



Review or Mini-review

## Wildfire smoke exposure and human health: Significant gaps in research for a growing public health issue



Carolyn Black<sup>a</sup>, Yohannes Tesfaigzi<sup>b</sup>, Jed A. Bassein<sup>a</sup>, Lisa A. Miller<sup>a,c,\*</sup>

<sup>a</sup> California National Primate Research Center, United States

<sup>b</sup> Lovelace Respiratory Research Institute, Albuquerque, NM, United States

<sup>c</sup> Department of Anatomy, Physiology, and Cell Biology, School of Veterinary Medicine, University of California Davis, Davis, CA, United States

### ARTICLE INFO

#### Keywords:

Wildfire  
Smoke  
Particulate  
Exposure  
Health  
Inhalation  
Toxicology

### ABSTRACT

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

### 1. Introduction

Wildfire and other biomass smoke exposures are increasingly recognized as an important public health issue. While air quality in the United States has generally improved in recent decades due to increased regulatory control, emissions from wildfires have trended upward and are projected to increase as climate change increases the frequency and severity of wildfires (Flannigan et al., 2000; Kinney, 2008). In 2012, wildfires in the US contributed over half of all estimated methane emissions, and 20% of all fine particulate emissions (EPA, 2011). While in Canada, approximately one third of all particulate emissions came from forest fires (Rittmaster et al., 2006). Recent American Thoracic Society reports highlight the growing interest in understanding the impact of climate change on human health, including better understanding how climate change will affect human exposures to respiratory irritants (Pinkerton et al., 2012; Rice et al., 2014). Changes in land cover and in policies concerning fire control and surveillance further complicate future projections of wildfire emissions estimates, however many aspects of climate change are directly related to wildfire risk,

including temperature and drought (Flannigan et al., 2013). For example, half of fine particulate emissions were attributed to wildfire during the recent drought in California (California, 2012). Due to climate change and development, wildfire emissions are expected to increase an additional 19–101% in California through 2100 (Hurteau et al., 2014). Increasing numbers of wildfires and acreage burned is also expected to increase across the western United States and Europe (Abatzoglou and Williams, 2016; Lozano et al., 2016).

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

**Abbreviations:** CC16, Clara cell secretory protein 16; COPD, chronic obstructive pulmonary disease; CXCL1, C-X-C motif chemokine ligand 1; CYP1A1, cytochrome P450 family 1 subfamily A member 1; CYP1B1, cytochrome P450 family 1 subfamily B member 1; EPA, Environmental Protection Agency; FEPS, Fire Emissions Production Simulator; FEV1, forced expiratory capacity in 1 s; GM-CSF, granulocyte macrophage colony-stimulating factor; HBE, human bronchial epithelial cells; IL-1 $\alpha$ , interleukin-1 alpha; IL-1 $\beta$ , interleukin-1 beta; IL-6, interleukin-6; IL-8, interleukin-8; LPS, lipopolysaccharide; MIP-1 $\alpha$ , macrophage inflammatory protein-1 alpha; NAAQS, National Ambient Air Quality Standards; PAH, polycyclic aromatic hydrocarbons; PM<sub>2.5</sub>, particulate matter less than 2.5  $\mu$ m; PM<sub>10</sub>, particulate matter less than 10  $\mu$ m; TNF $\alpha$ , tumor necrosis factor alpha

\* Corresponding author at: California National Primate Research Center, UC Davis School of Veterinary Medicine, University of California, Davis, CA 95616, United States.

E-mail address: [lmiller@ucdavis.edu](mailto:lmiller@ucdavis.edu) (L.A. Miller).

<http://dx.doi.org/10.1016/j.etap.2017.08.022>

Received 6 August 2017; Accepted 26 August 2017

Available online 30 August 2017

1382-6689/© 2017 Elsevier B.V. All rights reserved.

wildfire health effects, human and animal wood smoke exposure studies, and *in vitro* studies. We will discuss the limitations of current studies, and emphasize critical research topics for the future.

## 2. Wildfire smoke composition

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

Wildfire smoke is a major contributor to particulate air pollution. Wildfires produce proportionately more fine (under 2.5  $\mu\text{m}$ ) and ultrafine (under 1  $\mu\text{m}$ ) particulate, compared to coarse particulate, defined as particles fewer than 10  $\mu\text{m}$  in size ( $\text{PM}_{10}$ ) (Makkonen et al., 2010; Radke et al., 1991). Fine particles generally settle out of the atmosphere more slowly than coarse particles, and therefore disperse farther from the source (Kinney, 2008). Fine and ultrafine particulate is also of particular concern in human health because of its ability to penetrate more deeply into the lung. For this reason  $\text{PM}_{2.5}$  has been singled out for special consideration in government documents and guidelines (EPA, 2009). Ultrafine particulate constitutes a substantial proportion of wildfire-generated particulate, although the average size of smoke particulate depends on the intensity of the fire, type of fuel, and whether the fire is smoldering or flaming (Reid et al., 2005).

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

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

Many of the organic species found in wildfire smoke are unique to biomass combustion compared to fossil fuels, such as levoglucosan and other byproducts of cellulose combustion (Sillanpää et al., 2005). Surprisingly, no studies have been published on the health effects of these biomass smoke-specific species. The majority of wood smoke

particulate is composed of organic carbon, compared to a higher level of elemental carbon found in fossil fuel emissions (Kocbach et al., 2006). Production of volatile and semi-volatile organic compounds is also higher in wood smoke compared to fossil fuel emissions, whereas nitrogen oxides and sulfur dioxide levels are lower (Mauderly et al., 2014). The health significance of PAH produced by wildfires is controversial. Studies comparing PAH in laboratory-generated wood smoke with PAH collected from traffic sources generally find higher levels in wood smoke (Bølling et al., 2012; Forchhammer et al., 2012). However, collection of ambient wildfire particulate demonstrates relatively low levels of PAH compared to urban sources, perhaps due to decomposition of PAH species during atmospheric transit (Jalava et al., 2006; Kocbach Bølling et al., 2009).

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

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

## 3. Health effects of wildfire smoke

### 3.1. Population health effects

Exposure to wildfire smoke has been a longstanding concern in

Download English Version:

<https://daneshyari.com/en/article/5559767>

Download Persian Version:

<https://daneshyari.com/article/5559767>

[Daneshyari.com](https://daneshyari.com)