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A systematic review of the physical health impacts from non-occupational exposure to wildfire smoke

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Abstract

Background—Climate change is likely to increase threat of wildfires, and little is known about how wildfires affect health in exposed communities. A better understanding of the impacts of the resulting air pollution has important public health implications for the present day and the future.

Method—We performed a systematic search to identify peer-reviewed scientific studies published since 1986 regarding impacts of wildfire smoke on health in exposed communities. We reviewed and synthesized the state of science of this issue including methods to estimate exposure, and identified limitations in current research.

Results—We identified 61 epidemiological studies linking wildfire and human health in communities. The U.S. and Australia were the most frequently studied countries (18 studies on the U.S., 15 on Australia). Geographic scales ranged from a single small city (population about 55,000) to the entire globe. Most studies focused on areas close to fire events. Exposure was most commonly assessed with stationary air pollutant monitors (35 of 61 studies). Other methods included using satellite remote sensing and measurements from air samples collected during fires. Most studies compared risk of health outcomes between 1) periods with no fire events and periods during or after fire events, or 2) regions affected by wildfire smoke and unaffected regions. Daily pollution levels during or after wildfire in most studies exceeded U.S. EPA regulations. Levels of PM_{10}, the most frequently studied pollutant, were 1.2 to 10 times higher due to wildfire smoke compared to non-fire periods and/or locations. Respiratory disease was the most frequently studied health condition, and had the most consistent results. Over 90% of these 45 studies reported that wildfire smoke was significantly associated with risk of respiratory morbidity.

Conclusion—Exposure measurement is a key challenge in current literature on wildfire and human health. A limitation is the difficulty of estimating pollution specific to wildfires. New

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methods are needed to separate air pollution levels of wildfires from those from ambient sources, such as transportation. The majority of studies found that wildfire smoke was associated with increased risk of respiratory and cardiovascular diseases. Children, the elderly and those with underlying chronic diseases appear to be susceptible. More studies on mortality and cardiovascular morbidity are needed. Further exploration with new methods could help ascertain the public health impacts of wildfires under climate change and guide mitigation policies.

Keywords

Wildfire; Air pollution; Health; Smoke; Forest Fire

1. Introduction

Much remains unknown regarding the public health impacts of forest fire smoke, but interest in the topic is growing as forest fire incidence rises in many parts of the world (Dimopoulou and Giannikos 2004). There is broad consensus that climate change is increasing the threat of forest fires (Albertson *et al.*, 2010; Balling *et al.*, 1992; Flannigan and Vanwagner 1991; Keeton *et al.*, 2007; Malevsky-Malevich *et al.*, 2008; Spracklen *et al.*, 2009), with fires that burn more intensely, occur more frequently, and can spread faster (Fried *et al.*, 2008; Fried *et al.*, 2004; Parry *et al.*, 2007; Westerling and Bryant 2008). The U.S. Forest Service noted that forest fires have already become more intense and that the forest fire season has expanded (U.S. Forest Service 2009). While an increasing frequency of forest fires has often been attributed to many factors including changes in land use, higher spring and summer temperatures may be more relevant (Westerling *et al.*, 2006). The Intergovernmental Panel on Climate Change (IPCC) anticipates that climate change will lengthen the window of high summertime forest fire risk in North America by 10–30%, and result in increased frequency of forest fires in many other parts of the world (Parry *et al.*, 2007). As a result, exposure to air pollution from forest fires is anticipated to increase in coming decades (Interagency Working Group on Climate Change and Health 2010).

The U.S. Forest Service recognizes forest fire smoke as a hazard to human health and identifies airborne particulate matter (PM) as the component of greatest concern for the public (U.S. Forest Service 2010). Numerous studies have demonstrated links between airborne particles and health outcomes including mortality and hospital admissions (Lepeule *et al.*, 2012; Medina-Ramon *et al.*, 2006; Peng *et al.*, 2008; Pope and Dockery 2006). However, not all particles appear to be equally toxic as research indicates that the size and chemical composition of airborne particles affect its impact on health (Ebisu and Bell 2012; Franck *et al.*, 2011; Zanobetti *et al.*, 2009). In general, effects are stronger for smaller particles, which can deposit deeper in the respiratory tract (Valavanidis *et al.*, 2008). The specific mechanistic pathways to adverse health outcomes remain unclear, but chemical composition, particle size, number, and shape have been identified as of putative importance. As the chemical composition of forest fire smoke is likely to differ from those of other sources (e.g., vehicles) (Mao *et al.*, 2011; Pio *et al.*, 2008; Robinson *et al.*, 2011), the observed health associations for more commonly studied air pollutants and sources, such as particulate matter in urban settings, may not be generalizable to pollution from forest

fires. Thus, scientific evidence is needed on the health burden from forest fire smoke specifically.

Understanding how forest fire smoke affects public health has the potential to inform intervention-focused policies to protect public health in the present day, climate change mitigation policies, research on health impacts from a changing climate, and economic estimates of the health costs of forest fires. We reviewed and summarized the published literature regarding the public health impacts of forest fire smoke with the goals of synthesizing existing information and identifying gaps in scientific knowledge.

2. Methods

Eligibility criteria

We reviewed peer-reviewed journal articles on the topic of forest fire/wildfire smoke and health, published between 1 Jan 1986 and 30 May 2014. We included studies written in English or Portuguese (with English abstract), and excluded papers written in other languages. We considered all papers relevant to non-occupational exposure to wildfire smoke and physical health impact. We excluded experimental/chamber studies because it is not clear how relevant the exposure level/composition is to those experienced by the community. We excluded conference abstracts, unpublished studies, and non-research publications, such as commentaries. Natural fires were included and controlled prescribed burns were excluded. We did not exclude studies based on type or diversity of vegetation, such as trees peat bog or savannah. All fires are referred to as ‘wildfire’ hereon. We excluded studies of indoor and outdoor wood burning for heating or cooking purposes. Studies that investigated occupational exposures were excluded, as the focus of this review was impacts on communities or broader populations. Therefore, we excluded studies of fire fighters. Since mental health issues are not direct physical health consequences from exposure to wildfire smoke, we excluded studies that investigated only mental health outcomes. As this review focussed on wildfire smoke we also excluded studies that investigated non-smoke related morbidities, such as burns and accidents. Thus, we focused on wildfire smoke and its physical health impacts on the general population.

Information sources

We considered papers indexed in PubMed, a database of biomedical literature and life science journals, managed by the U.S. National Library of Medicine (NIH 2011) and Scopus, a comprehensive database of research literature (Elsevier 2013). References of the resulting papers were examined to better ensure a complete assessment of the literature.

Search terms

Detailed information on the search terms is provided in the supplemental material. Briefly, key words included “wildfire”, “forest fire”, or “bushfire” with any of the following: “health”, “hospital*”, “respir*”, “pulmon*”, “asthma*”, “cardiac”, “cardiovascular”, or “mortality”, where “*” stands for any combination of letters (*e.g.*, hospital* can represent hospitalizations or hospital) (Appendix A).

Summary measures

We summarized the papers with respect to study setting, study design, exposure and outcome assessment, participant vulnerability, key findings, and estimates of association (*e.g.*, odds ratios) when provided.

Study assessment

As exposure assessment is a critical challenge in the study of health impacts from wildfire smoke, we described the approaches used by identified studies to estimate exposures. We assessed the overall state of scientific evidence on associations between wildfire smoke and health outcomes for respiratory morbidity, cardiovascular morbidity, mortality, and other outcomes. The approaches to assess health outcomes are diverse, and we summarized the sources of health data for each study. We grouped the studies by health outcomes and summarized the results on health effects. We described factors that might have influenced the summary of evidence based on the studies reviewed. Finally, we highlighted the limitations of these studies and identified needs for future research.

3. Results

The database searches identified 926 papers. We then excluded 277 duplicates (i.e., papers identified by more than one search). We eliminated papers that did not meet the inclusion criteria, by first screening the titles and abstracts (526 papers excluded) and then by a review of the full articles (62 papers excluded). We also excluded studies for which wildfire smoke exposure was not a dominant component relative to other ambient sources (e.g. Sarnat *et al.*, 2008). The final review included 61 studies of human health impacts of wildfires in community populations (Table 1).

Study setting

More studies were identified for more recent years, with 4 studies published before 2000 and 35 studies published in the last 5 years. Most studies focused on the Brazilian Amazon, Southeast Asia and the Pacific, the North American West, and the Mediterranean, where wildfires are common. The U.S. and Australia were the most frequently studied countries (18 U.S. studies, 15 Australian studies). Southeast Asia was also frequently studied (9 studies). No studies were set in Africa. Geographic scales ranged from a single small city (population about 55,000) (Huttunen *et al.*, 2012) to the entire globe (Johnston *et al.*, 2012). Most studies focused on cities or regions close to fire events.

Study design

The majority of studies were based on either spatially or temporally aggregated populations, such as ecological studies (37 of 61 studies). There were relatively fewer cohort or panel studies (14 of 61 studies). Most of the studies compared the risk of health outcomes between 1) periods with no fire events and periods during or after the fire events, or 2) regions not affected by wildfire smoke and regions affected by wildfire smoke. The selection of model adjustment variables was not universal, but can be classified as 1) meteorological; 2) air pollutants other than the pollutants of interest; 3) community-level socio-demographics; and 4) temporal effects (seasonal or secular trend). Of these, meteorological factors were the

most prevalent adjustment variables. Some studies controlled for individual variables, such as age group and sex, by stratification (Analitis *et al.*, 2012; Castro *et al.*, 2009; Delfino *et al.*, 2009; Frankenberg *et al.*, 2005; Henderson *et al.*, 2011; Mott *et al.*, 2005; Nunes *et al.*, 2013; Prass *et al.*, 2012; Rappold *et al.*, 2011; Sarnat *et al.*, 2008)

Health outcomes investigated and outcome assessment

Respiratory disease was the most frequently studied outcome (45 studies (74% of 61 studies)) (Supplementary Table A.4). The outcomes included contacts with emergency departments (ED), hospitals or other primary care providers (33 studies (54%)), respiratory symptoms or lung function measurements (9 studies (15%)), and dispensation or consumption of medication (three studies (5%)). Relatively few studies examined cardiovascular morbidity (14 studies) or mortality (13 studies) (Table 2).

Other outcomes investigated were diarrhea due to power outage after wildfire events (identified from surveillance records), birth weight (obtained from hospital birth records), blood biomarkers for systemic inflammation and bone marrow content. The studies of lung-function, blood biomarker concentration and bone marrow content were all cohort studies measuring subjects' lung function or blood samples both before and after fire events.

The most common source of information for health outcomes was the use of datasets maintained by governmental agencies or statistical bureaus (32 studies), followed by hospital admission records or billing records (19 studies), interviews or surveys (10 studies), and subject tests such as lung function or blood samples (seven studies). Some studies used multiple methods to assess health outcomes. All mortality data came from governmental agencies or bureaus. Use of individual surveys (*e.g.*, "smell of wildfire smoke indoors" (Kunzli *et al.*, 2006)) was the most employed method in assessing personal exposure and self-reported symptoms for short-term studies.

Exposure assessment

The most commonly used method for either designating a fire period or area, or assessing exposure for previously designated fire and non-fire periods or areas, was use of measurements from land-based air pollutant monitors (35 studies), followed by satellite-based imagery or models (11 studies), air quality modelling (six studies) and personal exposure from individual surveys, personal reports, or personal photometers (three studies) (Supplementary Table A.3). Of the 61 studies, seven studies used other methods to assess exposure, such as air sample analysers. Satellite-based methods became popular in studies from recent years.

Pollutant data from air monitors were usually obtained by governmental agencies or research institutions and were used as the exposure variable in statistical models. The monitoring data usually covered pre-, during- and post-fire periods. Most of the studies determined "exposed period" based on the start/end dates of fire events but did not specify how the start/end days were identified. Some studies used thresholds of air monitoring data to categorize days, for example, high PM days with aerodynamic diameter <2.5μm ($PM_{2.5}$) >40μg/m^3, low PM days with $PM_{2.5}$<10μg/m^3 (*e.g.*, Johnston *et al.*, 2002). Personal surveys

and reports generally asked questions such as “did you smell any smoke?” or “did you have any health symptoms?” plus the respondents’ personal characteristics, such as age and education. Personal photometers were used to measure personal exposure to $PM_{2.5}$ (Huttunen *et al.*, 2012).

Satellite-based imagery or models are increasingly common in the recent studies to aid exposure assessment. Some satellite-based studies used satellite images to detect “hotspots”, which were used as indicators of fire events (*e.g.*, Castro *et al.*, 2009; de Mendonca *et al.*, 2006)). Some studies determined “exposed region” based on either satellite images or proximity to fire events (*e.g.*, Kunii *et al.*, 2002). The majority of the studies using satellite-based methods measured exposure for at least 5 years. In contrast, studies using individual photometers or reports usually investigated individual-specific exposure among subjects of a prospective cohort for a shorter period of a few days to a few months (Frankenberg *et al.*, 2005; Kunii *et al.*, 2002; Kunzli *et al.*, 2006).

The length of exposure measurement varies from a few days to over a dozen years. Huttunen *et al.* assessed daily average exposure of $PM_{2.5}$ and PM with aerodynamic diameter < 10μm (PM_{10}) during a 12-day fire that occurred in Kotka, Finland from Apr. 25 to May 6, 2006 (2012). Many studies compared longer-term exposure across months or seasons (Hanigan *et al.*, 2008; Johnston *et al.*, 2007; Smith *et al.*, 1996). Elliott *et al.* (2013) measured exposure during fire seasons (Apr. 1 to Sep. 30) in each year (2003–2010) and compared the health risk during fire seasons with non-fire seasons. Evaluation of long-term exposure was more common in regions with distinct fire seasons, such as Australia (*e.g.*, Hanigan *et al.*, 2008; Johnston *et al.*, 2011; Morgan *et al.*, 2010; Smith *et al.*, 1996) and Canada (Elliott *et al.*, 2013). Johnston *et al*. (2011) investigated long-term mortality effect by measuring PM_{10} exposure attributed to wildfires over 13.5 years, from 1994 to 2007 in Sydney, Australia.

Other studies compared exposure and health during the period when forests were burning to the periods before and/or after the fire (Supplementary Table A.3). Of these studies, Duclos *et al.* (1990), Frankenberg *et al.* (2005), and Moore *et al.* (2006) compared exposure and health during the fire events or seasons with control periods in preceding and/or subsequent years. Many studies estimated short-term (*e.g.*, a few days to one or two weeks) exposure under a certain fire event and compared the health risk during the fire event with that during short pre- or post-fire periods (*e.g.*, Schranz *et al.*, 2010; Sutherland *et al.*, 2005; Vora *et al.*, 2011). This exposure timeframe was common in studies based on local populations and a single fire event. Many studies compared longer-term exposure across months or seasons (*e.g.*, Hanigan *et al.*, 2008; Johnston *et al.*, 2007; Smith *et al.*, 1996).

Almost all studies mentioned that air pollutant levels, especially particulate matter levels, increase dramatically during wildfire events. Figure 1 shows estimated air pollutant levels during fire periods compared with levels in control periods. $PM_{2.5}$ levels in most studies exceeded the U.S. EPA National Ambient Air Quality Standard for 24-hour $PM_{2.5}$ ($35\mu g/m^3$). Some studies indicated particulate levels during fire periods over 100 $\mu g/m^3$ for $PM_{2.5}$ and over 500 $\mu g/m^3$ for PM_{10} (*e.g.* Hänninen *et al.*, 2009; Holstius *et al.*, 2012; Kolbe and Gilchrist 2009; Kunii *et al.*, 2002)

3.1 Association between wildfire smoke and health outcomes

3.1.1 Respiratory morbidity—Of the health outcomes examined, respiratory morbidity had the strongest evidence of an association with wildfire smoke, with a statistically significant adverse association reported for 43 of the 45 respiratory studies (Supplementary Table A.4). Analysis of respiratory-related contacts with primary care providers constituted 31 studies that reported associations and 2 studies that did not detect an adverse association. ED contacts for asthma in Darwin, Australia were 2.4 (95% confidence interval 1.5–3.9) times greater on a fire day (PM_{10}>40μg/m^3) than on a non-fire day (PM_{10}<10 μg/m^3) (Johnston *et al.*, 2002). Two other Australian studies reported greater risk of hospital admission for elevated exposure two days before the hospital admission day (Morgan *et al.*, 2010) and five days before the admission day (Chen *et al.*, 2006). Associations for longer lags (greater than five days) between exposure and hospitalization were not directly investigated in any study. From cross-sectional studies there were increases in primary care contacts for a 12-week period of exposure to wildfire smoke in California (Lee *et al.*, 2009) and a five-week exposure period in Canada (Moore *et al.*, 2006) compared to the same period in previous years when there were no fires. However, it remains unclear as to whether admissions increased due to high acute exposures over short periods (days) and/or lower levels accumulated over a longer period (months). Associations were consistently reported between wildfire related exposure and respiratory symptoms or dispensation/use of medication (all 12 studies). Adverse associations were observed for cough, wheeze and eye irritation (Supplementary Table A.4).

A statistically significant association between exposure to wildfire smoke and hospital or emergency room admissions for respiratory diseases was not reported in two of the 45 studies (Azevedo *et al.*, 2011; Smith *et al.*, 1996). A study of Sydney compared ED records in seven hospitals during a two-week fire period with that during the same period in the previous year. The researchers found no difference in asthma ED visits during the two periods (Smith *et al.*, 1996). The Northern Portugal study reported that high ozone level (greater than 100μg/m^3) during the three-month fire period was not associated with respiratory disease admissions.

3.1.2 Cardiovascular morbidity—Of the 14 studies that assessed the relationship between wildfires and cardiovascular morbidity, six reported a statistically significant increase in risk of cardiovascular outcomes with exposure to wildfire smoke. Some authors reported change in risk per unit (such as per 100 μg/m^3) increase in daily measurement of certain wildfire-promoted pollutants, such as ozone, PM_{10} or $PM_{2.5}$ (Azevedo *et al.*, 2011; Lee *et al.*, 2009; Rappold *et al.*, 2012). Others reported changes in risks comparing regions or time periods of wildfires with non-wildfire regions or times (Delfino *et al.*, 2009; Rappold *et al.*, 2011). PM_{10} was the most commonly studied pollutant for cardiovascular diseases and most of the PM_{10}-CVD studies (eight out of nine) did not find any significant association. Other air pollutants from wildfires were less studied and their impact on cardiovascular illness remains unclear. Study findings varied geographically, with no report of a statistically significant cardiovascular impact of wildfire smoke in any study from Australia and Canada (seven out of 14) (Crabbe 2012; Hanigan *et al.*, 2008; Henderson *et al.*, 2011; Johnston *et al.*, 2007; Martin *et al.*, 2013; Moore *et al.*, 2006; Morgan *et al.*,

2010). Contrastingly, five out of six U.S. studies reported that exposure to wildfire smoke was associated with hospital admissions for cardiovascular diseases, such as cardiac arrests, or symptoms such as chest pain (Delfino *et al.*, 2009; Lee *et al.*, 2009; Rappold *et al.*, 2012; Rappold *et al.*, 2011). All studies assessed cardiovascular disease by hospital admissions or emergency room visits. A U.S. study found that a 100μg/m^3 increase in wildfire smoke-related $PM_{2.5}$ was associated with a significant 42% (95%CI: 5%–93%) increase in emergency room visits for congestive heart failure (CHF) (Rappold *et al.*, 2012). However, there were too few studies on specific cardiovascular endpoints, such as ischemic heart disease (*e.g.*, Azevedo *et al.*, 2011; Crabbe 2012; Moore *et al.*, 2006) to establish consistency of associations.

3.1.3 Mortality—Mortality was associated with wildfire smoke for nine of 13 studies. Only three of these studies assessed non-accidental mortality (Analitis *et al.*, 2012; Johnston *et al.*, 2011; Vedal and Dutton 2006). Two investigated cause-specific mortality for respiratory and COPD (Castro *et al.*, 2009; Nunes *et al.*, 2013). Other studies examined total all-cause mortality. The increase in mortality under exposure to wildfire smoke, compared with periods of no fires, ranged from 1.2% for children during the fire event (Jayachandran 2009) to 92.0% for respiratory mortality during days with large fires (Analitis *et al.*, 2012). Large fires (>3000 hectares burned) had larger estimated associations with mortality than smaller fires (Analitis *et al.*, 2012). As wildfire events occur more often in summer, Shaposhnikov *et al.*, (2014) examined the interaction between heat and wildfire smoke. They found that temperature and PM_{10} (largely due to wildfires) collectively contributed to over 2000 deaths. One of the three studies that investigated shorter-term exposure and did not report a statistically significant association did not provide numeric results (Vedal and Dutton 2006) while the effect estimates reported in the other two studies were in the positive direction, i.e., adverse mortality effects (Hänninen *et al.* (2009) and Morgan *et al.* (2010)).

3.1.4 Other health outcomes—Eleven studies investigated other health outcomes in relation to wildfire smoke. These included studies on birth weight (Holstius *et al.*, 2012; Prass *et al.*, 2012), bone marrow content (Tan *et al.*, 2000), systematic inflammation (Huttunen *et al.*, 2012), physical strength and overall health (Frankenberg *et al.*, 2005), diarrhea (Viswanathan *et al.*, 2006), diabetes (Lee *et al.*, 2009), and injuries (Cameron *et al.*, 2009; Cleland *et al.*, 2011). For the two studies that investigated birth weight, results were inconsistent (Holstius *et al.*, 2012; Prass *et al.*, 2012). All three cohort studies reported significant adverse associations between wildfires and health: systemic inflammation (Huttunen *et al.*, 2012), bone marrow content (Tan *et al.*, 2000), and physical strength and overall health (Frankenberg *et al.*, 2005). Diarrhea and diabetes were mentioned as health outcomes of interest in multiple studies (Aditama 2000; Jalaludin *et al.*, 2000; Lee *et al.*, 2009; Viswanathan *et al.*, 2006), but only two reported the results (Lee *et al.*, 2009; Viswanathan *et al.*, 2006). Exposure to wildfire smoke did not show discernible effects on either diarrhea or diabetes.

Vulnerable sub-populations: A limited number of studies assessed whether some populations face higher health risk from exposure to wildfire smoke than others, examining population characteristics such as age categories. The age cut-offs for age categories varied

by study. Larger positive associations between wildfire smoke and cardiorespiratory morbidities were observed for middle-aged adults (Henderson *et al.*, 2011) and older adults compared to other age groups (Analitis *et al.*, 2012; Castro *et al.*, 2009; Delfino *et al.*, 2009; Frankenberg *et al.*, 2005; Morgan *et al.*, 2010; Nunes *et al.*, 2013; Shaposhnikov *et al.*, 2014). Elevated levels of wildfire smoke had larger risk estimates for asthma hospitalizations among adults aged 40–64 years (Mott *et al.*, 2005), 15–64 years (Morgan *et al.*, 2010), and 19–64 years (Rappold *et al.*, 2011) compared to other age groups. Risk of respiratory-related hospital contacts associated with wildfire smoke was higher for children (<5 years) compared with other age groups (Ignotti *et al.*, 2010).

Men and women may have different health risks when exposed to wildfire smoke. Risks for asthma-related symptoms or visits in relation to wildfire smoke were greater for women than men (Lee *et al.*, 2009; Rappold *et al.*, 2011). However, Henderson *et al*. (2011) and Prass *et al.* (2012) did not find differences in wildfire effect estimates between men and women in respiratory and cardiovascular physician visits, and birth weight, respectively.

Three studies reported effect modification by socio-economic status (SES), race, or co-morbidities. Larger risk estimates between wildfire smoke and risk of asthma and congestive heart failure were observed among counties of lower SES compared to higher SES counties (Rappold *et al.*, 2012). Aboriginal Australians had higher risk of respiratory admissions and emergency admissions than other races when exposed to PM_{10} (Hanigan *et al.*, 2008; Johnston *et al.*, 2007). Johnston *et al.*, (2007) did not detect an association between PM_{10} and cardiovascular admissions for the general population, but restriction of analyses to the Aboriginal population with ischemic heart disease resulted in findings of the greatest risk of respiratory-related hospital admissions three days after exposure (Johnston *et al.*, 2007). It is plausible that associations at longer lags might have only been observable for such high-risk sub-populations, most susceptible to wildfire. Lee *et al.* (2009) and Mirabelli *et al.*, (2009) reported that adults with pre-existing respiratory conditions or weakness (i.e. small airway size) were more likely to seek care or have additional symptoms after wildfire exposure than persons without those conditions. However, Künzli *et al.* (2002) reported opposite results, as children without pre-existing asthmatic conditions had greater increase in respiratory symptoms under exposure than did other children. The authors suggested that children with pre-existing asthmatic conditions tended to be on medication and have better access to care, hence their smaller increase in symptoms when exposed to wildfire smoke. In an Australian study, no adverse association was observed between wildfire related PM_{10} and lung function (peak expiratory flow) except when analysis was restricted to children with no bronchial hyper-reactivity (Jalaludin *et al.*, 2000).

4. Discussion

Overall, wildfire smoke exposures, as measured by proxies such as criteria air pollutants, were consistently associated with mortality and respiratory morbidities. Respiratory-related effects of wildfire smoke included increases in risk of hospitalization, use of respiratory medication, cough, wheeze and eye irritation. In one study, risk of emergency department contact for asthma could be more than two times greater after exposure to wildfire smoke (Johnston *et al.*, 2002). As most mortality studies investigated all-cause mortality, further

research is needed to better identify the specific causes of mortality most strongly associated with wildfire smoke exposures. The magnitude of the effects on mortality varied by study. Respiratory mortality almost doubled from exposure to a wildfire in Greece (Analitis *et al.*, 2012), but some wildfires were not associated with changes in the mortality rate (Morgan *et al.*, 2010). The only global study posited that 339,000 deaths per year were attributable to wildfires, with Sub-Saharan Africa and Southeast Asia the most affected regions (Johnston *et al.*, 2012). However, this review highlighted disproportionately fewer studies in Southeast Asia and no other studies conducted in Sub-Saharan Africa. Some parts of the world such as Sub-Saharan Africa are affected by wildfire events but have not been studied. Those places, usually the less-developed regions, may contribute the most to the global burden of many diseases. It is also unlikely that these parts of the world can respond to such risk as well as more developed nations. Therefore, more studies are needed in these less studied countries.

Although our review of studies on forest fires and health is the most extensive to date, past reviews on related topics have also contributed substantially towards knowledge on the health effects of wildfire smoke. An early review by Naeher *et al.* (2007) focused on the toxicity of wood smoke, thereby establishing biological plausibility of the association, and called for further studies on the topic. Two later reviews investigated effects on respiratory outcomes of bushfire smoke (Dennekamp and Abrahmson 2011) and on respiratory outcomes for forest fires (Henderson and Johnston 2012). Dennekamp and Abramson (2011) identified that elevated PM concentrations from bushfire smoke explained associations with increased respiratory morbidity. Henderson and Johnston (2012) confirmed consistency of associations with acute respiratory outcomes and identified the need for studies in equatorial regions with rainforest depletion. Finlay et al. (2012) included non-respiratory outcomes and focused on demonstrating the current stage of investigation on this issue in the U.K. and identified literature gaps for the U.K. Finlay et al. identified the potential burden on cardiovascular and ophthalmic outcomes. Our review confirms that there still remain too few studies on these endpoints to establish consistency. The findings of our comprehensive review add to those of the previous reviews that focused on specific types of wildfire, health outcomes, or countries. Our review also quantified the substantial increase in exposure levels from wildfires and how these increases differed across studies. This was the first review to identify the dearth of studies from sub-Saharan Africa and paucity of studies in Southeast Asia, which are regions that experience a large health burden and are less able to respond to the increasing frequency and intensity of wildfires that accompany climate change. Our review also identified the shift in exposure assessment from the dominant use of measurements from ground-based air monitors to use of satellite imagery and chemical transport models.

In our review we found that results were most consistent among cohort studies, as almost all cohort studies found significant impact of wildfire smoke on health in at least one of the health outcomes and part of the population studied. Studies involving direct physiological measurements on recruited patients, such as bone marrow (Tan *et al.*, 2000) and Peak Expiratory Flow Rates PFFR (e.g. Jalaludin *et al.*, 2000), also tend to discern significant impacts. Ecological studies generally had inconsistent results. However, it is difficult to draw conclusions as to how study design and methods affected the reported associations

because of heterogeneity in these and other design factors across studies, significant difference between pollutant levels during wildfire and non-wildfire periods, and how this difference varied across studies.

Studies consistently reported substantially higher levels of air pollution during fire periods and locations compared to non-fire periods and areas. Daily average PM_{10} levels in an exposed city (Jambi, Indonesia) exceeded 1800μg/m^3 during fire events (Kunii *et al.*, 2002), which was 12 times the WHO interim target-1 standard (150μg/m^3 24-hour) and 36 times the WHO air quality guideline (50μg/m^3 24-hour). Daily average $PM_{2.5}$ levels during wildfires exceeded 150μg/m^3, more than 6 times greater than the WHO air quality guideline (25μg/m^3 24-hour) (Moore *et al.*, 2006). Levels of carbon monoxide can increase 30–40% during wildfire periods compared with periods with no fires (Sutherland *et al.*, 2005; Tan *et al.*, 2000). These results indicate that wildfire events can result in severe levels of exposures. In addition to high levels, the chemical composition of wildfire smoke is distinctive. Wildfire smoke is accompanied by elevated levels of black carbon (Crabbe 2012), and polycyclic aromatic hydrocarbons can be 15 times higher than background levels (Aditama 2000).

4.1 Methods used to assess exposure to wildfire smoke

This review identified assessment of exposure as a key challenge in health studies of wildfires, with a range of methods applied. It is difficult to identify a direct marker that can represent air pollutants only from wildfires. Studies used indicators such as criteria air pollutants, aerosol optical depth or area burnt as indirect proxies. Although use of indirect proxies can be a useful approach, it is difficult to ascertain the fraction of health morbidity due to wildfire smoke excluding health morbidities due to those proxies in non-wildfire periods and from other sources during wildfire periods. The most commonly used marker for wildfire smoke used in the reviewed studies was particulate matter (PM) (Phuleria *et al.*, 2005). Although the fine fraction of particulate matter ($PM_{2.5}$) has been more consistently associated with adverse health effects than larger particles in studies of particulate matter more generally (Pope and Dockery 2006), fewer studies investigated the health effects of wildfire smoke-related $PM_{2.5}$. Notably, in all countries, the measurement of $PM_{2.5}$ began more recently than PM_{10}. A further exposure-related limitation of many of the reviewed studies was the coarse spatial resolution of exposure, due primarily to the use of ground-based ambient air monitors and the available monitoring network. An exception to this was studies that used remotely sensed satellite-derived imagery of area burnt (de Mendonca *et al.*, 2006). However, it is unclear as to whether area burnt is a suitable proxy for wildfire smoke exposure because it must be interpreted relative to population's distance to the wildfire, wind speed and direction, and atmospheric mixing depth (Naeher *et al.*, 2007; Ward 1990). Wildfire smoke also varies with vegetation type as, for example, wood from eucalypt forest has more oil content and releases higher concentrations of PM_{10} than pine, acacia or cork oak (Goncalves *et al.*, 2010).

Exposure assessment is an ongoing challenge in epidemiological studies of wildfire smoke. Ground-based monitors do not measure the complicated mixture of pollution from the source of wildfires specifically. Monitors measure the level of a specific pollutant, such as

$PM_{2.5}$, and cannot measure the pollution solely from fires as opposed to other sources. Therefore, it is difficult to separate the health effect of wildfire-emitted pollutants from that of pollutants from other sources. Moreover, ground-based air pollution monitors are not located in all places or time periods with affected populations. Exposure estimates based on satellite data provide more comprehensive spatial coverage (Kloog *et al.*, 2011; Lee *et al.*, 2011), but do not address the issue of specificity of the exposure estimates for wildfire smoke. It is critical to better understand the levels of wildfire smoke-specific pollutants (e.g., particulate matter from wildfires), as the range of health responses to the chemical signature specific to wildfire smoke is currently unclear (Wegesser *et al.*, 2009). Recent developments in chemical transport models may help address this limitation in future work. Chemical transport models, such as GEOS-Chem models, can estimate air pollutants specifically from wildfires (e.g. Singh *et al.*, 2010). Johnston et al (2012) employed this method to estimate the global exposure to wildfire-emitted $PM_{2.5}$. They found that 339,000 deaths could be attributed to wildfires annually. One limitation of using chemical transport models is that the wildfire-specific pollutant estimates may be difficult to validate. Modeled data could also be computationally expensive and requires collaboration efforts of atmospheric scientists (Kleeman *et al.*, 2009).

4.2 Health outcomes affected by wildfire smoke

The health endpoints investigated by the reviewed studies mainly focused on mortality and respiratory morbidity. Over 90% of the studies on respiratory morbidity and about 70% of the studies on mortality found significant association with wildfire smoke. There was insufficient evidence to conclude a consistent association between wildfire smoke and cardiovascular morbidities due to the relatively fewer number of studies. Despite the inconsistent association for cardiovascular morbidities globally, the association was mostly consistent in North America (five out of six studies found significant impact), where prevalence of cardiovascular diseases are higher than many other study areas. Causal links have been established between PM_{10} more generally and a range of cardiovascular endpoints (Brook *et al.*, 2010). Other potential health endpoints that have been studied in the context of air pollution are hypertensive disorders (*e.g.* van den Hooven *et al.*, 2011), ophthalmic outcomes (*e.g.* Versura *et al.*, 1999), adverse pregnancy outcomes (*e.g.* Ritz *et al.*, 2002), and non-respiratory atopic disease (Morgenstern *et al.*, 2008). Future studies on the health impacts from wildfires may investigate these outcomes.

4.3 Susceptibility/Vulnerability

Among other factors, variation in the magnitude and statistical significance of observed effect estimates across the reviewed studies was likely attributable, in part, to differences in the underlying characteristics of the study population, including biological susceptibility, sociodemographic vulnerability, or other factors. Air pollution research more broadly has acknowledged population characteristics that can lead to greater biological susceptibility or sociodemographic vulnerability (Gouveia and Fletcher 2000). However, for wildfire smoke exposure, our review identified a paucity of studies on potentially vulnerable/susceptible subpopulations. There was some indication of elevated vulnerability to adverse health-effects of wildfire smoke among certain sub-populations: young children, older adults, and individuals of lower socioeconomic status. It is plausible that individuals with pre-existing

respiratory morbidities are more susceptible to the respiratory effects of wildfire smoke possibly due to elevated sensitivity to environmental hazards by weaker immune systems. Pre-existing morbidities, such as asthma, that may not be fully controlled by medication might lead to greater susceptibility to adverse health effects of wildfire smoke. Although not specific to wildfire smoke, PM_{10} has been associated with poorly controlled asthma among adults (Jacquemin *et al.*, 2012) and the effect of air pollutants on respiratory exacerbation among asthmatic children appears to be greater for those not on anti-inflammatory medication (Delfino *et al.*, 2002).

In the identified studies, five of six U.S. studies reported associations between wildfire smoke and cardiovascular hospital admissions, whereas associations were not observed in studies for other locations, including Australia and Canada. Cardiovascular diseases are more prevalent in U.S. adults (more than 1 in 3 adult Americans have cardiovascular diseases) (Lloyd-Jones *et al.*, 2010) than in Australia (about 1 in 6) (The Heart Foundation 2011). The mortality rates due to cardiovascular diseases are also higher in the U.S. than in Canada or Australia (Lloyd-Jones *et al.*, 2010). The different findings by region may result from higher risk for cardiovascular responses from wildfire smoke for population with high CVD prevalence.

4.4 Recommendations for future research

More studies in wildfire-affected but less-developed regions, such as Africa and Southeast Asia are needed. These regions face the highest health risk to wildfire smoke because they lack well-developed health care infrastructure and resources (Watson *et al.*, 2007). They are also less able to adapt to climate change compared to the developed world (Matthes 2008), leading to even higher risk to wildfires in the future. The populations are particularly vulnerable because behavioral interventions are complex (e.g., remaining indoors might increase exposure due to use of solid fuels, and chronic exposure to indoor solid fuels can lead to higher susceptibility to respiratory diseases (Po *et al.*, 2011)) (Smith *et al.*, 2004).

More large-scale studies are needed to obtain more reliable results on health impact of wildfires. Most of the identified studies were based on single-episode fire events, with fewer long-term studies. Studies based on multiple-episode fire events might be useful to identify consistency of an association over time or change in vulnerability or behavioral adaptation (*e.g.*, remaining indoors) to wildfire smoke exposure. Similarly, most studies focused on local regions, with few studies at national or other large geographic scales. Investigating larger geographies will introduce greater sociodemographic variation that might reveal communities at the greatest risk of wildfire smoke-related health responses. Large-scale studies can also help policy-makers by identifying the most vulnerable communities and populations for policy reference.

In addition, future studies could also adapt more new technologies to advance exposure assessment. Chemical transport models, dispersion models and satellite-based models could help address the limitations of assessing wildfire smoke exposure using air monitors. Moreover, as wildfire potential has been projected to increase in the future (Liu *et al.*, 2010), studies that estimate future wildfire-related health impact are needed. In our review, no identified studies projected the future health risk from wildfires under climate change, or

identified high-risk regions or populations under future conditions. Studies projecting future health impact of wildfires can raise awareness of the health impact of wildfires in communities, promote preventive public health programs in high-risk communities, and aid in our understanding of the health consequences of a changing climate.

5. Conclusion

Our review indicates that wildfire events have potential to induce a substantial health burden. As wildfires are likely to occur more frequently and intensely under the impact of climate change, this health burden may increase in the future. Air pollution from wildfires was consistently associated with respiratory outcomes, and more studies are needed to investigate cardiovascular morbidity and mortality in community populations. Most of the current studies were based on single episodes and local populations. Conducting multiple episode and larger scale studies may reveal effects of wildfire smoke and help elucidate changes in wildfire frequency and possible adaptation. It was not possible to separate completely the health effect of wildfires from that of other ambient sources for the reviewed studies. Key challenges in current research include the assessment of exposure of wildfire-specific pollutants and the health risk modelling for source-specific air pollutant estimates. More research is needed to investigate the health effects of fine particulate matter from wildfires in Africa and Southeast Asia, the susceptible/vulnerable populations under exposure to wildfire smoke, and future health burden from wildfires under climate change.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Wildfire smoke dramatically increased ambient air pollutant levels
- Wildfire smoke consistently associated with increased risk of respiratory disease
- Suggestive evidence wildfire smoke linked with cardiovascular diseases & mortality
- Key challenge of exposure assessment: estimating fire-specific pollutants

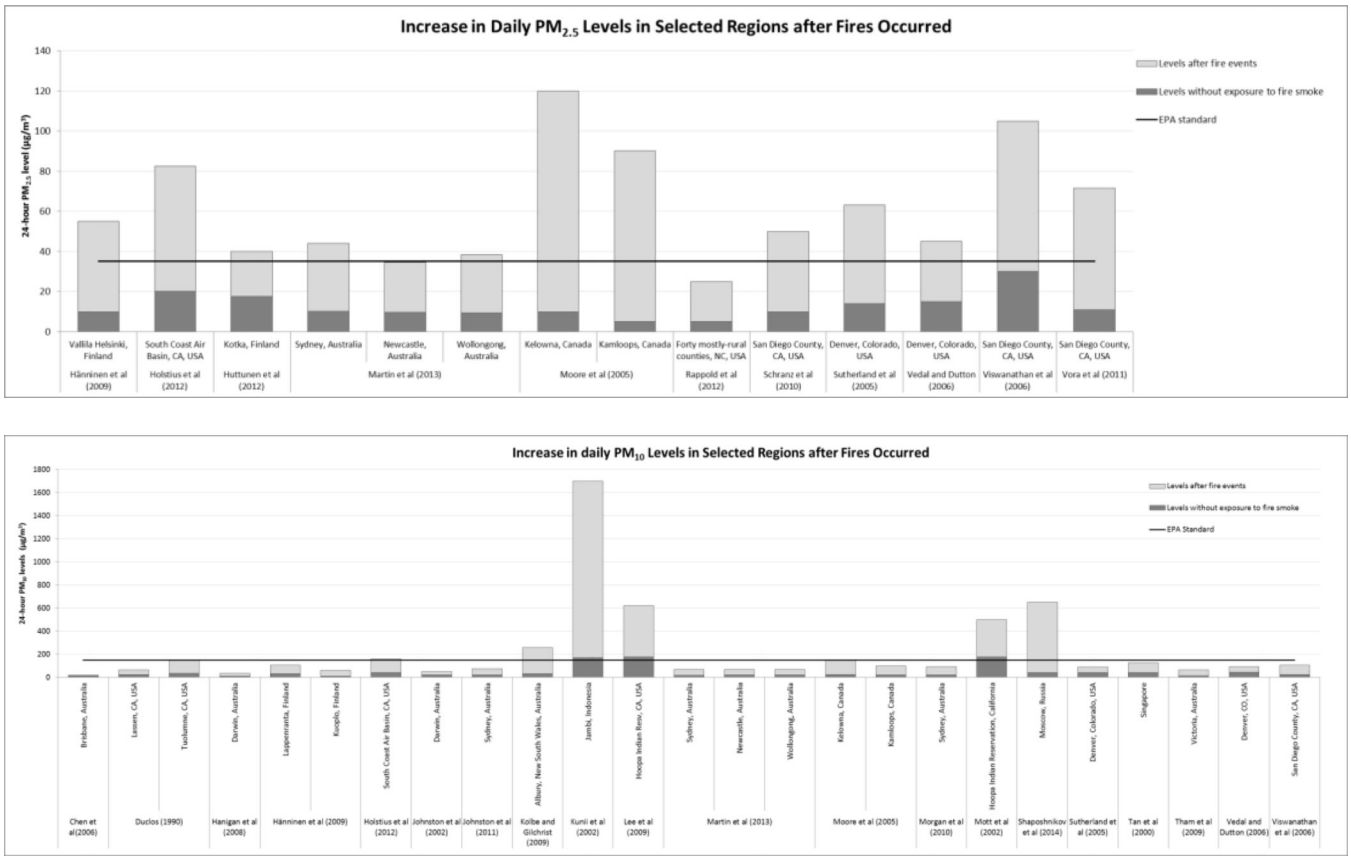

Figure 1.

$PM_{2.5}$ (top) and PM_{10} levels (bottom) during wildfire events and non-fire periods

Table 1

Summary of studies on wildfire smoke and population health

Study	Location	Background population or cohort size	Time of fire	Major health outcome	Exposure metric
Aditama (2000)	Multiple provinces in Indonesia	12,360,000 residents exposed to smoke	major fire: July–Oct. 1997	Respiratory symptoms	CO, SO_2, PM_{10}, TSP, NOx, O_3, organic compounds
Analitis *et al*., (2012)	Athens, Greece	More than 3 million residents	1994–2004	Mortality	Sizes of area burned
Azevedo *et al*., (2011)	Northern coast of Portugal	Elderly among Porto (total population 1.4 million)	June to Aug. 2005	Cardiovascular (CVD), respiratory admissions	O_3
Caamano-Isorna et al., (2011)	Galicia, Spain	About 2 million inhabitants	Summer 2006	Respiratory medicine usage	Exposure classified into three categories based on number of fires
Cameron *et al*., (2009)	Victoria, Australia	5.2 million residents	Feb. 2009	Injuries	Not specified
do Carmo *et al.*, (2010)	Alta Floresta municipality, Mato Grasso, Brazil	51,136 residents in Alta Floresta, Mato Grosso (9% children <5y, 5% elderly >64y)	Jan. 2004 – Dec. 2005	Respiratory admissions	$PM_{2.5}$
Castro *et al*., (2009)	State of Rondônia, western Brazil	1.6 million residents	1998–2005	Mortality	Number of fire “hotspots”
Centers for Disease Control and Prevention (CDC) (1999)	Central Florida	Not specified	Jun.– Jul. 1998	Respiratory and cardiovascular Emergency Room (ER) visits	Wildfire v. non-wildfire periods
Centers for Disease Control and Prevention (CDC) (2007)	Panhandle region and 9 other counties, Texas, U.S.	Not specified	March 12–20, 2006	Mortality	Presence of wildfire smoke
Centers for Disease Control and Prevention (CDC) (2008)	San Diego Co., California, U.S.	Not specified	Oct. 22–26, 2007	Respiratory ER visits	Wildfire v. non-wildfire periods
Chen *et al*., (2006)	Brisbane, Australia	Not specified	Fire seasons 1997–2000	Respiratory admissions	PM_{10}
Cleland *et al*., (2011)	Melbourne, Australia	Not specified	Feb. 2007	Injuries	Not specified
Crabbe (2012)	Darwin, Australia	110,000 residents	1993–1998	Respiratory, CVD ER visits	PM_{10}, black carbon
Delfino *et al*., (2009)	Southern California, U.S.	20.5 million residents	Oct. 21–30, 2003	CVD, respiratory admissions	$PM_{2.5}$
Dohrenwend *et al*., (2013)	San Diego Co., California, U.S.	Not specified	Oct 21–Nov 6, 2007	Respiratory ER visits	Wildfire v. non-wildfire periods
Duclos *et al*., (1990)	6 counties in California, U.S.	Residents in 6 counties (population size not specified)	Aug. 30–Sep. 3, 1987	Respiratory ER visits	PM_{10}, TSP

Study	Location	Background population or cohort size	Time of fire	Major health outcome	Exposure metric
Elliott *et al.*, (2013)	British Columbia (BC), Canada	Residents from 29 local health areas (LHA) in BC; population ranges 7,024–352,783 people	Fire seasons 2003–2010	Respiratory medicine usage	$PM_{2.5}$, PM_{10}
Emmanuel (2000)	Singapore	> 3 million residents	End of Aug. to early Nov. 1997	Respiratory admissions; all-cause mortality	PM_{10}, SO_2, NO_2, O_3, CO, total hydrocarbon
Frankenberg *et al.*, (2005)	Kalimantan and Sumatra, Indonesia	10,869 subjects > 30y	July–Oct., 1997	Respiratory illness/symptoms; physical strength, overall health	Aerosol
Hanigan *et al.*, (2008)	Darwin, Australia	110,000 residents	Dry seasons (Apr. – Nov.) of 1996–2005	Respiratory, CVD admissions	PM_{10}
Hänninen *et al.*, (2009)	11 provinces in southern Finland	3.4 million residents	Aug. 26–Sep. 8, 2002	Mortality	$PM_{2.5}$, PM_{10}
Henderson *et al.*, (2011)	Southeastern corner of BC, Canada	281,711 subjects	Summer 2003	CVD, Respiratory admissions	PM_{10}
Holstius *et al.*, (2012)	South Coast Air Basin, California, U.S.	886,034 infants in exposed group; 747,590 infants in control group	Oct. 2003	Birth weight	Exposed or unexposed to fire during pregnancy
Huttunen *et al.*, (2012)	Kotka, Finland	52 elderly people (>50 y) with ischemic heart disease	Apr. 25–May 6, 2006	Blood concentration of inflammatory markers	$PM_{2.5}$
Ignotti *et al.*, (2010)	Microregions in northern states of Brazilian Amazon, with Mato Grosso and Maranhão	24 million inhabitants affected; subpopulations: Children (<5 y), elderly (>64), and an intermediate age group (5–64 y)	2004–2005	Respiratory admissions	$PM_{2.5}$
Jalaludin *et al.*, (2000)	Sydney, Australia	32 children	Jan. 1994	Peak expiratory flow rates (PEFR)	PM_{10}, NO_2, O_3
Jayachandran (2009)	Indonesia	~1.3 million children (<3 y), infants or fetuses	Aug.–Oct. 1997	Mortality	Aerosols
Johnston *et al.*, (2002)	Darwin, Australia	115,000 residents	Apr. 1–Oct. 31, 2000	Asthma ER visits	PM_{10}
Johnston *et al.*, (2006)	Darwin, Australia	251 asthmatic adults and children, about half < 18y	7 months in 2004	Asthmatic symptoms	$PM_{2.5}$, PM_{10}
Johnston *et al.*, (2007)	Darwin, Australia	110,000 residents	Fire seasons of 2000, 2004, 2005	Respiratory, CVD admissions	PM_{10}
Johnston *et al.*, (2011)	Sydney, Australia	~ 4 million residents	1994–2007	Mortality	PM_{10}, O_3
Johnston *et al.*, (2012)	Global	Not specified	1997–2006	Mortality	$PM_{2.5}$
Kolbe and Gilchrist (2009)	Albury, New South Wales, Australia	389 interviewees	Jan–Feb, 2002	Respiratory symptoms	PM_{10}
Kunii *et al.*, (2002)	Jambi, Sumatra (affected) and Jakarta, Java (control), Indonesia	543 subjects in Jambi	July–Oct. 1997	Respiratory symptoms	CO, CO_2, SO_2, NO_2, O_3, PM_{10}, inorganic ions, PAHs

Study	Location	Background population or cohort size	Time of fire	Major health outcome	Exposure metric
Kunzli *et al.*, (2006)	16 communities in Southern California, U.S.	873 high school students, 5551 elementary school students	Oct. 2003	Respiratory symptoms	PM_{10}
Lee *et al.*, (2009)	Hoopa Indian Reservation, California, U.S.	2,633 residents	Late summer and fall 1999	Respiratory, CVD, diabetes admissions	PM_{10}
Martin *et al.*, (2013)	Sydney, Newcastle and Wollongong, Australia	About 4.5 million residents	Fire seasons 1994–2007	All non-trauma admissions	PM_{10}, $PM_{2.5}$
Mascarenhas *et al.*, (2008)	Rio Branco, Brazil	19,581 ER visits	Sep. 1–30, 2005	Respiratory ER visits	$PM_{2.5}$
de Mendonca *et al.*, (2006)	261 districts in Brazilian Amazon	Residents in Amazon regions (population size not specified)	Fire seasons 1996–2000	Respiratory admissions	hot pixels from satellite data
Mirabelli *et al.*, (2009)	12 counties in California, U.S.	465 non-asthmatic students (16–19 y) in the Children's Health Study	Oct. – Nov. 2003	Respiratory symptoms	Number of days subjects smelled smoke
Moore *et al.*, (2006)	Kelowna and Kamloops regions in British Columbia, Canada	146,199 residents in Kaelowna; 100,548 residents in Kamloops	Aug. 2003	Respiratory, CVD	PM_{10}, $PM_{2.5}$
Morgan *et al.*, (2010)	Sydney, Australia	~ 3.48 million residents	Jan. 1994–June 2002	Respiratory admissions; Mortality	PM_{10}
Mott *et al.*, (2002)	Hoopa Reservation, California	289 residents in Humboldt Co. interviewed (26% of population)	Aug. 23–Nov. 3, 1999	Respiratory admissions	PM_{10}
Mott *et al.*, (2005)	Kuching, Malaysia	~400,000 residents affected	Aug. 1–Dec. 31, 1997	Respiratory symptoms	PM_{10}
Nunes *et al.*, (2013)	107 micro areas in Brazilian Amazon	Not specified	Dry season 2005	Mortality due to circulatory diseases	Annual % hours with $PM_{2.5}$ greater than 25μg/m^3
Prass *et al.*, (2012)	Porto Velho, Amazon region	22,012 live births	2001–2006	Birth weight	Number of fires
Rappold *et al.*, (2011)	42 contiguous counties in eastern North Carolina, U.S.	Not specified	June 2008	Respiratory, CVD ER visits	Aerosol optical depth (AOD)
Rappold *et al.*, (2012)	40 mostly rural counties, North Carolina, U.S.	Not specified	June to July, 2008	Asthma, CVD ER visits	$PM_{2.5}$
Sastry (2002)	Kuala Lumpur and Kuching, Malaysia	Not specified	July–Dec. 1997	Mortality	PM_{10}
Schranz *et al.*, (2010)	San Diego Co., California, U.S.	Not specified	Oct. 21–24. 2007	Respiratory ER visits	$PM_{2.5}$
Shaposhnikov *et al.*, (2014)	Moscow, Russia	11.5 million residents	Jul–Aug 2010	Mortality	PM_{10}, O_3
Shusterman *et al.*, (1993)	Alameda Co., California, U.S.	Not specified	Oct. 20–21, 1991	Respiratory, injury ER visits	Not specified
Smith *et al.*, (1996)	Western Sydney, Australia	907,450 residents	Jan. 5–12,1994	Respiratory, asthma ER visits	PM_{10}, NO_2

Study	Location	Background population or cohort size	Time of fire	Major health outcome	Exposure metric
Sutherland *et al.*, (2005)	Denver, Colorado, U.S.	21 residents who are >40 y, smoke, and with pre-existing COPD	June 8 to July 18, 2002	Respiratory symptoms	$PM_{2.5}$, PM_{10}, CO
Tan *et al.*, (2000)	Singapore	30 male volunteers	Sep.–Oct. 1997	Bone marrow content	SO_2, PM_{10}, NO_2, O_3; CO
Tham *et al.*, (2009)	Northeastern and Alpine district, Victoria, Australia	Not specified	Jan.–March, 2003	Respiratory ER visits	PM_{10}
Thelen *et al.*, (2013)	San Diego Co., California, U.S.	Not specified	Oct. 2007	Respiratory ER visits	$PM_{2.5}$, PM_{10}
Vedal and Dutton (2006)	Denver, Colorado, U.S.	~ 2 million residents	June 9–18, 2002	Mortality	$PM_{2.5}$, PM_{10}
Viswanathan *et al.*, (2006)	San Diego Co., California, U.S.	2.8 million residents	Oct. 2003	Respiratory, CVD, diarrhea admissions	$PM_{2.5}$, PM_{10}, O_3, NO_2, SO_2, CO
Vora *et al.*, (2011)	San Diego Co., California, U.S.	8 subjects in downtown San Diego with asthma	Oct. 2007	Respiratory function, rescue medication use	$PM_{2.5}$
(Wiwatanadate and Liwsrisakun (2011))	Chiang Mai, Northern Thailand	1.7 million residents	Aug. 2005 – June 2006	PEFR, asthma symptoms	CO, O_3, NO_2, SO_2, $PM_{2.5}$, PM_{10}

Table 2

Summary of studies based on health outcome and observed associations

	Total number of studies	Statistically significant associations observed	No statistically significant associations observed	Studies that found significant association
Blood biomarker concentration	1	1	0	Huttunen *et al*. (2012)
Asthma	5	4	1	Johnston *et al.* (2006); Martin et al (2013); Rappold *et al.* (2012); Johnston *et al.* (2002)
Birth weight	2	1	1	Holstius *et al*. (2012)
Bone marrow content	1	1	0	Tan *et al*. (2000)
Cardiovascular	14	6	8	Azevedo *et al*. (2011); CDC (1999); Delfino *et al.* (2009); Lee *et al.* (2009); Martin et al (2013); Rappold *et al*. (2011); Rappold *et al.* (2012)
Diabetes	1	0	1	
Diarrhea	1	0	1	
Injuries	3	3	0	Cleland *et al.* (2011); Cameron *et al.* (2009); Shusterman *et al.* (1993)
Mortality	13	9	4	Analitis *et al.* (2012); CDC (2007); de Castro, *et al.* (2009); Jayachandran (2009); Johnston *et al.* (2011); Johnston *et al.* (2012); Nunes et al, (2013); Sastry (2002); Shaposhnikov *et al.* (2014)
Opthalmic symptoms	5	5	0	Aditama (2000); Hänninen et al, (2009); Kunzli *et al.*,(2006); Mirabelli et al (2009); Viswanathan et al (2006)
PEFR	2	2	0	Jalaludin *et al.* (2010); Wiwatanadate and Liwsrisakun (2011)
Physical strength and overall health	1	1	0	Frankenberg *et al.* (2005)
Rescue medication use	3	3	0	Vora *et al.* (2011); Elliott *et al.* (2013); Caamano-Isorna (2011)
Other Respiratory diseases	37	35	2	Aditama (2000); Cardoso de Mendonça (2006); CDC (2008); Chen *et al.* (2006); Delfino *et al.* (2009); do Carmo *et al.* (2010); CDC (1999); Dohrenwend *et al*, (2013); Duclos, (1990); Emmanuel, (2000); Hanigan *et al.* (2008); Henderson *et al*. (2011); Ignotti *et al.* (2010); Kolbe and Gilchrist (2009); Kunii *et al.* (2002); Künzli *et al.* (2006); Lee *et al.* (2009); Martin *et al* (2013); Mirabelli *et al.* (2009); Moore *et al.* (2005); Morgan et al. (2010); Mott *et al.* (2002); Mott *et al*. (2005); Schranz *et al.* (2010); Sutherland *et al.* (2005); Viswanathan *et al.* (2006); Crabbe (2012); Frankenberg *et al.* (2005); Johnston *et al.* (2007); Mascarenhas *et al.* (2008); Shusterman et al. (1993); Tham *et al.* (2009); Thelen *et al* (2013); Rappold *et al.* (2011); Vora *et al.* (2011)