors at project wildfire operations.

Work durations

Wildland firefighters work long hours and extended deployments, which must be considered when interpreting exposures vs. OELs. The NWCG and federal agency policy allows firefighters to work 14 days on assignment followed by a mandatory 2-day break. Table 1 summarizes the quantitative work duration data for personnel by fire type. Prescribed burning and initial attack were the only fire management activities with arithmetic mean fireline durations less than 8 hr. For initial attack days, the fireline time was the sum of hours across each fire when there were multiple events in a day.

Overall exposure result summary

Basic summary statistics for all exposure metrics were calculated prior to the GLMM analysis using the package STAND Version 2.0 in R, applying the Kaplan-Meier nonparametric method for subsets with significantly censored data, using the methods recommended by the American Industrial Hygiene Association Exposure Assessment Committee.[27,28] Table 2 summarizes the exposure data obtained during the study versus typical metrics. The maximum CO exposure over a 1-min period is compared with the Immediately Dangerous to Life and Health (IDLH) value of 1,200 parts per million (ppm) recommended by the National Institute of Occupational Safety and Health (NIOSH). The maximum CO exposure over a 5-min period is compared with the STEL of 200 ppm enforced by Washington State. The maximum 8-hr CO exposure is compared with a 35 ppm CO TWA recommended by NIOSH and enforced by some states, and the fireline-average CO exposure is compared with a 25 ppm Threshold Limit Value (TLV) to account for the longer fireline exposures. We do not view the 50 ppm OSHA PEL as sufficiently protective at the ventilation rates associated with firefighting, especially at high altitudes. The shift-average CO exposure is compared with the National Wildfire Coordinating Group's 2012 Interim Guideline for wildland firefighting of 16 ppm, to reduce exposures in consideration of long deployments with limited recovery time, and in view of the multiple contaminants present in smoke.[29] A critical issue in exposure management is the lack of a consensus OEL for PM4 that is mainly from wildland fire smoke. Our view is that the Particulate Not Otherwise Regulated (PNOR) PEL is inappropriate because wildland fire smoke has appreciable solubility, toxicity, and carcinogenic potential.[30–33] Therefore, as a working OEL guideline to interpret these data, we have reduced the target for PM4 exposure from the PNOR PEL of 5.0 milligrams per cubic meter (mg/m^3) down to a fireline-average OEL of 1.0 mg/m^3. This is reduced a further 30% to 0.7 mg/m^3 to arrive at an OEL guideline for shift-average PM4 exposure, using the ratio of 8 hr to the average shift duration.

Quartz was the only form of respirable crystalline silica observed in any of these exposure samples, and Table 2 shows the shift-average respirable quartz by fire type. Because the shift durations varied as noted above, the significance of the shift-average respirable quartz exposures was checked by comparing them to a shift duration-adjusted PEL. The adjusted PEL was obtained by multiplying the respirable quartz PEL (0.05 mg/m^3) by the ratio of 8 (hrs) divided by the duration of each firefighter's work shift. The second

to last column criterion of 0.029 mg/m^3 represents the adjustment for a 14-hr shift (the prescribed burning 10-hr shift results in an adjusted PEL of 0.4 mg/m^3). In the last column, the actual shift durations were used for each firefighter, thus values over 100% indicate exposures above the shift-duration adjusted PEL. We noted the substantial variation in these data, as indicated by the geometric standard deviations. This report summarizes further analysis of exposure factors undertaken to identify which factors can be used to develop similar exposure groups (SEGs) for management purposes.

Factors affecting fireline-average CO exposure

Some of the variables denoted in this paper were not observed in the first year of data collection, nor during every period of the time on the fireline for every firefighter in subsequent years, creating a significant data completeness problem. After exploratory review using tree-based methods, fixed factors not discussed here were dropped from further analysis because they could not be adequately assessed without a more complete, and preferably larger data set.[34]

For the fireline-average CO exposures, the best null model (clustering by the factor daycrew) had an intraclass correlation coefficient (ICC) of 0.67. Much of the variation in fireline-average CO exposures among firefighters was captured by which crew and day they happened to be observed on, a random factor that likely represents embedded characteristics that may be fruitful predictors of exposure in the future (such as situational and crew/crew leader parameters), but with this moderately sized data set could not be usefully examined as fixed factors. Therefore the 621 observations of firefighter's fireline-average CO exposure were clustered in the GLMM among 208 unique crew-days (factor "daycrew"). After stepwise backward elimination with LmerTest, the final CO model included the following categorical and continuous fixed-effect factors:

- crew type, with levels of *Dozer(I/II/III)*, *Engine*, and three types of handcrews: *I/I(IHC)*, *II (IA)*, or *II/Fuels*;
- position in the wind relative to the fire, with levels of *Calm*, *Downwind*, *Upwind*, or *Both*;
- majority activity during the fireline time (labeled *Activity2.1.1* in graphics), with levels of *Ancillary*, *DozerOps* (including Dozer or tractor plow operator or dozer operations boss), *Handline/Saw(Dir)* (for direct handline construction and sawyer), and *Handline/Saw(Ind)* for indirect activities, *Holding*, *LightingOps* (including both lighters and lighting boss tasks), *Mop up*, and *Pump Op* (for fire engine or portable water pump operators). The "ancillary" work activity included all activities not directly managing fire, such as: driving or hiking to a work zone, attending a pre-task briefing, standing by for orders or logistical support, staging at a strategic location near the fire, taking meals or other breaks, etc.;
- percentage of fireline time in the combination of two variables (Activity and Up/Downwind) that made up the highest percentage of fireline time for the firefighter in that shift (*Ctr.PctFireline1*). This was then centered as a hedge against collinearity by subtracting the grand mean percentage (42%) across all firefighters; and
- the interaction between the *Ctr.PctFireline1* and the specific work activity during that main activity time. If an activity was an important determinant of exposure, we would expect an enhanced influence on the exposure the more time that activity occurred.

Fire type just missed significance as a factor in the GLMM (p value of 0.15 in the final model retaining it). Simplified categories of fuel model (light, medium, and heavy fuels) during the majority activity also did not significantly improve the GLMM. Region of the country was not significant, and there were too many missing values to retain other potential factors in the model. The GLMM for fireline-average CO (including the daycrew random effects) explained 74% of the variance (conditional r^2), while these fixed effects explained 25% (marginal r^2).[35]

Figure 1 plots the population marginal means of log-transformed fireline-average CO concentrations (denoted "*LogDLFireline.CO*") among the levels of crew type, work activity, and wind position versus nearby fire activity. The 95% confidence intervals on the marginal means are shown (the dark line with end-hatches) overlaid on the mean for each level of each categorical variable (the thick colored or gray bars). Red bars represent factor levels with highly significant differences (probability $p < 0.001$), orange represent levels that were very significantly different ($p < 0.01$), and yellow represent levels that were significantly different ($p < 0.05$). Gray bars were not significantly different. The topmost plot titled "*Activity2.1.1*" shows that for the majority task/wind position combinations (which averaged 42% of fireline time):

- ancillary tasks correlated with lower fireline-average CO exposures than were observed for most other fireline tasks;

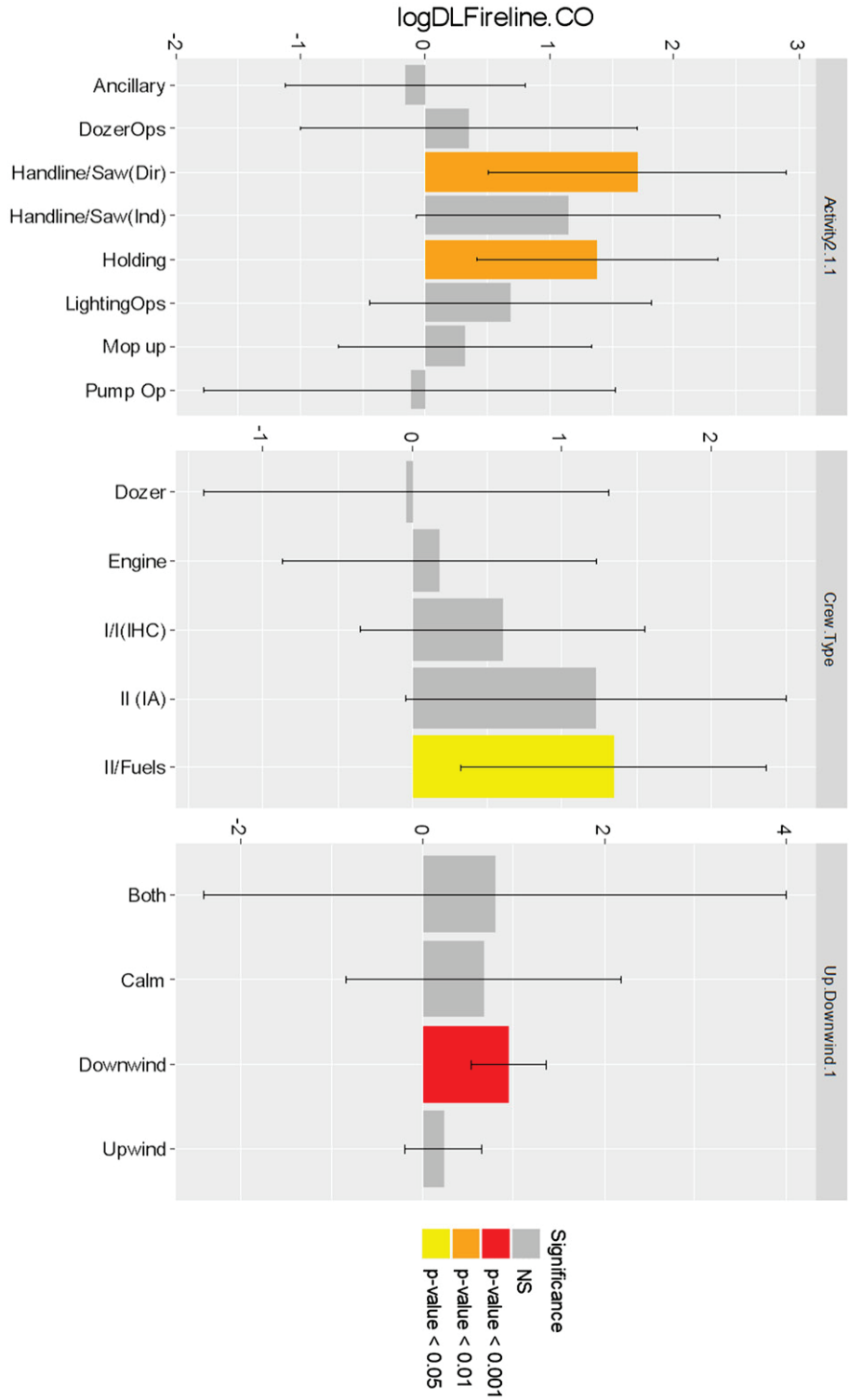


Figure 1. Contrast of fireline-average carbon monoxide exposure marginal means.

- handline/Sawyer(Direct) tasks had significantly higher fireline-average CO exposures than most other tasks; and
- holding line was associated with significantly higher fireline-average CO exposure than most other tasks.

The center plot (titled "Crew.Type") shows that for this factor:

- fireline-average CO exposure was significantly higher among Type II(Fuels) crews than other crew types; and

- engine and dozer personnel tended to have lower fireline-average CO exposures.

Finally, the bottom plot of Figure 1 (titled "*Up.Downwind.1*") confirms that a campfire axiom also applies to wildland fire operations: persons downwind of the fire are exposed to significantly more CO than persons upwind.

Table 3 summarizes the key model parameters for use in estimating fireline-average CO exposures among wildland firefighters. As an example using the model coefficients in Table 3, we predict that for a Type I/I(IHC) crewperson who spent 62% of their fireline time (0.2 more than the average of 0.42) performing direct handline construction downwind of a fire, the fireline-average CO exposure would be:

$$Fireline\ [CO] = e^{(0.06+1.868-4.416\times0.2+7.421\times0.2)},$$

where Fireline[CO] is the estimated fireline-average carbon monoxide exposure (12.5 ppm).

Another example would be a Type III/IV Engine crewperson performing mop up upwind of the fire for 73% of their fireline time. The model would estimate their fireline-average exposure to be:

$$Fireline\ [CO] = e^{(0.06-0.425+0.48-4.416\times0.31-0.712+4.355\times0.31)}.$$

Amounting to 0.5 ppm, on average.

Figure 2 is a scatterplot matrix showing the observed and model-fitted log fireline-average CO exposure by US Forest Service Region. In general, the model overpredicts at low exposure conditions and slightly underpredicts in high exposure situations, but is a reasonably consistent fit across all regions.

Despite having much fewer samples and correspondingly lower statistical power, the GLMMs for PM4 and quartz identified some significant factors, including: Crew Type and Main Activity. Wind position versus the fire was of marginal significance for PM4 and insignificant for respirable quartz. Figure 3 shows the marginal mean comparisons of log PM4 exposure by main work activity. PM4 exposure during mop-up was significantly higher than among those doing mainly ancillary tasks. The PM4 exposures for those mainly doing mop-up was higher than those doing mainly indirect handline construction.

The GLMM for fireline-average respirable quartz found that:

- the respirable quartz exposure during dozer operations and mop-up was significantly higher than the ancillary tasks; and
- as we would have predicted based on experience, respirable quartz exposures during mop-up were significantly higher than while holding line or lighting.

These key points are illustrated by the comparison of marginal mean respirable quartz exposures by main work activity in Figure 4.

Discussion

Wildland firefighters work long hours. Their time on the fireline frequently exceeds the 8-hr duration that is the norm of industrial workplaces for which OELs were established (Table 1), and staging/travel time often brackets each day's time on the fireline to create long work shifts. The long shifts reduce recovery time and need to be factored into appropriate OEL selection. Although it just missed significance in the fireline-average CO exposure model, we recommend retaining fire type as a broad means of classifying smoke exposure potential. Especially for prescribed burning, the intentional ignition of the landscape provides a strong incentive for firefighters to exert all efforts to maintain the fire within the designated unit boundaries, even when incurring significant smoke exposure. Initial attack, the first response to an incipient wildfire, is also a unique situation for shift-average exposures, because there is usually substantial shift time spent waiting and mobilizing to and from the fire, as found in an earlier study.[9] As Table 2 shows, this study finds that roughly 5% of occupational exposures among firefighters exceed recommended 5-min STELs, 8-hr TWA OELs and fireline-average OELs for CO at prescribed burns and prescribed natural fires, and occasionally CO OELs are exceeded at project fires and initial attacks. The arithmetic mean shift-average CO exposures we observed at prescribed burns (6.5 ppm) were higher than the 1.9 ppm reported among prescribed burn operations in the Southeastern U.[36] At 1.3 ppm, our shift-average CO geometric mean exposure results from project wildfires is about half the 2.8 ppm reported for 84 firefighters in 2004, but this should not be surprising considering our much larger sample across 417 firefighter shifts and greater emphasis on random selection of crews and days than the convenience-based approach used in the earlier study.[9] Our shift- and fireline-average CO results for initial attack personnel are consistent with the 2004 study finding that unexposed time in the day lowers the shift-average CO exposures for initial attack personnel from fireline-

Table 2. Summary metrics for occupational exposures among U.S. wildland firefighters and fire managers (2009–2012).

Distribution Metrics	CO 1-Min Avg. (ppm)	CO 5-Min 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	35	25	16	0.7[C]	0.029[F]	100
Initial Attack (n)	60[A]	60[A]	50	50	50	18	18	18
UTL (95%/95% UCL)	317	224	41	60	26	1.1	0.321	1370[D]
95th percentile	212[B]	141[B]	20[B]	30[B]	13[B]	0.54[B]	0.116[B]	430[B]
95% UCL of mean	61[B]	34[B]	3.1[B]	4.3[B]	2.1[B]	0.28[B]	0.043[B]	144[B]
Arithmetic Mean	51[B]	28[B]	2.4[B]	3.5[B]	1.6[B]	0.21[B]	0.028[B]	90[B]
Geometric Mean	29	14	0.8	1.4	0.58	0.13	0.012	31
GSD (unitless)	3.4	4.0	7.0	6.4	6.6	2.4	4.0	4.9
Nondetects (%)	1.7	1.7	2.0	2.0	2.0	61	44	44
Exposures > OEL (%)	0.0[B]	0.0[B]	0.0[B]	0.0[B]	0.0[B]	5.6[B]	28[B]	28[B]
95% UCL of > OEL (%)	4.9[B]	4.9[B]	5.8[B]	5.8[B]	5.8[B]	24[B]	50[B]	50[B]
Project Fire Crews (n)	417	417	417	417	417	80	80	80
UTL (95%,95% UCL)	572	368	68	62	44	1.9	0.191	649
95th percentile	489[B]	308[B]	53[B]	48[B]	34[B]	1.4[B]	0.119[B]	403[B]
95% UCL of mean	119[B]	60[B]	6.1[B]	5.6[B]	3.8[B]	0.57[B]	0.034[B]	115[B]
Arithmetic Mean	108[B]	54[B]	5.5[B]	4.9[B]	3.5[B]	0.49[B]	0.027[B]	90[B]
Geometric Mean	61	29	2.0	1.7	1.3	0.35	0.010	33
GSD (unitless)	3.6	4.2	7.3	7.7	7.3	2.4	4.6	4.6
Nondetects (%)	1.7	1.7	1.7	1.9[E]	1.7	10	38	38
Exposures > OEL (%)	0.5[B]	1.7[B]	0.5[B]	1.0[B]	1.0[B]	22[B]	28[B]	28[B]
95% UCL of > OEL (%)	1.5[B]	3.1[B]	1.5[B]	2.2[B]	2.2[B]	30[B]	37[B]	37[B]
Fire Managers (n)	31	31	31	31	31	31	31	31
UTL (95%/95% UCL)	706	600	77	110	46	0.68	0.03	132
95th percentile	311[B]	228[B]	21[B]	26[B]	12[B]	0.47[B]	0.022[B]	92[B]
95% UCL of mean	66[B]	40[B]	2.6[B]	3.6[B]	1.5[B]	0.24[B]	0.011[B]	44[B]
Arithmetic Mean	48[B]	28[B]	1.7[B]	2.1[B]	1.0[B]	0.19[B]	0.010[B]	37[B]
Geometric Mean	18	8.1	0.23	0.19	0.14	0.14	0.008	29
GSD (unitless)	5.6	7.6	15	20	16	2.0	1.9	2.0
Nondetects (%)	6.5	6.5	6.5	9.7	6.5	33	27	27
Exposures > OEL (%)	0.0[B]	0.0[B]	0.0[B]	0.0[B]	0.0[B]	3.3[B]	0.0[B]	0.0[B]
95% UCL of > OEL (%)	9.2[B]	9.2[B]	9.2[B]	9.2[B]	9.2[B]	15[B]	9.5[B]	9.5[B]
Prescr. Natural Fire (n)	83	83	83	83	83	16	16	16
UTL (95%/95% UCL)	707	521	151	118	83	1.2	0.026	95
95th percentile	472[B]	320[B]	74[B]	57[B]	41[B]	0.63[B]	0.018[B]	63[B]
95% UCL of mean	102[B]	51[B]	8.0[B]	6.5[B]	4.8[B]	0.32[B]	0.011[B]	37[B]
Arithmetic Mean	87[B]	43[B]	6.2[B]	5.0[B]	3.8[B]	0.23[B]	0.009[B]	30[B]
Geometric Mean	46	19	1.1	0.9	0.7	0.16	0.008	26
GSD (unitless)	4.1	5.6	13	13	12	2.3	1.6	1.7
Nondetects (%)	3.6	3.6	3.6	4.8[E]	3.6	44	38	38
Exposures > OEL (%)	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 > OEL (%)	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	15	15	15
UTL (95%/95% UCL)	476	314	60	55	36	2.9[B]	0.097	342
95th percentile	360	206	45	49	29	1.5[B]	0.038[B]	114[B]
95% UCL of mean	150	92	15	14	9.3	0.75[B]	0.027[B]	77[B]
Arithmetic Mean	123	72	10	10.4	6.5	0.50[B]	0.014[B]	39[B]
Geometric Mean	80	42	3.2	4.4	2.6	0.32	0.006	13
GSD (unitless)	2.5	2.9	4.6	3.7	3.9	2.5	3.1	3.7
Nondetects (%)	0	0	0	0	0	20	53	53
Exposures > OEL (%)	0.17	6.7	5.8	9.2	9.0	20[B]	6.7[B]	6.7[B]
95% UCL of > OEL (%)	0.62	11	9.7	14	14	44[B]	28[B]	28[B]

Notes: [A]60 individual fires (Peak/STEL metrics) among 50 firefighters.
[B]Nonparametric (i.e., Kaplan-Meier/nonparametric) estimate for left-censored data.
[C]This is a shift-duration-ratio reduction of a working OEL of 1 mg/m^3, until a risk assessment identifies a more suitable standard.
[D]The highest detected exposure, because the highest exposure was nondetected and the UTL is unstable with this small data set.
[E]Percentage increased because one firefighter's CO exposure occurred before deployment on the fireline.
[F]Quartz PEL (0.05 mg/m^3) adjusted for typical shift duration ratio (8/14 hr = 0.029 mg/m^3 criterion for all wildfires, and a criterion of 8/10 hr = 0.04 mg/m^3 for prescribed burns).

average exposures proportionally more than it does for project fire personnel.[9] Our project fire geometric mean CO exposure is within the 0.6–8.2 ppm range of exposures reported at one project fire in Montana; that study also found that sawyers operating chainsaws and swampers who removed the cut debris had geometric mean exposures at the higher end of that range.[37]

The UTL, 95th percentile and 95% UCL on the exceedance fraction of PM4 and quartz exposure summary metrics in Table 2 are likely to be less reliable than the Kaplan-Meier estimates of the arithmetic mean because of the relatively high proportion of nondetects in the data, especially for the initial attack and prescribed burn groups. Regardless, further focus

Table 3. Log(fireline-average co) exposure model parameters.

Random effects:			
Factor: Daycrew	**Intercept**	**Residual**	
Std. Deviation	1.479	1.074	
Fixed effects:			
Factor: Level	**Value**	**Std. Error**	**Prob.**[B]
(Intercept)[A]	0.060	0.264	
Crew Type			
Dozer	−0.644	0.587	
Engine	−0.425	0.314	
II(IA)	0.624	0.468	
II/Fuels	0.741	0.366	*
Main Activity			
DozerOps	0.511	0.574	
Handline/Saw(Dir)	1.868	0.421	***
Handline/Saw(Ind)	1.311	0.453	**
Holding	1.544	0.303	***
LightingOps	0.850	0.392	*
Mop up	0.480	0.296	
Pump Op	0.040	0.727	
Difference from average proportion of fireline time (0.42) in the main activity			
	−4.416	0.964	***
Wind position vs. fire			
Both	−0.144	1.628	
Calm	−0.270	0.775	
Upwind	−0.712	0.220	**
Activity-specific factor times the Δ from the average of 42% of fireline duration			
DozerOps	−0.266	2.341	
Handline/Saw(Dir)	7.421	1.993	***
Handline/Saw(Ind)	6.207	2.163	**
Holding	2.744	1.454	
LightingOps	4.498	1.630	**
Mop up	4.355	1.372	**
Pump Op	0.815	4.021	

Notes: [A]The intercept represents the conditions: Crew.Type = I/I(IHC), Activity = Ancillary, Position = Downwind of fire emissions, and the Ancillary activity coefficient (0) * Difference from average main activity time of 42% (0.42)
[B]Probability (of the calculated t value): *** = 0.001, ** = 0.01, * = 0.05.

on data collection and exposure controls is warranted, because these data indicate that exposures to respirable crystalline silica can frequently exceed the recently-lowered PEL of 0.05 mg/m^3. Our shift-average results for respirable quartz exposure among project fire and prescribed burn personnel are similar to task-duration respirable crystalline silica results reported among five wildland firefighters in 1989.[6] Because PNOR is not an applicable criterion, we used a working guideline of 1 mg/m^3, a fivefold reduction from the OSHA PNOR standard that may represent a reasonable control band for a properly risk-based OEL. If it were proven to be an appropriate OEL for wildland fire smoke, Table 2 shows that it would be exceeded by 22% of project fire crewpersons, and 20% of those at prescribed burns. The arithmetic mean fireline-average PM4 exposure of 0.5 mg/m^3 at prescribed burns is very similar to the arithmetic mean fine particulate (PM2.5) exposure of 0.462 mg/m^3 reported by others at prescribed burns.[38]

A GLMM using day and crew as a grouping factor was developed for CO. The CO model found that the crew type, the activity representing most of the fireline time, the position up or downwind of the fire and the amount of time this combination represented were significantly associated with fireline-average CO exposure. Being mainly downwind of the fire obviously leads to significantly higher exposures vs. upwind positions. Among the crew types, the 20-person Type II (Fuels) and Type II (IA) crews had the highest fireline-average CO exposures, significantly higher than the 3–5-person Engine and Dozer crews, and Type II (Fuels) crew exposures were even significantly higher than the more highly-trained 20-person Type I/I (IHC) crews. Fireline-average CO exposures among Type II (IA) crews were also higher than I/I (IHC) but not significantly so.

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, or mop up. Exposures among those mainly holding fireline were significantly higher than those doing the lighting tasks, and among those doing mop up. These results are generally consistent with task exposure comparisons among firefighters performing bushfire

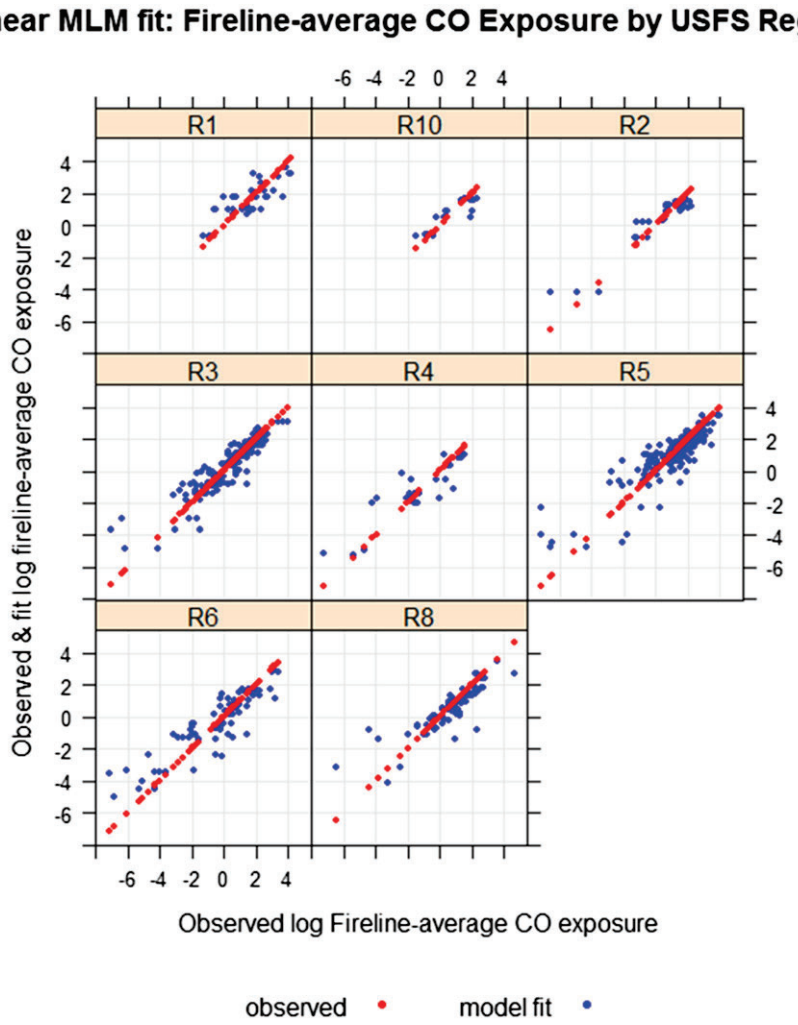


Figure 2. Model fit of log fireline-average carbon monoxide exposure among firefighters by U.S. Forest Service Region.

operations in Australia.[39] As might be expected, we found that CO exposures were low when the main tasks in a day were ancillary ones (hiking, standby/staging, briefing). Our fireline-average CO data indicate that management interventions will be most effective if focused on Type II and Type I/I(IHC) crews, especially when they are performing direct handline/sawyer assignments, holding firelines, and working downwind of the fire.

PM4 exposure management implications from these findings indicate that the most effective opportunities to reduce PM4 exposures would be among Type II and Type I crews downwind of the fire, holding firelines and performing mop-up, as they had higher exposures than engine crews or other tasks. Dozer crews also present opportunities to reduce PM4 exposures, including via engineering controls of enclosed cabs. We recommend that a research priority should be to derive a definitive PM4 OEL for wildland firefighters based on an epidemiological study of acute and chronic dose-response relationships.

For respirable quartz, the majority activity and the proportion of time it represented were the only significant factors. Wind position vs. the fire was not significant, unlike the case with CO. This is a reasonable difference, because unlike CO from fire, the soil dust is the source of the quartz exposure. As shown in Figure 4, 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 PM4 make sense when considering the source—respirable quartz arises mainly from soil disturbance, while PM4 can represent mainly smoke from the fire. Management implications for respirable quartz are that dozer operations and mop up tasks present the best opportunities to control dust exposures.

Respirable quartz controls should focus on personnel performing mop-up tasks, and although they

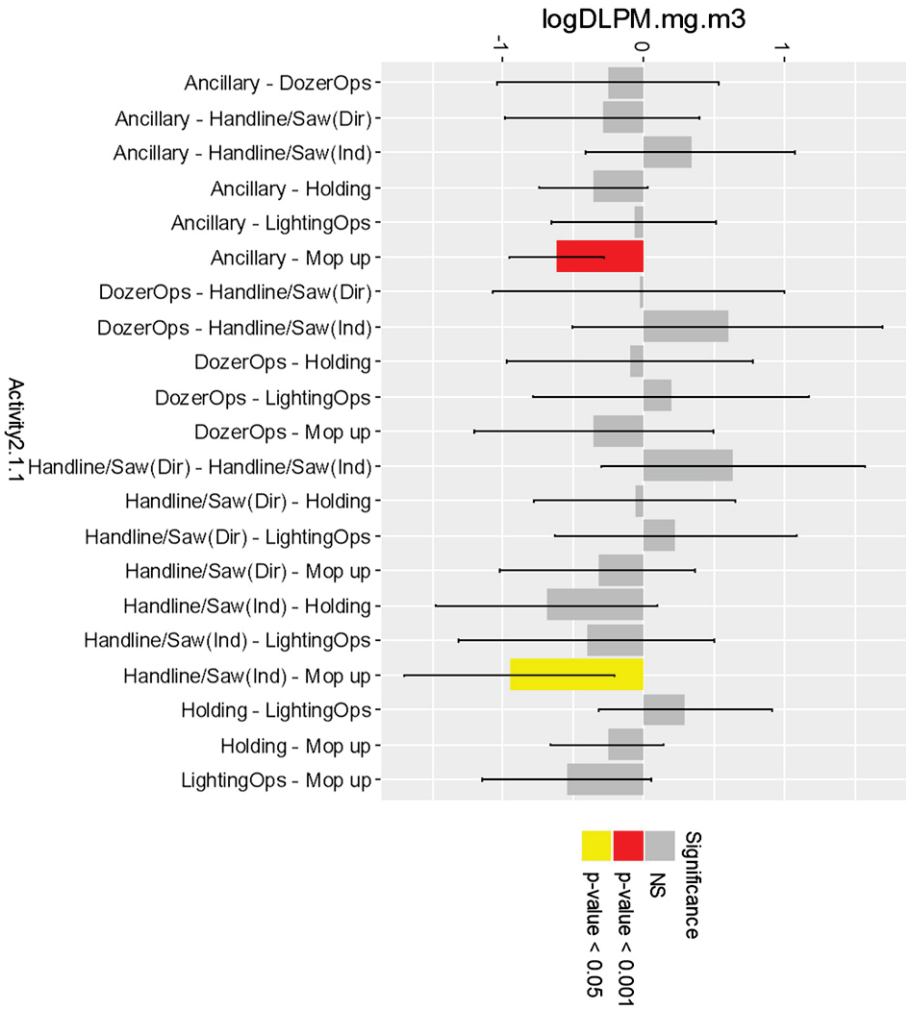


Figure 3. Contrast of fireline-average respirable particulate matter exposure marginal means by main activity.

weren't significantly higher than other tasks, the crews performing handline construction (because it involves soil disturbance). Dozers for wildland operations can be outfitted with air filtration that should be effective at reducing respirable quartz exposure, provided that filters are changed regularly, windows are kept closed and door seals are maintained.

Hand crews performing mop up are now, in most jurisdictions, trained to extinguish smoldering hot spots within a chain or so of the firelines rather than the longer distances that were the previous norm. Respirable quartz exposure during handline construction may be more difficult to mitigate. Increasing between-worker spacing may help reduce soil dust exposure potential.

Future refinements to data collection should collect task-based samples to refine SEGs among Type I/I(IHC), Type II(Fuels), or Type II(IA) crews performing fireline holding or Handline/Sawyer(Direct) tasks in downwind situations. Factors such as fuel model, wind speed, fire behavior, firefighter, and crew leader experience and other variables may ultimately prove to be useful predictors and should be considered in future data acquisition. A long-term CO surveillance project might be appropriately focused on tracking and assessing the effectiveness of CO exposure mitigation strategies 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. Grouping future data into SEGs by these crew, task and wind position categories may reduce the variability of the results and improve the ability to detect a real reduction of exposure from a given mitigation strategy. Given the ICC associated with "daycrew" as a grouping factor in the GLMM for CO exposure, we conclude that exposure results from only one fire or crew can easily over- or underrepresent results that might be obtained across a wider sampling of days and crews. This has importance in planning future surveillance in this industry—better to obtain more days of data at different fires or at least different crews, rather than replicates within a crew.

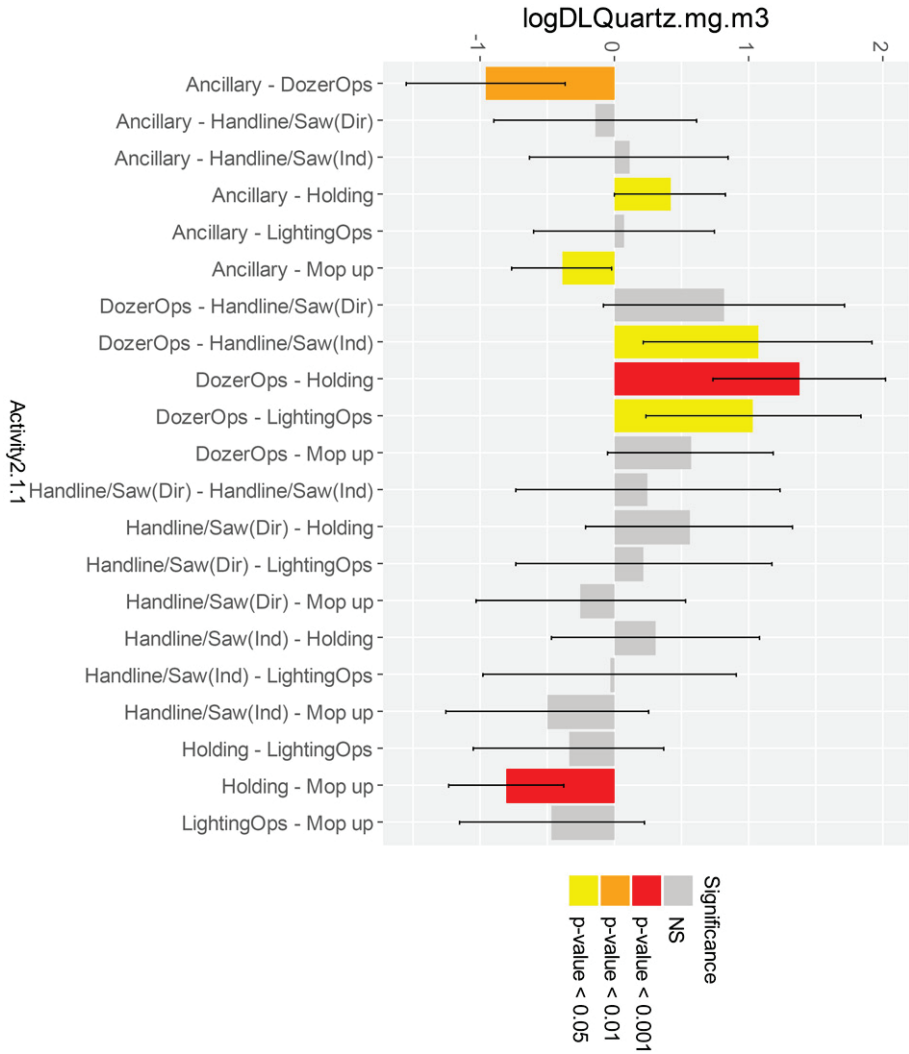


Figure 4. Contrast of fireline-average respirable quartz exposure marginal means by main activity.

Further action to control smoke and dust exposures is warranted by these results. Our data indicate that smoke and dust exposures are at times above a variety of consensus OELs for CO and respirable quartz, and thus deserving of a change in operations to reduce those exposures. Depending on the level at which an OEL based on smoke particle toxicity is set, the percentage of PM4 overexposures could be even higher than we have shown here with our working OEL guideline of 1.0 mg/m^3. Available actions include engineering and administrative controls. These may yet have promise but so far have not eliminated exposures of concern. Delineating prescribed burn and wildfire burnout operations to have easily-defended boundaries has been mentioned for years. Filtered-air sleeping trailers have been deployed at project fire camps, but these only provide off-shift respite. Pre-wetting firelines and resources at risk with sprinklers is an option discussed to mitigate the need for holding firelines during prescribed burning, but we are not aware of data documenting its implementation. In fact, we are not aware of research by any land management agency into the frequency of deployment and situational effectiveness of smoke exposure mitigation strategies. We think this is a step that should be undertaken for this industry. Respiratory protection using respirators certified by NIOSH to meet the NFPA 1984 Wildland Firefighter Respiratory Protection Standard could be an option to ensure that CO exposure is controlled along with removal of silica, smoke particulate and low-concentration organic chemicals and acid gases. The NFPA 1984 respirator sets a high bar for breathing resistance and performance. It may be an ideal solution for wildland-urban interface fires that are mainly wildland vegetation, but organizations adopting them must consider the effects of respirators on: (1) heat stress of firefighters; (2) their ability to communicate clearly in critical conditions; and (3) the likelihood that situational awareness will be dramatically reduced—especially by full-face

respirators, a logical choice considering the irritant nature of smoke on the eyes.

Currently, no manufacturers are marketing a respirator that meets NFPA 1984. We understand that they are feasible to produce but awaiting a viable market. Fire management agencies should consider their application to control respirable quartz and PM4 exposures, especially when fire entrapment risks are low.

Acknowledgments

No potential conflict of interest was reported by the author(s).

Funding

Joint Fire Science Program 13-1-02-14

References

[1] **Butler, C., S. Marsh, J.W. Domitrovich, and J. Helmkamp**: Wildland firefighter deaths in the United States: A comparison of existing surveillance systems. *J. Occup. Environ. Hyg. 14*:258–270 (2017).

[2] **Betchley, C., J.Q. Koenig, G. van Belle, H. Checkoway, and T. Reinhardt**: Pulmonary function and respiratory symptoms in forest firefighters. *Am. J. Industr. Med. 31*:503–509 (1997).

[3] **Adetona, O., T.E. Reinhardt, J. Domitrovich, et al**: Review of the health effects of wildland fire smoke on wildland firefighters and the public. *Inhal. Toxicol. 28*:95–139 (2016).

[4] **Gaughan, D.M., J.M. Cox-Ganser, P.L. Enright, et al**: Acute upper and lower respiratory effects in wildland firefighters. *J. Occup. Environ. Med. 50*:1019–1028 (2008).

[5] **U.S. Dept. of Health and Human Services**: *Asbestos Air Monitoring Soil Sampling. Plumas National Forest 'Chips Fire' Suppression Equipment Recovery Asbestos Sampling.* Public Health Service, Federal Occupational Health, San Francisco, December 2012.

[6] **Materna, B.L., J.R. Jones, P.M. Sutton, N. Rothman, and R.J. Harrison**: Occupational exposures in California wildland firefighting. *Am. Ind. Hyg. Assoc. J. 53*:69–76 (1992).

[7] **Araujo, J.A., B. Barajas, M. Kleinman, et al**: Ambient particulate pollutants in the ultrafine range promote early atherosclerosis and systemic oxidative stress. *Circ. Res. 102*:589–596 (2008).

[8] **Pope, C.A., R.T. Burnett, M.C. Turner, et al**: Lung cancer and cardiovascular disease mortality associated with ambient air pollution and cigarette smoke: Shape of the exposure-response relationships. *Environ. Health Perspect. 119*:1616–1621 (2011).

[9] **Reinhardt, T.E., and R.D. Ottmar**: Baseline measurements of smoke exposure among wildland firefighters. *J. Occup. Environ. Hyg. 1*:593–606 (2004).

[10] **Reisen, F., D. Hansen, and C.P. Meyer**: Exposure to bushfire smoke during prescribed burns and wildfires: Firefighters' exposure risks and options. *Environ. Int. 37*:314–321 (2011).

[11] **U.S. Department of Health and Human Services**: *Health Hazard Evaluation Report.* U.S. Department of the Interior, National Park Service, Yellowstone National Park, Wyoming, by C. Reh, and S. Deitchman (HETA # 88-320-2176). U.S. Public Health Service, Centers for Disease Control, National Institute for Occupational Safety & Health (1992).

[12] **Adetona, O., K. Dunn, D.B. Hall, G. Achtemeier, A. Stock, and L.P. Naeher**: Personal PM2.5 exposure among wildland firefighters working at prescribed forest burns in southeastern United States. *J. Occup. Environ. Hyg. 8*:503–511 (2011).

[13] **U.S. Department of Agriculture, Forest Service**: *Fireline Production Rates (Report 1151 1805P)*, by G. Broyles. Forest Service, National Technology & Development Program, April 2011.

[14] **Booze, T.F., T.E. Reinhardt, S.J. Quiring, and R.D. Ottmar**: A screening-level assessment of the health risks of chronic smoke exposure for wildland firefighters. *J. Occup. Environ. Hyg. 1*:296–305 (2004).

[15] **Woodfin, W.J.**: Carbon monoxide: Method 6604, Issue 1., In *NIOSH Manual of Analytical Methods*, 4th ed. Ashley, K., and P.F. O'Connor (Eds.) Atlanta, GA: National Institute for Occupational Safety and Health (1996).

[16] **Key-Schwartz, R., Ramsey, D. and Schlecht, P.**: Silica, Crystalline, By XRD (filter redeposition): Method 7500, Issue 4. In *NIOSH Manual of Analytical Methods*, 4th ed. Ashley, K., and P.F. O'Connor (Eds.) Atlanta, GA: National Institute for Occupational Safety and Health (2003).

[17] **American Conference of Governmental Industrial Hygienists (ACGIH)**: Appendix C: Particle size-selective sampling criteria for airborne particulate matter. In: 2015 TLVs® and BEIs®. ACGIH, Cincinnati, OH (2015).

[18] **Hornung, R.W., and L.D. Reed**: Estimation of average concentration in the presence of nondetectable values. *Apppl. Occup. Environ. Hyg. 5*:46–51 (1990).

[19] **U.S. Department of Agriculture**: *Aids to Determining Fuel Models for Estimating Fire Behavior*, by H.E. Anderson (General Technical Report INT-122). U.S. Forest Service. 1982.

[20] **Finch, W.H., J.E. Bolin, and K. Kelley**: *Multilevel Modeling Using R.* Boca Raton, FL Chapman & Hall/CRC Press, 2014.

[21] **Maindonald, J., and J. Braun**: *Data Analysis and Graphics Using R. An Example-Based Approach*. 2nd ed. New York: Cambridge University Press, (2007).

[22] **R Core Team**: R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. URL https://www.R-project.org/ (accessed 12/15/2017).

[23] **Pinheiro, J., D. Bates, S. DebRoy, D. Sarkar, and R Core Team**: nlme: Linear and Nonlinear Mixed Effects Models_. R package version 3.1-131, <URL: https://CRAN.R-project.org/package=nlme> (accessed 12/15/2017).

[24] **Bates, D., M. Maechler, B. Bolker, and S. Walker**: Fitting Linear Mixed-Effects Models Using lme4. *J. Statist. Software 67*:1–48 (2015).

[25] **Kuznetsova, A., P.B. Brockhoff, and R.H.B. Christensen**: lmerTest Package: tests in linear mixed effects models. *J. Statist. Software* 82:1–26 (2017).

[26] **Sarkar, D.**: *Lattice: Multivariate Data Visualization with R*. New York: Springer Science + Business Media, 2008.

[27] Ignacio, J.S., and W.H. Bullock, Editors: *A Strategy for Assessing and Managing Occupational Exposures*, 3rd ed. Fairfax: AIHA Press, 2006.

[28] **E.L. Frome and D.P. Frome (2015):** STAND: Statistical Analysis of Non-Detects. R package version 2.0. https://CRAN.R-project.org/package=STAND (accessed 12/15/2017).

[29] **National Wildfire Coordinating Group**: Monitoring and mitigating exposure to carbon monoxide and particulates at incident base camps. Memorandum NWCG 006-2012. Boise, ID. 2012.

[30] **Mauderly, J.L., and J.C. Chow**: Health effects of organic aerosols. *Inhal. Toxicol. 20*:257–288 (2008).

[31] **Leonard. S.S., V. Castranova, B.T. Chen, et al.:** Particle size-dependent radical generation from wildland fire smoke. *Toxicology 236*:103–113 (2007).

[32] **Wegesser, T.C., K.E. Pinkerton, and J.A. Last**: California wildfires of 2008: Coarse and fine particulate matter toxicity. *Environ. Health Perspect. 117*:893–897 (2009).

[33] **Naeher, L.P., M. Brauer, M. Lipsett, et al**: Woodsmoke health effects: A review. *Inhal. Toxicol. 19*:67–106 (2007).

[34] **Crawley, M.J.:** *The R Book*. Chichester, UK: John Wiley & Sons, Ltd. 2007. Ch. 21.

[35] **Nakagawa, S., and H. Schielzeth.** A general and simple method for obtaining R^2 from generalized linear mixed-effects models. *Meth. Ecol. Evol. 4*:133–142 (2013).

[36] **Dunn, K., I. Devaux, and L.P. Naeher.** Application of end-exhaled breath monitoring to assess carbon monoxide exposures of wildland firefighters at prescribed burns. *Inhal. Toxicol. 21*:55–61 (2009).

[37] **Gaughan, D., C. Piacitelli, B.T. Chen, et al**. Exposures and cross-shift lung function declines in wildland firefighters. *J. Occup. Environ. Health 11*:591–603 (2014).

[38] **Adetona, O., K. Dunn, G. Achtemeier, et al**. Personal PM2.5 exposure among wildland firefighters working at prescribed forest burns in southeastern United States. *J. Occup. Environ. Health 8*:503–511 (2011).

[39] **Reisen, F., D. Hansen, and C.P. Meyer.** Exposure to bushfire smoke during prescribed burns and wildfires: Firefighters' exposure risks and options. *Environ. Int. 37*:314–321 (2011).

Hi Amalia and Eric – with much thanks to Rachel Bailey and her colleagues at NIOSH (cc'd here), attached here are some additional resources for your consideration at the 8 May CalOSHA meeting.
I have obtained permission to attend, so I will look forward to meeting you and your colleagues in Oakland.
Best regards, Bob

Robert S. Bernstein, MD, PhD, MPH, FACPM
Health Officer
Tuolumne County Public Health
(209) 770-1991

From: Bailey, Rachel L. (CDC/NIOSH/RHD/FSB) [mailto:feu2@cdc.gov]
Sent: Wednesday, April 17, 2019 1:39 PM
To: Robert Bernstein
Cc: Castillo, Dawn N. (CDC/NIOSH/DSR); Romano, Nancy (CDC/NIOSH/DSR/SFIB); Nett, Randall J. (CDC/NIOSH/RHD/FSB); Cox-Ganser, Jean (CDC/NIOSH/RHD/OD); Moore, Melanie (CDC/NIOSH/DSR/SFIB)
Subject: Wildland Fire Fighters

Hi Dr. Bernstein,

Thank you for your voicemail requesting information about wildland fire fighters.

NIOSH has a webpage on fighting wildfires (https://www.cdc.gov/niosh/topics/firefighting/default.html) with links to NIOSH publications and other resources.

I have also attached a number of publications about wildland fire fighter exposures.

Please let us know if this is helpful or if we can be of further assistance.

Sincerely,

Rachel L. Bailey, DO, MPH
Captain, U.S. Public Health Service
Medical Officer
Health Hazard Evaluation Program Coordinator
Field Studies Branch
Respiratory Health Division
National Institute for Occupational Safety and Health (NIOSH)
Centers for Disease Control and Prevention (CDC)
1095 Willowdale Road; M/S H2800
Morgantown, WV 26505

Phone: (304) 285-5757
Fax: (304) 285-5820
Email: feu2@cdc.gov