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

Jia C. Liu[1], Gavin Pereira[2], Sarah A. Uhl[1], Mercedes A. Bravo[1], Michelle L. Bell[1]

1 School of Forestry and Environmental Studies, Yale University, New Haven, CT, USA, 06511
2 Center for Perinatal Pediatric and Environmental Epidemiology, School of Medicine, Yale University, New Haven, CT, USA, 06511

Corresponding author: Jia C. Liu, 195 Prospect St, School of Forestry and Environmental Studies, Yale University, New Haven, CT, USA, 06511. Email: coco.liu@yale.edu. Phone: +1 (203)432-9869

Email addresses of authors:
Jia C. Liu: coco.liu@yale.edu
Gavin Pereira: gavin.pereira@yale.edu
Sarah A. Uhl: sarah.uhl@gmail.com
Mercedes A. Bravo: Mercedes.bravo@yahoo.com
Michelle L. Bell: michelle.bell@yale.edu

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

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 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 $\leq$2.5μm ($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 $\leq$ 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}>$40\mu g/m^3$) than on a non-fire day (PM_{10}<10 $\mu g/m^3$) (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 $100\mu g/m^3$) 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 $\mu g/m^3$) 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 100$\mu g/m^3$ 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

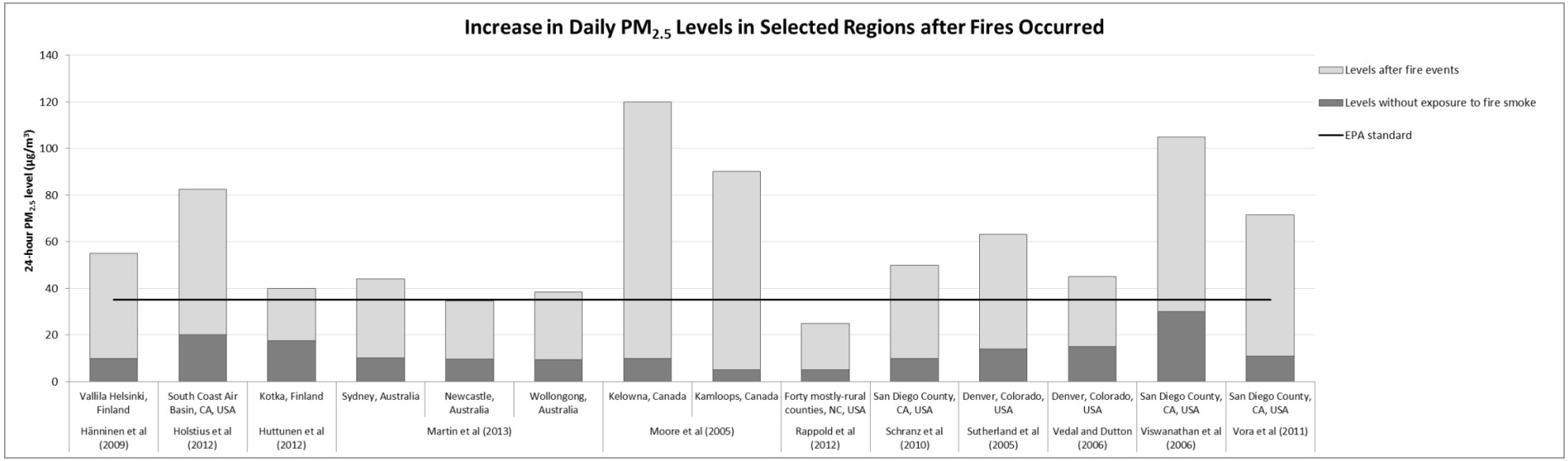
projected the future health risk from wildfires under climate change, or identified high-risk regions or populations under future conditions. Studies projecting future health impact of wildfires can raise awareness of the health impact of wildfires in communities, promote preventive public health programs 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.

6. Acknowledgements

This work was funded by NIH (R21ES021427), the U.S. EPA through the Harvard Clean Air Center (83479801), and the Yale Institute for Biospheric Studies.


Increase in Daily PM2.5 Levels in Selected Regions after Fires Occurred
24-hour PM2.5 level (µg/m3)
140
120
100
80
60
40
20
0
Levels after fire events
Levels without exposure to fire smoke
EPA standard
Vallila Helsinki, Finland
South Coast Air Basin, CA, USA
Kotka, Finland
Sydney, Australia
Newcastle, Australia
Wollongong, Australia
Kelowna, Canada
Kamloops, Canada
Forty mostly-rural counties, NC, USA
San Diego County, CA, USA
Denver, Colorado, USA
Denver, Colorado, USA
San Diego County, CA, USA
San Diego County, CA, USA
Hänninen et al (2009)
Holstius et al (2012)
Huttunen et al (2012)
Martin et al (2013)
Moore et al (2005)
Rappold et al (2012)
Schranz et al (2010)
Sutherland et al (2005)
Vedal and Dutton (2006)
Viswanathan et al (2006)
Vora et al (2011)

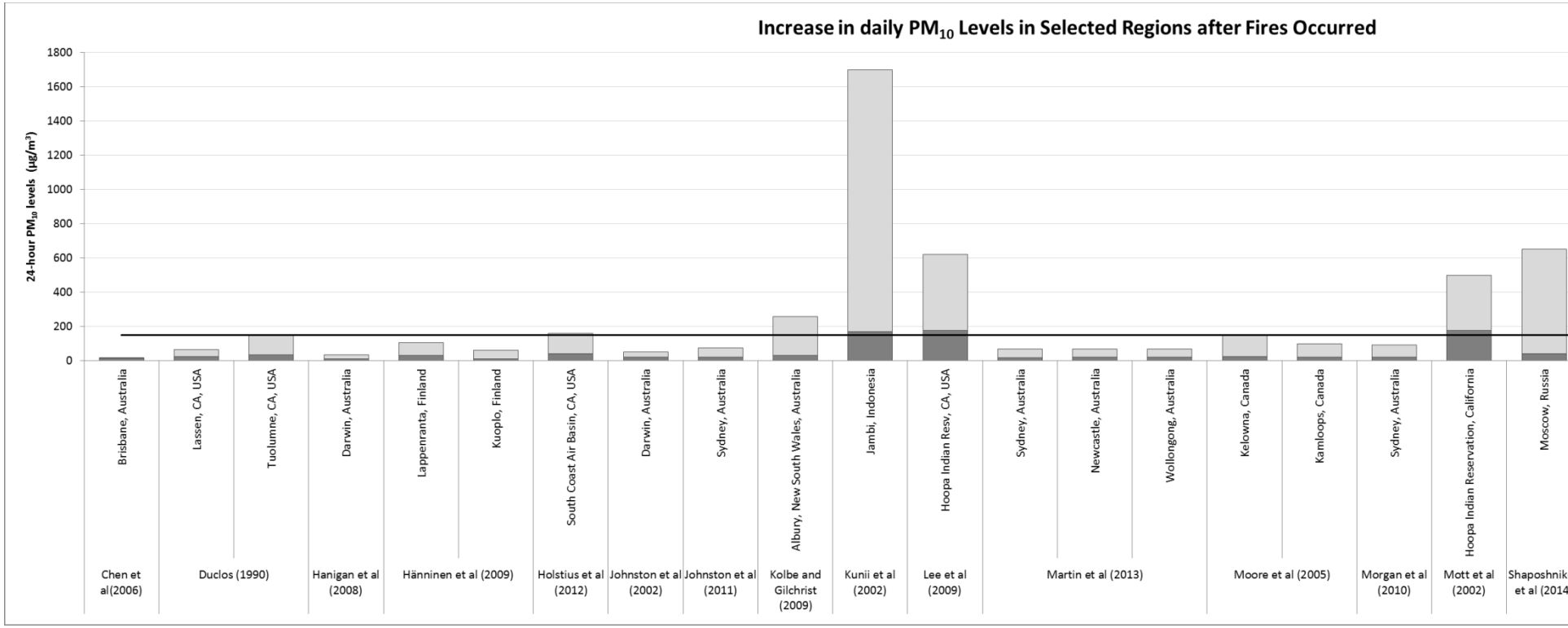


Figure 1. $PM_{2.5}$ (top) and PM_{10} levels (bottom) during wildfire events and non-fire periods

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}

Study	Location	Background population or cohort size	Time of fire	Major health outcome	Exposure metric
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
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 $\geq$ 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

Study	Location	Background population or cohort size	Time of fire	Major health outcome	Exposure metric
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; sub-populations: 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
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}

Study	Location	Background population or cohort size	Time of fire	Major health outcome	Exposure metric
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 $25\mu g/m^3$
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)

Study	**Location**	**Background population or cohort size**	**Time of fire**	**Major health outcome**	**Exposure metric**
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
Sutherland *et al.,* (2005)	Denver, Colorado, U.S.	21 residents who are $\geq$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)

References

Aditama, T.Y. Impact of haze from forest fire to respiratory health: Indonesian experience. Respirology (Carlton, Vic). 5:169-174; 2000

Albertson, K.*, et al.* Climate change and the future occurrence of moorland wildfires in the Peak District of the UK. Clim Res. 45:105-118; 2010

Analitis, A.*, et al.* Forest fires are associated with elevated mortality in a dense urban setting. Occupational and environmental medicine. 69:158-162; 2012

Azevedo, J.M.*, et al.* Long-range ozone transport and its impact on respiratory and cardiovascular health in the north of Portugal. International journal of biometeorology. 55:187-202; 2011

Balling, R.C.*, et al.* Climate Change in Yellowstone-National-Park - Is the Drought-Related Risk of Wildfires Increasing. Climatic Change. 22:35-45; 1992

Brook, R.D.*, et al.* Particulate matter air pollution and cardiovascular disease an update to the scientific statement from the American Heart Association. Circulation. 121:2331-2378; 2010

Cameron, P.A.*, et al.* Black Saturday: the immediate impact of the February 2009 bushfires in Victoria, Australia. The Medical journal of Australia. 191:11-16; 2009

Castro, H.A.*, et al.* [Trend of mortality from respiratory disease in elderly and the forest fires in the state of Rondonia/Brazil - period between 1998 and 2005]. Ciencia & saude coletiva. 14:2083-2090; 2009

Centers for Disease Control and Prevention (CDC). Surveillance of Morbidity During Wildfires -- Central Florida, 1998 MMWR Morbidity and mortality weekly report 48:78-79; 1999

Centers for Disease Control and Prevention (CDC). Wildfire-related deaths--Texas, March 12-20, 2006. MMWR Morbidity and mortality weekly report 2007

Centers for Disease Control and Prevention (CDC). Monitoring health effects of wildfires using the biosense system--San Diego County, California, October 2007. MMWR Morbidity and mortality weekly report; 2008

Chen, L.P.*, et al.* Air particulate pollution due to bushfires and respiratory hospital admissions in Brisbane, Australia. International journal of environmental health research. 16:181-191; 2006

Cleland, H.J.*, et al.* Multidisciplinary team response to a mass burn casualty event: outcomes and implications. Med J Australia. 194:589-593; 2011

Crabbe, H. Risk of respiratory and cardiovascular hospitalisation with exposure to bushfire particulates: new evidence from Darwin, Australia. Environmental geochemistry and health. 34:697-709; 2012

de Mendonca, M.J.*, et al.* Estimation of damage to human health due to forest burning in the Amazon. J Popul Econ. 19:593-610; 2006

Delfino, R.J.*, et al.* The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003. Occupational and environmental medicine. 66:189-197; 2009

Delfino, R.J.*, et al.* Association of asthma symptoms with peak particulate air pollution and effect modification by anti-inflammatory medication use. Environmental health perspectives. 110:A607-A617; 2002

Dennekamp, M.; Abramson, M.J. The effects of bushfire smoke on respiratory health. Respirology (Carlton, Vic). 16:198-209; 2011

Dimopoulou, M.; Giannikos, I. Towards an integrated framework for forest fire control. Eur J Oper Res. 152:476-486; 2004

do Carmo, C.N.*, et al.* [Association between particulate matter from biomass burning and respiratory diseases in the southern region of the Brazilian Amazon]. Revista panamericana de salud publica = Pan American journal of public health. 27:10-16; 2010

Dohrenwend, P.*, et al.* The Impact on Emergency Department Visits for Respiratory Illness During the Southern California Wildfires West J Emerg Med. 14:79-84; 2013
Duclos, P.*, et al.* The 1987 forest fire disaster in California: assessment of emergency room visits. Archives of environmental health. 45:53-58; 1990
Ebisu, K.; Bell, M.L. Airborne PM2.5 Chemical Components and Low Birth Weight in the Northeastern and Mid-Atlantic Regions of the United States. Environmental health perspectives. 120:1746-1752; 2012
Elliott, C.T.*, et al.* Time series analysis of fine particulate matter and asthma reliever dispensations in populations affected by forest fires. Environmental health : a global access science source. 12:11; 2013
Emmanuel, S.C. Impact to lung health of haze from forest fires: the Singapore experience. Respirology (Carlton, Vic). 5:175-182; 2000
Finlay, S.E.*, et al.* Health Impacts of Wildfires. . PLOS Currents Disasters; 2012
Flannigan, M.D.; Vanwagner, C.E. Climate Change and Wildfire in Canada. Can J Forest Res. 21:66-72; 1991
Franck, U.*, et al.* The effect of particle size on cardiovascular disorders - The smaller the worse. Sci Total Environ. 409:4217-4221; 2011
Frankenberg, E.*, et al.* Health consequences of forest fires in Indonesia. Demography. 42:109-129; 2005
Fried, J.S.*, et al.* Predicting the effect of climate change on wildfire behavior and initial attack success. Climatic Change. 87:S251-S264; 2008
Fried, J.S.*, et al.* The impact of climate change on wildfire severity: A regional forecast for northern California. Climatic Change. 64:169-191; 2004
Goncalves, C.*, et al.* Characterisation of PM10 emissions from woodstove combustion of common woods grown in Portugal. Atmos Environ. 44:4474-4480; 2010
Gouveia, N.; Fletcher, T. Time series analysis of air pollution and mortality: effects by cause, age and socioeconomic status. J Epidemiol Commun H. 54:750-755; 2000
Hanigan, I.C.*, et al.* Vegetation fire smoke, indigenous status and cardio-respiratory hospital admissions in Darwin, Australia, 1996-2005: a time-series study. Environmental health : a global access science source. 7:42; 2008
Hänninen, O.O.*, et al.* Population exposure to fine particles and estimated excess mortality in Finland from an East European wildfire episode. Journal of exposure science & environmental epidemiology. 19:414-422; 2009
Henderson, S.B.*, et al.* Three measures of forest fire smoke exposure and their associations with respiratory and cardiovascular health outcomes in a population-based cohort. Environmental health perspectives. 119:1266-1271; 2011
Henderson, S.B.; Johnston, F.H. Measures of forest fire smoke exposure and their associations with respiratory health outcomes. Current opinion in allergy and clinical immunology. 12:221-227; 2012
Holstius, D.M.*, et al.* Birth weight following pregnancy during the 2003 Southern California wildfires. Environmental health perspectives. 120:1340-1345; 2012
Huttunen, K.*, et al.* Low-level exposure to ambient particulate matter is associated with systemic inflammation in ischemic heart disease patients. Environmental research. 116:44-51; 2012
Ignotti, E.*, et al.* Impact on human health of particulate matter emitted from burnings in the Brazilian Amazon region. Revista de saude publica. 44:121-130; 2010
Interagency Working Group on Climate Change and Health. A Human Health Perspective on ClimateChange: A Report Outlining the Research Needs on the Human Health Effects of Climate Change. . in: NIEHS E.H.P.a., ed; 2010

Jacquemin, B.*, et al.* Air pollution and asthma control in the Epidemiological study on the Genetics and Environment of Asthma. J Epidemiol Commun H. 66:796-802; 2012
Jalaludin, B.*, et al.* Acute effects of bushfires on peak expiratory flow rates in children with wheeze: a time series analysis. Australian and New Zealand journal of public health. 24:174-177; 2000
Jayachandran, S. Air Quality and Early-Life Mortality Evidence from Indonesia's Wildfires. J Hum Resour. 44:916-954; 2009
Johnston, F.H.*, et al.* Ambient biomass smoke and cardio-respiratory hospital admissions in Darwin, Australia. Bmc Public Health. 7; 2007
Johnston, F.H.*, et al.* Extreme air pollution events from bushfires and dust storms and their association with mortality in Sydney, Australia 1994-2007. Environmental research. 111:811-816; 2011
Johnston, F.H.*, et al.* Estimated global mortality attributable to smoke from landscape fires. Environmental health perspectives. 120:695-701; 2012
Johnston, F.H.*, et al.* Exposure to bushfire smoke and asthma: an ecological study. The Medical journal of Australia. 176:535-538; 2002
Johnston, F.H.*, et al.* Vegetation fires, particulate air pollution and asthma: A panel study in the Australian monsoon tropics. International journal of environmental health research. 16:391-404; 2006
Keeton, W.S.*, et al.* Climate Variability, Climate Change, and WesternWildfire with Implications for the Urban-Wildland Interface. . in: Howarth R., ed. Advances in the Economics of Environmental Resources: Emerald Group Publishing; 2007
Kleeman, M.J.*, et al.* Enhanced Air Pollution Epidemiology using a Epidemiology using a Source-Oriented Chemical Transport Model. EPA; 2009
Kloog, I.*, et al.* Assessing temporally and spatially resolved PM2.5 exposures for epidemiological studies using satellite aerosol optical depth measurements. Atmos Environ. 45:6267-6275; 2011
Kolbe, A.; Gilchrist, K.L. An extreme bushfire smoke pollution event: health impacts and public health challenges. New South Wales public health bulletin. 20:19-23; 2009
Kunii, O.*, et al.* The 1997 haze disaster in Indonesia: Its air quality and health effects. Archives of environmental health. 57:16-22; 2002
Kunzli, N.*, et al.* Health effects of the 2003 Southern California wildfires on children. American journal of respiratory and critical care medicine. 174:1221-1228; 2006
Lee, H.J.*, et al.* A novel calibration approach of MODIS AOD data to predict PM2.5 concentrations. Atmos Chem Phys. 11:7991-8002; 2011
Lee, T.S.*, et al.* Risk factors associated with clinic visits during the 1999 forest fires near the Hoopa Valley Indian Reservation, California, USA. International journal of environmental health research. 19:315-327; 2009
Lepeule, J.*, et al.* Chronic Exposure to Fine Particles and Mortality: An Extended Follow-up of the Harvard Six Cities Study from 1974 to 2009. Environmental health perspectives. 120:965-970; 2012
Liu, Y.*, et al.* Trends in global wildfire potential in a changing climate. Forest Ecology and Management. 259:685-697; 2010
Lloyd-Jones, D.*, et al.* Heart Disease and Stroke Statistics-2010 Update A Report From the American Heart Association. Circulation. 121:E46-E215; 2010
Malevsky-Malevich, S.P.*, et al.* An assessment of potential change in wildfire activity in the Russian boreal forest zone induced by climate warming during the twenty-first century. Climatic Change. 86:463-474; 2008
Mao, Y.H.*, et al.* Biomass burning contribution to black carbon in the Western United States Mountain Ranges. Atmos Chem Phys. 11:11253-11266; 2011

Martin, K.L.*, et al.* Air pollution from bushfires and their association with hospital admissions in Sydney, Newcastle and Wollongong, Australia 1994-2007. Australian and New Zealand journal of public health. 37:238-243; 2013

Mascarenhas, M.D.*, et al.* [Anthropogenic air pollution and respiratory disease-related emergency room visits in Rio Branco, Brazil--September, 2005]. Jornal brasileiro de pneumologia : publicacao oficial da Sociedade Brasileira de Pneumologia e Tisilogia. 34:42-46; 2008

Matthes, F.C. Climate change 2007. The physical science basis, impacts, adaptation and vulnerability mitigation of climate change. Int Politik. 63:130-132; 2008

Medina-Ramon, M.*, et al.* The effect of ozone and PM10 on hospital admissions for pneumonia and chronic obstructive pulmonary disease: A national multicity study. Am J Epidemiol. 163:579-588; 2006

Mirabelli, M.C.*, et al.* Respiratory symptoms following wildfire smoke exposure: airway size as a susceptibility factor. Epidemiology (Cambridge, Mass). 20:451-459; 2009

Moore, D.*, et al.* Population health effects of air quality changes due to forest fires in British Columbia in 2003: estimates from physician-visit billing data. Canadian journal of public health = Revue canadienne de sante publique. 97:105-108; 2006

Morgan, G.*, et al.* Effects of bushfire smoke on daily mortality and hospital admissions in Sydney, Australia. Epidemiology (Cambridge, Mass). 21:47-55; 2010

Morgenstern, V.*, et al.* Atopic diseases, allergic sensitization, and exposure to traffic-related air pollution in children. American journal of respiratory and critical care medicine. 177:1331-1337; 2008

Mott, J.A.*, et al.* Cardiorespiratory hospitalizations associated with smoke exposure during the 1997, Southeast Asian forest fires. International journal of hygiene and environmental health. 208:75-85; 2005

Mott, J.A.*, et al.* Wildland forest fire smoke: health effects and intervention evaluation, Hoopa, California, 1999. The Western journal of medicine. 176:157-162; 2002

Naeher, L.P.*, et al.* Woodsmoke health effects: a review. Inhalation toxicology. 19:67-106; 2007

Nunes, K.V.R.*, et al.* Circulatory disease mortality rates in the elderly and exposure to PM2.5 generated by biomass burning in the Brazilian Amazon in 2005. Cadernos de saude publica. 29:589-598; 2013

Parry, M.L.*, et al.* Technical Summary. Climate Change 2007: Impacts, Adaptation, and Vulnerability. in: Parry M.L., Canziani O.F., Palutikof J.P., van der Linden P.J., Hanson C.E., eds. Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge, UK: Cambridge University Press; 2007

Peng, R.D.*, et al.* Coarse particulate matter air pollution and hospital admissions for cardiovascular and respiratory diseases among Medicare patients. JAMA : the journal of the American Medical Association. 299:2172-2179; 2008

Phuleria, H.C.*, et al.* Air quality impacts of the October 2003 Southern California wildfires. J Geophys Res-Atmos. 110; 2005

Pio, C.A.*, et al.* Chemical composition of atmospheric aerosols during the 2003 summer intense forest fire period. Atmos Environ. 42:7530-7543; 2008

Po, J.Y.T.*, et al.* Respiratory disease associated with solid biomass fuel exposure in rural women and children: systematic review and meta-analysis. Thorax. 66:232-239; 2011

Pope, C.A.; Dockery, D.W. Health effects of fine particulate air pollution: Lines that connect. J Air Waste Manage. 56:709-742; 2006

Prass, T.S.*, et al.* Amazon Forest Fires Between 2001 and 2006 and Birth Weight in Porto Velho. B Environ Contam Tox. 89:1-7; 2012

Rappold, A.G.*, et al.* Cardio-respiratory outcomes associated with exposure to wildfire smoke are modified by measures of community health. Environmental health : a global access science source. 11:71; 2012
Rappold, A.G.*, et al.* Peat bog wildfire smoke exposure in rural North Carolina is associated with cardiopulmonary emergency department visits assessed through syndromic surveillance. Environmental health perspectives. 119:1415-1420; 2011
Ritz, B.*, et al.* Ambient air pollution and risk of birth defects in southern California. Am J Epidemiol. 155:17-25; 2002
Robinson, M.S.*, et al.* Characterization of PM2.5 collected during broadcast and slash-pile prescribed burns of predominately ponderosa pine forests in northern Arizona. Atmos Environ. 45:2087-2094; 2011
Sarnat, J.A.*, et al.* Fine particle sources and cardiorespiratory morbidity: an application of chemical mass balance and factor analytical source-apportionment methods. Environmental health perspectives. 116:459-466; 2008
Sastry, N. Forest fires, air pollution, and mortality in southeast Asia. Demography. 39:1-23; 2002
Schranz, C.I.*, et al.* The 2007 San Diego Wildfire impact on the Emergency Department of the University of California, San Diego Hospital System. Prehospital and disaster medicine. 25:472-476; 2010
Shaposhnikov, D.*, et al.* Mortality related to air pollution with the moscow heat wave and wildfire of 2010. Epidemiology (Cambridge, Mass). 25:359-364; 2014
Shusterman, D.*, et al.* Immediate health effects of an urban wildfire. The Western journal of medicine. 158:133-138; 1993
Singh, H.B.*, et al.* Pollution influences on atmospheric composition and chemistry at high northern latitudes: Boreal and California forest fire emissions. Atmos Environ. 44:4553-4564; 2010
Smith, K.R.*, et al.* Indoor air pollution from household use of solid fuels. Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors. 2:1435-1493; 2004
Smith, M.A.*, et al.* Asthma presentations to emergency departments in western Sydney during the January 1994 Bushfires. International journal of epidemiology. 25:1227-1236; 1996
Spracklen, D.V.*, et al.* Impacts of climate change from 2000 to 2050 on wildfire activity and carbonaceous aerosol concentrations in the western United States. J Geophys Res-Atmos. 114; 2009
Sutherland, E.R.*, et al.* Wildfire smoke and respiratory symptoms in patients with chronic obstructive pulmonary disease. The Journal of allergy and clinical immunology. 115:420-422; 2005
Tan, W.C.*, et al.* The human bone marrow response to acute air pollution caused by forest fires. American journal of respiratory and critical care medicine. 161:1213-1217; 2000
Tham, R.*, et al.* The impact of smoke on respiratory hospital outcomes during the 2002-2003 bushfire season, Victoria, Australia. Respirology (Carlton, Vic). 14:69-75; 2009
The Heart Foundation. Data and Statistics. Information for professionals; 2011
Thelen, B.*, et al.* Modeling acute respiratory illness during the 2007 San Diego wildland fires using a coupled emissions-transport system and generalized additive modeling. Environ Health-Glob. 12; 2013
U.S. Forest Service. Today's Challenges and Opportunities: climate change Briefing paper; 2009
U.S. Forest Service. Wildland Fire Smoke 2010
Valavanidis, A.*, et al.* Airborne Particulate Matter and Human Health: Toxicological Assessment and Importance of Size and Composition of Particles for Oxidative Damage and Carcinogenic Mechanisms. J Environ Sci Heal C. 26:339-362; 2008
van den Hooven, E.H.*, et al.* Air Pollution, Blood Pressure, and the Risk of Hypertensive Complications During Pregnancy The Generation R Study. Hypertension. 57:406-U138; 2011

Vedal, S.; Dutton, S.J. Wildfire air pollution and daily mortality in a large urban area. Environmental research. 102:29-35; 2006
Versura, P.*, et al.* Eye discomfort and air pollution. Ophthalmologica. 213:103-109; 1999
Viswanathan, S.*, et al.* An analysis of effects of San Diego wildfire on ambient air quality. Journal of the Air & Waste Management Association (1995). 56:56-67; 2006
Vora, C.*, et al.* 2007 San Diego Wildfires and Asthmatics. J Asthma. 48:75-78; 2011
Ward, D.E. Factors influencing the emissions of gases and particulate matter from biomass burning. Fire in the Tropical Biota: Springer Berlin Heidelberg; 1990
Watson, J.T.*, et al.* Epidemics after natural disasters. Emerging infectious diseases. 13:1-5; 2007
Wegesser, T.C.*, et al.* California wildfires of 2008: coarse and fine particulate matter toxicity. Environmental health perspectives. 117:893-897; 2009
Westerling, A.L.; Bryant, B.P. Climate change and wildfire in California. Climatic Change. 87:S231-S249; 2008
Westerling, A.L.*, et al.* Warming and earlier spring increase western US forest wildfire activity. Science. 313:940-943; 2006
Wiwatanadate, P.; Liwsrisakun, C. Acute effects of air pollution on peak expiratory flow rates and symptoms among asthmatic patients in Chiang Mai, Thailand. International journal of hygiene and environmental health. 214:251-257; 2011
Zanobetti, A.*, et al.* Fine particulate air pollution and its components in association with cause-specific emergency admissions. Environ Health-Glob. 8; 2009

Supplementary material

Appendix A

Table A.1. Keywords for systematic search [Supplement]

Pubmed search	
Pubmed 1	Forest AND fire AND health
Pubmed 2	(Forest AND fire AND hospital*) OR (wildfire AND hospital*)
Pubmed 3	Wildfire* AND (respir* OR pulmon* OR asthma* OR cardiac OR cardiovascular)
Pubmed 4	Bushfire AND health
Pubmed 5	Bushfire AND (respir* OR pulmon* OR hospital* OR asthma* OR cardiac OR cardiovascular)
Pubmed 6	Mortality AND ((Forest AND fire) OR wildfire OR bushfire)

Scopus search	
Scopus 1	(wildfire AND smoke) AND (health OR respir* OR pulmon* OR asthm* OR hospital* OR mortality OR cardiac OR cardiovascular) AND NOT (stove OR indoor OR heat* OR cook*)
Scopus 2	(forest AND fire AND smoke) AND (health OR respir* OR pulmon* OR asthm* OR hospital* OR mortality OR cardiac OR cardiovascular) AND NOT (stove OR indoor OR heat* OR cook*)
Scopus 3	(bushfire OR "peat bog fire" OR "urban fire" OR "landscape fire" OR grassfire OR “vegetation fire”) AND (health OR respir* OR pulmon* OR asthm* OR hospital* OR mortality OR cardiac OR cardiovascular)

	AND NOT (stove OR indoor OR heat* OR cook*)

Note: * indicates a wild character (e.g., hospital* can represent hospitals or hospitalizations)

Figure A.1. Flowchart of systematic search

Note: The search method was developed with consideration of PRISMA (Preferred Reported Items for Systematic Reviews and Meta-Analyses) guidelines (Liberati *et al.*, 2009).

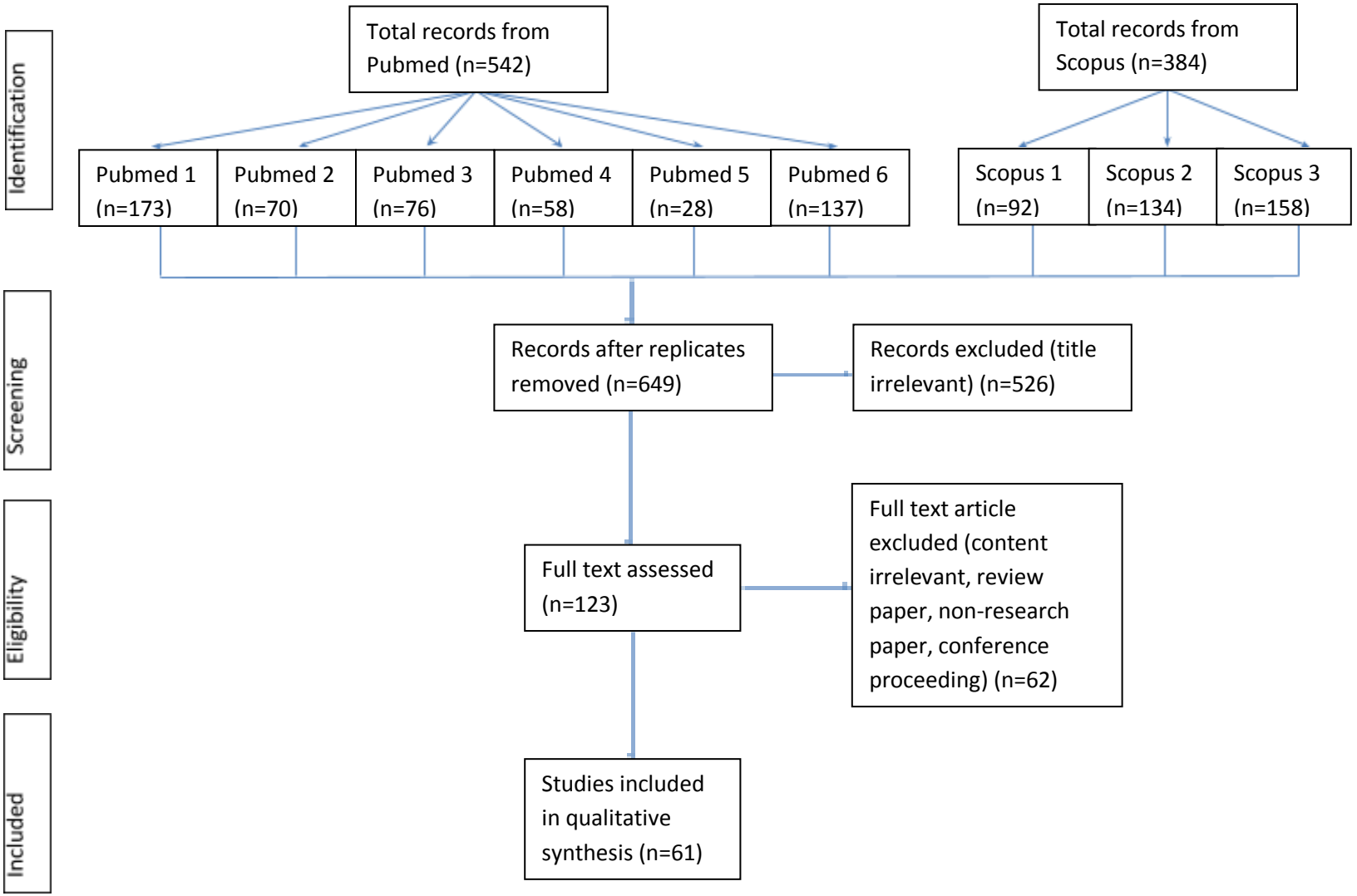


Table A.2: Summary of health outcomes considered in relation to wildfire smoke

Study	**Health outcomes**	**Data source**	**Timeframe of health data**
Aditama (2000)	Respiratory diseases and responses such as ARI, bronchial asthma	Multiple records: Personal health data from pulmonologists from different provinces/ districts; self-reported surveys; respiratory incidence data from Indonesian Central Bureau of Statistics	10 months (case period: Sep. 1997-June 1998, control period: 1995-1996)
Analitis *et al.*, (2012)	Daily total (non-accidental), CVD, and respiratory mortality	Hellenic Statistical Authority	11 years
Azevedo *et al.*, (2011)	Hospital admissions for hypertensive disease (ICD codes 401–405); ischemic heart disease (410–414); other cardiac diseases, including heart failure (426–428); COPD and allied conditions, including bronchitis and asthma (490–496); pneumoconiosis and other lung diseases due to external agents (500–507)	Sum of admissions from 3 hospitals in exposed areas from Urgency Services	Unspecified months within 2005
Caamano-Isorna et al., (2011)	Medicine usage for obstructive airway diseases consumption	The primary health care pharmaceutical billing database; the Individual Health Card database; Ministry of Health	Two 12-month period before and after Aug 2006
Cameron *et al.*, (2009)	Bushfire-related injuries	Records from the Alfred Trauma Registry and trauma center	72 hours after fire
do Carmo *et al.*, (2010)	Daily primary care visits for respiratory complaints among children and elderly in 14 health units	Hospital records	2 years (2004-2005)
Castro *et al.*, (2009)	Respiratory and COPD mortality	Death certificates from Mortality Information System	8 years (1998-2005)
Center for Disease Control and Prevention (CDC) (1999)	Respiratory and cardiovascular ER visits	Surveillance monitoring data on 8 hospitals	In total 72 days: 36 days during fire (June 1-July, 1998); 36 days in the previous year with no fire (June 1-July 6, 1997)
Centers for Disease Control and Prevention (CDC) (2007)	Accidental and non-accidental mortality resulting from (1) direct contact with fire smoke, or (2) reasons indirectly related to the fires, such as poor visibility.	Texas Dept. of State Health Services	9 days (March 12-20, 2006)

Study	Health outcomes	Data source	Timeframe of health data
Centers for Disease Control and Prevention (CDC) (2008)	Emergency department (ED) visits for respiratory diseases	CDC BioSence System	25 days (20 days before fire; 5 days during fire)
Chen *et al.*, (2006)	Respiratory hospital admissions	Queensland Dept. of Health	3.5 years (July 1, 1997-Dec. 31, 2000
Cleland *et al.*, (2011)	Injuries due to bushfires	Records from Ambulance Victoria	Length of fire (a few days)
Crabbe (2012)	ER visits for respiratory and CVD diseases	Royal Darwin Hospital; Northern Territory Government's Dept. of Health and Community Services	6 years (1993-1998)
Delfino *et al.*, (2009)	Cardiorespiratory hospital admissions, including asthma, acute bronchitis, COPD, ischaemic heart disease, CHF, cardiac dysrhythmia, cerebrovascular disease and stroke	Zip-code level information from California State Office of Statewide Health Planning and Development	1.5 months (Oct. 1-Nov. 15, 2003)
Dohrenwend et al, (2013)	Respiratory ER visits	Kaiser Permanente electronic database	Oct 1 – Nov 6, 2007
Duclos *et al.*, (1990)	Hospital ER visits	ER records; individual-level information about cause and symptoms from ER log and additional interviews	In total 47 days t: 17 days during and after fire (Aug. 30-Sep. 17, 1987), two 15-day reference periods before fire (Sep. 1-15, 1986; Aug. 15-29, 1987)
Elliott *et al.*, (2013)	Pharmaceutical dispensations for salbutamol	BC PharmaNet database	8 years (2003-2010)
Emmanuel (2000)	Hospital admissions for respiratory diseases, all-cause mortality	Hospital network system and ER records for hospital admissions; database from Registration of Births and Deaths Act for mortality	1 year (Jan.-Dec. 1997)
Frankenberg *et al.*, (2005)	Respiratory morbidities; degree to which subjects were able to carry out strenuous tasks; overall health	Individual health information from population-based longitudinal survey (Indonesia Family Life Survey)	Survey conducted during 5 months (Aug.-Dec. 1997)

Study	Health outcomes	Data source	Timeframe of health data
Hanigan *et al.*, (2008)	Hospital admissions for CVD	De-identified individual admissions records in Royal Darwin Hospital were obtained from Northern Territory Dept. of Health and Community Services	Ten 8-months periods (Apr. –Nov. in each year during 1996-2005)
Hänninen *et al.*, (2009)	Daily mortality	Statistics Finland	14 days (Aug. 26-Sep. 8, 2002)
Henderson *et al.*, (2011)	Doctor and hospital visits for respiratory and CVD illnesses	Billed physician visits from Medical Services Plan of BC	92 days (July 1 to Sep. 30, 2003)
Holstius *et al.*, (2012)	Birth weight	California's Center for Health Statistics at the California Dept. of Health Services	5 years (2001-2005)
Huttunen *et al.*, (2012)	Blood concentration of inflammatory markers: interleukin (IL)-1b, IL-6, IL-8, IL-12, interferon (IFN)g, C-reactive protein (CRP), fibrinogen, myeloperoxidase and white blood cell count	Blood samples of subjects	24 weeks (Nov. 2005-May 2006)
Ignotti *et al.*, (2010)	Hospitalization for respiratory diseases	Ministry of Health	2 years (2004-2005)
Jalaludin *et al.*, (2000)	PEFR in children with wheezing history	Self-reported diary; daily measured night-time PEFR values	30 days (Jan. 1- 31, 1994)
Jayachandran (2009)	Mortality for children under 3, infant and fetus	2000 Census of Population, with month of birth	1.5 years (Dec. 1996-May 1998)
Johnston *et al.*, (2002)	Asthma ED visits	ED of Royal Darwin Hospital	7 months (Apr. 1- Oct. 31, 2000)
Johnston *et al.*, (2006)	Asthmatic symptoms	Self-reported surveys	7 months (Apr. 7-Nov. 7, 2004)
Johnston *et al.*, (2007)	Respiratory and CVD Hospital admission by cause	De-identified individual admissions records in Royal Darwin Hospital	Three 8-month periods (Apr.-Nov. in 2000, 2004 and 2005)
Johnston *et al.*, (2011)	Non-accidental, CVD, and respiratory mortality	Australian Bureau of Statistics	13.5 years (Jan. 1994-June 2007)

Study	Health outcomes	Data source	Timeframe of health data
Johnston *et al.*, (2012)	All-cause mortality	Modelled for 1997-2006 based on the subregions defined in WHO Global Health Observatory published 2011	10 years (1997-2006)
Kolbe and Gilchrist (2009)	Respiratory or depression symptoms, requested medical treatment or not	Telephone surveys	Survey conducted in late Feb. and early March, 2003
Kunii *et al.*, (2002)	Respiratory symptoms	Individual health data from interviews; lung function tests and respiratory health examinations on ¼ of subjects	Survey during 9 days of major fire events (Sep. 29-Oct. 7, 1997)
Kunzli *et al.*, (2006)	Health problems including eye irritation, coughing, wheezing, asthma, bronchitis, and nose and throat-related symptoms	Health data from Children's Health Study; surveys for individual health outcomes	Survey conducted over 2 months (Nov.-Dec., 2003)
Lee *et al.*, (2009)	Doctor visits for asthma, circulatory-only illness, coronary artery disease, headache, diabetes, and respiratory-only diseases	Hospitalization data from database of the only clinic serving this area	12 weeks (Aug. 17-Nov. 4, 1999), compared with same 12-week period in 1998
Martin et al (2013)	All non-trauma admissions, including cardiovascular, asthma, COPD, and other respiratory admissions	Department of Health in NSW	14 years (1994-2007)
Mascarenhas *et al.*, (2008)	ER visits for respiratory disease including diagnosis of asthma, bronchitis, COPD, upper respiratory tract infection or pneumonia, or medical record of coughing or breathlessness in the absence of other diagnosis	Not specified	30 days (Sep. 1–30, 2005)
de Mendonca *et al.*, (2006)	Respiratory illness	Municipal morbidity data	5 years (1996-2000)
Mirabelli *et al.*, (2009)	Respiratory and eye symptoms for students with different quartiles of airway size (ratio of maximum mid-expiratory flow (MMEF) and forced vital capacity (FVC) as indicator)	Airway sizes measured; respiratory and eye symptoms assessed from questionnaire	Survey 5-10 months before fire and on average 65 days after last day of fire, Nov. 3, 2003
Moore *et al.*, (2006)	Weekly rate of doctor visits for respiratory, CVD, and mental health illnesses	Billed physician visits from Medical Services Plan of BC	7 months in each year during 11-year period 1993-2003
Morgan *et al.*, (2010)	Hospital admissions and mortality	Mortality data from Australian Bureau of Statistics, hospital admissions data from New South Wales Dept. of Health	8.5 years (Jan. 1994- June 2002)

Study	Health outcomes	Data source	Timeframe of health data
Mott *et al.*, (2002)	Clinic visits for respiratory illness and self-reported symptoms (ICD codes 460-519)	Clinical visits records on respiratory diseases and survey results on respiratory symptoms before, during and after fire	Over 11 weeks (surveyed health symptoms before fire, during fire: Aug. 23–Oct. 26, 1999; after fire: Oct. 27-Nov. 15, 1999)
Mott *et al.*, (2005)	Hospital admissions for cardio-respiratory-related symptoms and asthma	Hospital record database	4 years (1995-1998)
Nunes et al, (2013)	Mortality due to circulatory diseases, including CVD	Brazilian Health Informatics Department	1 year (2005)
Prass *et al.*, (2012)	Birth weight	Hospital birth records	6 years (2001-2006)
Rappold *et al.*, (2011)	ED visits for CVD and respiratory diseases	Surveillance program NC Disease Event Tracking and Epidemiologic Collection Tool	2 weeks (June 1-14, 2008)
Rappold *et al.*, (2012)	ED visits for CHF and asthma	NC Disease Event Tracking and Epidemiologic Collection Tool	6 weeks (June 1- July 14, 2008)
Sastry (2002)	All-cause, non-traumatic, CVD, respiratory, and other mortality	Malaysian Vital Statistics records with individual-level data	4 years (1994-1997)
Schranz *et al.*, (2010)	Respiratory ED visits and complaints	UCSD hospital ED computerized records, including demographic information	12 days (before fire: Oct. 14-19; after fire: Oct. 21-26, 2007)
Shaposhnikov *et al*. (2014)	Mortality	Russian State Statistics	5 years (2006-2010)
Shusterman *et al.*, (1993)	Fire-related ED visits for "all trauma, burns, chest pain, respiratory disorders, smoke inhalation, conjunctivitis, corneal abrasions, mental health problems, and problems placing chronically ill patients"	ED logs and medical records	6 days (Oct. 21-26, 1991)
Smith *et al.*, (1996)	ED visits for asthma and related respiratory diagnostics	ED records in 7 hospitals in the study area	Two 6-week periods (study period: Dec. 17, 1993-Jan. 31, 1994; control period: Dec. 17, 1992-Jan. 31, 1993)
Sutherland *et al.*, (2005)	Respiratory symptoms	Daily phone interviews of cohort subjects	Survey conducted during 22 days (June 8- 29, 2002) during fire

Study	Health outcomes	Data source	Timeframe of health data
Tan *et al.,* (2000)	Bone marrow health; count of peripheral white blood cells and lung function tests	Blood samples collected	6 months (Jun-Dec. 1997; blood tests conducted 5 times during fire and 3 times after fire during the 6-month period)
Tham *et al.,* (2009)	ED and hospital admissions for respiratory diseases	Victorian Department of Human Services	7 months (Oct. 2002-Apr. 2003)
Thelen et al (2013)	ED visits for respiratory symptoms	"Syndromic surveillance data-base"	4 months (Aug. to Nov. 2007)
Vedal and Dutton (2006)	All-cause (non-accidental) and cardio- respiratory daily mortality	Colorado Health Information Dataset	2 years (2001-2002)
Viswanathan *et al.,* (2006)	Number of doctor visits for asthma, bronchitis, or emphysema; other respiratory conditions with no fever; eye irritation; smoke inhalation; chest pain or cardiac arrests; and diarrhea	Surveillance records from San Diego Co. Health and Human Services Agency, Public Health Services	3 weeks (1 week before fire, 2 weeks after fire)
Vora *et al.,* (2011)	Pulmonary function and rescue medication use	Lung function tests	Three 5-day periods (before fire: Oct. 14-18; during fire: Oct. 22-26; after fire: Nov. 13-17)
Wiwatanadate and Liwsrisakun (2011)	PEFR and asthma symptoms	Self-reported surveys; PEFR measured daily with Mini-Wright peak flow meter.	10.5 months (Aug. 15, 2005-June 30, 2006)

Table A.3: Summary of exposure methods for wildfire smoke

Study	Air pollutant/exposure	Data source	Period of exposure assessment
Aditama (2000)	Fire period defined a priori; CO, SO_2, PM_{10} (daily), TSP, NOx, O_3, and organic compounds (pollutants considered vary by province) during fire period compared with same periods in previous 2 years. Measurement frequency of pollutant other than PM_{10} not fully specified	Air monitors	~ 3 months (Sep. to Nov., 1997)
Analitis *et al.*, (2012)	Indicator of fires as area burned: (1) small (10,000-1 million m^2 burned), (2) medium (>1 million to 30 million m^2 burned), and (3) large (>30 million m^2 burned); daily black smoke (black particles with diameter <4μm) index measured	Date and area burned information from Fire Service of Greece; smoke data from 5 monitoring sites of Ministry of Environment, Energy, and Climate Change	11 years
Azevedo *et al.*, (2011)	Fire period defined a priori; hourly O_3 during fire period	Hourly data from monitoring stations near hospitals; fire trajectory modelled from Hybrid Single-Particle Lagrangian Integrated Trajectory (HYSPLIT) model	3 months (Jun-Aug, 2005)
Caamano-Isorna et al., (2011)	Fire period defined a priori; Exposure classified into three categories based on number of fires: no exposure (0-3 fires), medium exposure (4-10 fires), high exposure (11-58 fires)	Ministry of the Environment	1 month (Aug 2006)
Cameron *et al.*, (2009)	Bushfires (air pollutants not specified)	Not specified	Length of fire (a few days)
do Carmo *et al.*, (2010)	Fire period defined a priori; daily $PM_{2.5}$ during fire period	Estimated from mathematical models developed by National Institute for Space Research (INPE)	2 years (2004-2005)
Castro *et al.*, (2009)	Number of fire "hotspots" each year	Hotspots identified with satellite remote sensing imagery from Advanced Very High Resolution Radiometer (AVHRR), Moderate Imaging Spectroradiometer (MODIS-Aqua, MODIS-Terra), and Geostationary Operational Environmental Satellites (GOES)	8 years (1998-2005)
Center for Disease Control and Prevention (CDC) (1999)	Fire period (defined a priori) compared to non-fire periods	Not specified	About 2 months (Jun- Jul 1998)
Centers for Disease Control	Wildfire smoke in the study period	Not specified	9 days (March 12-20, 2006)

Study	Air pollutant/exposure	Data source	Period of exposure assessment
and Prevention (CDC) (2007)			
Centers for Disease Control and Prevention (CDC) (2008)	Fire period (defined a priori) compared to non-fire periods	Not specified	5 days
Chen *et al.*, (2006)	Fire period defined a priori; daily PM_{10} during bushfire compared with non-bushfire periods	Air monitors of Queensland Environmental Protection Agency.	3.5 years (July 1, 1997-Dec. 31, 2000)
Cleland *et al.*, (2011)	Bushifires (air pollutants not specified)	Not specified	Length of fire (a few days)
Crabbe (2012)	Fire season defined a priori during a 6-year study period; daily PM_{10} (calculated by adding fine PM (FPM, <2µm) and coarse PM (2-10 µm) concentrations) and black carbon for 6-year study period (covering fire periods in each year)	Air monitors for PM and black carbon provided by Charles Darwin University; Meteorological information obtained from Bureau of Meteorology	~ 6 years (1993-1998)
Delfino *et al.*, (2009)	Fire period defined a priori; 2-day moving average $PM_{2.5}$ during fire period compared with pre- and post- fire periods	Estimated from MODIS satellite images at 250m resolution	1.5 months (Oct. 1-Nov. 15, 2003)
Dohrenwend et al, (2013)	Fire period defined a priori; ER visits compared pre- and during fire period; AQI also obtained	Fire period defined by the State of California; AQI obtained from Airnow	Oct 17, Oct 21- Nov 6, and Nov 10, 2007
Duclos *et al.*, (1990)	Fire period defined a priori; PM_{10} and TSP during fire period compared with pre- and post-fire periods	Air monitors	~ 1 month
Elliott *et al.*, (2013)	LHA "fire-affected" when daily aggregate "fire radioactive" data (proportional to aerosol emissions) in MODIS data >95th percentile of that in all LHAs in $\geq$ 3 of 9 fire seasons; daily $PM_{2.5}$ and PM_{10} used in analysis comparing affected and not-affected LHAs	Air monitors; for LHAs with limited $PM_{2.5}$ data, $PM_{2.5}$ estimated from PM_{10} data; exposed populations determined using satellite image of fires.	Fire seasons during 8-year period (Apr. 1 to Sep. 30 of each year 2003-2010)
Emmanuel (2000)	Fire period defined a priori; daily PM_{10}, SO2, NO_2, O_3, CO, and total hydrocarbon during fire period	Telemetric monitors at 15 stations of the Ministry of the Environment	~ 4 years (1994-1997)
Frankenberg *et al.*, (2005)	Fire period defined a priori; daily TOMS aerosol index during fire period compared with that period in previous years (1996-2002)	Estimates from satellites of NASA Total Ozone Monitoring System (TOMS); IFLS Survey provided locations of subjects matched with satellite imagery of exposure	6-month period of major-fire period (late Sep. 1997 to March 1998) compared with other periods during

Study	Air pollutant/exposure	Data source	Period of exposure assessment
			June 1996-June 2002 (no data available 1993-1996)
Hanigan *et al.*, (2008)	Fire season defined a priori; daily PM_{10} during study period	Estimated using a predictive model based on visibility data	Ten 8-months periods (Apr.–Nov. in each year 1996-2005)
Hänninen *et al.*, (2009)	Fire period defined a priori; daily $PM_{2.5}$ and PM_{10}; 2-week average $PM_{2.5}$ and PM_{10} during fire period compared with monthly average of pollutants in same period a year later	Hourly measurements from 8 monitors; for locations without $PM_{2.5}$ monitors, $PM_{2.5}$ estimated from PM_{10} data; backward trajectory analysis to estimate origin of aerosols	14 days (Aug. 26-Sep. 8, 2002)
Henderson *et al.*, (2011)	Fire period defined a priori; daily PM_{10} during fire period; binary variable of exposed areas detected by SMOKE satellite imagery	6 regulatory tapered element oscillating microbalance (TEOM) air quality monitors; CALPUFF dispersion modelling; SMOKE satellite imagery for plume visibility. Risk estimated for each exposure method separately	92 days (July 1 to Sep. 30, 2003)
Holstius *et al.*, (2012)	Fire period defined a priori; women who gave birth 2001-2005 categorized as exposed if pregnancy and fire periods overlapped, unexposed otherwise	Not specified	21 days (Oct. 21-Nov. 10, 2003)
Huttunen *et al.*, (2012)	Fire period defined a priori; daily $PM_{2.5}$ and PM_{10} during fire compared with pre-fire periods	PM_{10} and $PM_{2.5}$ air monitors; personal exposure to $PM_{2.5}$ from photometers 24 hour before each clinical visit	12 days (Apr. 25-May 6, 2006)
Ignotti *et al.*, (2010)	% of hours/year with $PM_{2.5}$ >80µg/m^3 as indicator of exposure (indicator named % of annual hours (AH %))	Hourly $PM_{2.5}$ measurements from National Space Research Institute.	2 years (2004-2005)
Jalaludin *et al.*, (2000)	Fire period defined a priori; daily PM_{10} and NO_2, and daily max O_3 during fire compared with 1 week and 2 weeks after fire	Air monitors	1 year (1994)
Jayachandran (2009)	Fire period defined a priori; daily aerosol index during month of birth and 3 months before and after birth	Estimated from TOMS satellite	1.5 years (Dec. 1996-May 1998)
Johnston *et al.*, (2002)	Fire season defined a priori; daily PM_{10} during fire season	2 sites in suburban Darwin. One site used a tapered element oscillating mass balance; the other used a Microvol aerosol sampler.	7 months (Apr. 1- Oct. 31, 2000)

Study	Air pollutant/exposure	Data source	Period of exposure assessment
Johnston *et al.*, (2006)	Fire season defined a priori; daily $PM_{2.5}$ and PM_{10} during fire season.	Air monitors	7 months (Apr. 7-Nov. 7, 2004)
Johnston *et al.*, (2007)	Fire season defined a priori; daily PM_{10} during fire season.	Measured using a Tapered Element Oscillating Microbalance in 2000, and using Rupprecht and Patashnick Partisol plus model 2025 air sampler in 2004 and 2005	Three 8-month periods (Apr.-Nov. in 2000, 2004 and 2005)
Johnston *et al.*, (2011)	Fire period defined a priori; daily PM_{10} and 1-h max O_3 during events compared with non-fire periods	Air monitor data from New South Wales Dept. of Environment, Climate Change and Water	13.5 years (Jan. 1994-June 2007)
Johnston *et al.*, (2012)	Annual $PM_{2.5}$ modelled for populated continents for the study period	Estimated by combining outputs from chemical transport model and satellite images	10 years (1997-2006)
Kolbe and Gilchrist (2009)	Fire period defined a priori; daily PM_{10} for 2-month period covering fire period	Air monitors	2 months (Jan. 1 to Feb. 28, 2003)
Kunii *et al.*, (2002)	Fire period defined a priori; daily CO, CO_2, SO_2, NO_2, O_3, PM_{10}, inorganic ions, and PAHs in affected cities during fire period compared with those in unaffected cities. Health risks and pollutant levels compared for fire and non-fire periods	Air monitors of various technologies: PM_{10} at 3 sites; size distribution of particulates, CO and CO_2 indoors and outdoors at 8 sites; SO_2, NO_2, O_3	3 days (Oct. 1 and Oct. 3-4, 1997)
Kunzli *et al.*, (2006)	Fire period defined a priori; exposure assessed: 1) smell of fire smoke during the 1^{st}-5^{th} days of fire and after 6^{th} day of fire, and 2) daily PM_{10} during fire period	Community exposure from monitors during 5-day highest fire activity; individual indoor smoke exposure from surveys on number of days with smell of fire smoke	Survey-based
Lee *et al.*, (2009)	Fire period defined a priori; daily PM_{10} during fire period compared with same period in previous year	Hourly data from tapered element oscillating microbalance ambient particulate monitor of Hoopa's Tribal Environmental Protection Agency	12 weeks (Aug. 17-Nov. 4, 1999)
Martin et al (2013)	Smoke event days defined as: days with daily city-wide average $PM_{2.5}$ and PM_{10} exceeding the 99th percentile of the daily distribution for the study period. Smoke event days were compared with non-smoke event days.	New South Wales Office of Environment and Heritage	PM_{10}: 14 years (1994-2007); $PM_{2.5}$: 12 years (1996-2007)
Mascarenhas *et al.*, (2008)	Fire period defined a priori; daily $PM_{2.5}$ during fire period compared with the same period in previous year	Monitoring station at Federal University of Acre	30 days (Sep. 1–30, 2005)
de Mendonca *et al.*, (2006)	Binary indicator of fire based on area burned estimated from "hot pixels" representing outbreaks of fires from satellite images	Satellite images from Wood Hole Research Center	5 years (1996-2000)

Study	Air pollutant/exposure	Data source	Period of exposure assessment
Mirabelli *et al.*, (2009)	Fire period defined a priori; number of days subjects smelled smoke; daily PM_{10} during fire period (for statistical adjustment)	Smoke exposure self-reported from questionnaire; PM_{10} monitors	Survey based (respondents' feeling about smoke during fire)
Moore *et al.*, (2006)	Fire period defined a priori; daily PM_{10} and $PM_{2.5}$ during fire periods compared with aggregates of previous 10 years	Air monitors of BC Ministry of Water, Land and Air Protection	10 weeks (July 13-Sep. 21, 2003)
Morgan *et al.*, (2010)	Fire season defined a priori during 8.5-year study period; daily PM_{10} for fire seasons during study period	Air monitors of New South Wales Dept. of Environment and Climate Change	8.5 years (Jan. 1994- June 2002)
Mott *et al.*, (2002)	Fire period defined a priori; weekly average PM_{10} during fire and 1 week before fire compared with same period of previous year	Not specified	12 weeks (Aug. 14 to Nov. 4, 1999)
Mott *et al.*, (2005)	Fire period defined a priori; daily PM_{10} during fire period compared with 2 previous years	Air monitors	3 months of fire period (Aug.-Oct., 1997) compared with 1 month of post-fire period (Nov.-Dec., 1997)
Nunes et al, (2013)	Exposure defined as annual % of hours with $PM_{2.5}$ greater than 25µg/m^3	Center for Weather Forecasts and Climate Studies of the National Institute for Space Research	1 year (2005)
Prass *et al.*, (2012)	Number of fires	Count of fires from heat spots in satellite images	6 years (2000-2005)
Rappold *et al.*, (2011)	High exposure window determined by AOD	Half-hour, 4x4km resolution gridded maps created from satellite data	2 weeks (June 1-14, 2008)
Rappold *et al.*, (2012)	Fire period defined a priori; daily $PM_{2.5}$ during fire period	Estimates from NOAA Smoke Forecasting System based on satellite models	6 weeks (June 1-July 14, 2008)
Sastry (2002)	Fire period defined a priori; daily PM_{10} (Kaula Lumpur only) during fire period compared to previous year; visibility as an "alternative measure of air quality"	PM_{10}: Malaysian Meteorology Bureau; climate data and visibility: Global Weather Station Database assembled by National Climatic Data Center at US NOAA	2 years (1996-1997) for PM_{10}; 4 years (1994-1997) for visibility
Schranz *et al.*, (2010)	Fire period defined a priori; daily $PM_{2.5}$ during fire period compared with a week before and 3 weeks after fire	Hourly data from $PM_{2.5}$ sensors of San Diego Air Pollution Control District.	41 days (Oct. 14-Nov. 23, 2007)
Shaposhnikov *et al*. (2014)	Fire period defined a priori; effect of PM_{10} estimated using time-series model with interaction term with temperature	PM_{10} and temperature data from monitors of State environmental Protection institution Mosecomonitoring	5 years (2006-2010)

Study	Air pollutant/exposure	Data source	Period of exposure assessment
Shusterman *et al.*, (1993)	Grass fire (air pollutants not specified)	Not specified	Duration of fire (not specified)
Smith *et al.*, (1996)	Fire period defined a priori; PM_{10}, O_3, NO_2 during 6-week fire period compared with same period in previous year. Also statistical model of daily maximum pollution levels.	Air monitors of New South Wales Environmental Protection Agency	Two 6-week periods (study period: Dec. 17, 1993-Jan. 31, 1994; control period: Dec. 17, 1992-Jan. 31, 1993)
Sutherland *et al.*, (2005)	Fire period defined a priori; daily $PM_{2.5}$, PM_{10}, and CO during fire period; levels on spike days during fire period (June 9 and 18) compared with non-spike days	Air quality monitor	22 days (June 8- 29, 2002, with June 9 and 18 as spike days and the rest as non-spike days)
Tan *et al.*, (2000)	Fire period defined a priori; daily SO_2, PM_{10}, NO_2, O_3; CO during fire period compared with days after fires were cleared	15 air monitors of the Ministry of the Environment	1 year (Jan. 1997-Jan. 1998)
Tham *et al.*, (2009)	Fire season defined a priori; daily PM_{10} (derived from hourly concentrations), airborne particle index and daily 4-h max O_3	Hourly PM_{10} data from the Alphington air quality monitoring station; O_3 using chemiluminescence.	7 months (Oct. 2002-Apr. 2003)
Thelen et al (2013)	Fire season defined a priori; daily $PM_{2.5}$ and PM_{10} during fire compared with non-fire condition	Empirical PM emissions model and atmospheric advection and dispersion model	4 months (Aug. – Nov. 2007)
Vedal and Dutton (2006)	Fire period defined a priori; daily $PM_{2.5}$ and PM_{10} during fire compared with a year before fire event	Air monitors of Colorado Dept. of Public Health and the Environment	1 month in each of the 2 years (June 2002 and June 2001 (for comparison)
Viswanathan *et al.*, (2006)	Fire period defined a priori; daily $PM_{2.5}$,PM_{10}, O_3, NO_2, SO_2 and CO measured for three periods: (1) 4 weeks before fire; (2) 10 days during fire; (3) 4 weeks after fire	PM_{10} from air monitors of San Diego Air Pollution Control District (APCD); $PM_{2.5}$ data at 3 sites from air samples; $PM_{2.5}$ at the rest of sites from monitors	69 days (Sep. 28-Dec. 5)
Vora *et al.*, (2011)	Fire period defined a priori; daily$PM_{2.5}$ before, during and after fire	Downtown San Diego air monitors	Three 5-day periods in 2007 (before fire: Oct. 14-18; during fire: Oct. 22-26; after fire: Nov. 13-17)
Wiwatanadate and Liwsrisakun (2011)	Fire period defined a priori; daily CO, O_3, NO_2, SO_2 were measured; hourly $PM_{2.5}$, and PM_{10} used to calculate daily average concentrations and daily 1-h max concentrations	Hourly $PM_{2.5}$ and PM_{10} and daily measures of other pollutants from air monitors of Pollution Control Dept., Ministry of National Resources and Environment	10 months (Aug. 29, 2005- June 30, 2006)

Table A.4: Summary of results on the association between wildfire smoke and health

Category	Study (author)	Statistically significant association?	Association estimates
Birth weight			
1	Prass et al. (2012)	No	No significant association between number of forest fires during pregnancy and birth weight for either girls or boys
2	Holstius et al. (2012)	Yes	Infants whose mothers were exposed to fire episodes during pregnancy had lower birth weight than the non-exposed group by 7.0 g [95% CI: -11.8, -2.2] for 3rd trimester exposure; 9.7 g (95% CI: -14.5, -4.8) for 2nd trimester exposure; and 3.3 g (95% CI: -7.2, 0.6) for 1st trimester exposure
Systemic inflammation (blood biomarker)			
1	Huttunen et al. (2012)	Yes	Median values of IL-12 increased the most of the health outcomes (227%) during fire episode compared to non-fire periods; Fibrinogen and WBC also increased significantly; low ambient PM may also increase systemic inflammation for elderly subjects with coronary heart disease within a few days of exposure
Bone marrow content			
1	Tan et al. (2000)	Yes	Significant increase in percentage of polymorphonuclear neutrophil band cells during the haze (during-haze content: 5%-8.98%), compared to after the haze had cleared (post-haze content: 3.62%-6.12%)
Cardiovascular			
1	Morgan et al (2010)	No	No significant association between cardiovascular diseases and PM_{10}
2	Crabbe (2012)	No	CVD admissions not significantly associated with PM
3	Martin et al (2013)	No	No significant association between smoke event days and CVD health outcomes
4	Moore et al. (2005)	No	No significant difference in CVD diseases in regions exposed to fires compared with unexposed regions
5	Viswanathan et al. (2006)	No	No noticeable increase for chest pain and cardiac arrest visits when exposed to elevated $PM_{2.5}$ and PM_{10}

6	Henderson et al. (2011)	No	No significant association between PM_{10} or plume presence and CVD health outcomes
7	Hanigan et al. (2008)	No	No significant association between CVD diseases and PM_{10}
8	Johnston et al. (2007)	No	No significant association between PM_{10} and CVD admissions
9	Azevedo et al. (2011)	Yes	Significant association between the CVD disease admissions and O_3 >100 μg/m^3
10	CDC (1999)	Yes	ED visits for chest pain increased 37% during fire period compared to non-fire period
11	Lee et al. (2009)	Yes	Significant association between clinic visits for coronary artery disease and PM_{10} during fire period compared to non-fire period.
12	Rappold et al. (2011)	Yes	Cardiopulmonary symptoms increased 23% (95% CI: 1.06-1.43) in counties exposed to wildfire smoke compared to unexposed counties
13	Rappold et al. (2012)	Yes	Relative risk increased 42% (95% CI: 5%-93%) for CHF per 100 μg/m^3 increase in $PM_{2.5}$
14	Delfino et al. (2009)	Yes	Significant 6.1% increase for total CVD disease ($p<0.05$) after fires compared with pre-fire period
Diabetes			
1	Lee et al. (2009)	No	No significant association between $PM_{10}$and diabetes.
Diarrhea			
1	Viswanathan et al. (2006)	No	No significant association for diarrhea and elevated $PM_{2.5}$ or PM_{10}
Mortality			
1	Emmanuel (2000)	No	No significant association between hospital admissions and mortality
2	Hänninen et al. (2009)	No	Positive but not significant 0.8% (-3.5-5.3%) increase in daily mortality per 10 μg/m^3 in same-day $PM_{2.5}$
3	Vedal and Dutton (2006)	No	No statistically significant mortality rate increases during the 2 highest-pollution days compared with other days.

4	Morgan et al. (2010)	No	Positive but not significant association between bushfire-related PM_{10} and all-cause mortality (0.80%; 95% CI: -0.24%-1.86%)
5	CDC (2007)	Yes	Wildfires estimated to cause 12 deaths
6	Jayachandran (2009)	Yes	15,600 child, infant and fetal deaths (1.2% decrease in survival) attributed to fire smoke. Higher effects in poorer districts.
7	Analitis et al. (2011)	Yes	Small fires not associated with mortality. Compared to non-fire periods, medium-size fires were associated with a 4.9% (0.3-9.6%), 6.0% (-0.3-12.6%), and 16.2% (1.3-33.4%) increase in total, CVD, and respiratory mortality, respectively. The 1 large fire had the strongest health effect with a 49.7% (37.2-63.4%), 60.6% (43.1-80.3%), and 92.0% (47.5-150.5%) increase in total, CVD, and respiratory mortality. Increase in deaths due to CVD larger for people <75y; while increase in deaths due to respiratory diseases larger for those >75 y.
8	de Castro, et al. (2009)	Yes	Positive and significant correlations between number of hotspots/fires and respiratory mortality rates for people 65-74y (r=0.76) and >75y (r=0.91). Correlations similar for number of hotspots/fires and COPD mortality rates for those 65-74y (r=0.71) and >75y (r=0.79). Authors noted trend towards more hotspots/fires in more recent years
9	Johnston et al (2011)	Yes	A 5% increase in non-accidental mortality associated with days with bushfire smoke compared with non-smoke days at one day lag (95%CI: 1.00-1.10)
10	Johnston et al. (2012)	Yes	Annually 339,000 deaths attributed to wildfires; Sub-Saharan Africa and Southeast Asia are the most affected regions.
11	Nunes et al, (2013)	Yes	Significant correlations between annual percentage of hours with $PM_{2.5}$ >25μg/m^3 and 1) cardiovascular disease ($r = 0.33$; $p < 0.001$)
12	Sastry (2002)	Yes	Higher mortality when PM_{10} >210 μg/m^3, with 19.2% higher mortality compared to days with PM_{10} <210 μg/m^3. Mortality 21.8% higher on low visibility days in Kaula Lumper and 15.8% higher in Kuching
13	Shaposhnikov *et al*. (2014)	Yes	The interaction between temperature and PM_{10} (largely due to wildfires) contributed over 2000 deaths
Opthalmic symptoms			
1	Aditama, (2000)	Yes	In areas exposed to fire smoke, 100% of doctor visits for eye irritation were due to 10-12 hours of exposure to fire smoke
2	Hänninen et al (2009)	Yes	Smoke was associated with eye symptoms
3	Künzli et al (2006)	Yes	Self-reported indoor smoke exposure lasting for >6 days was significantly associated with all 17 of the questionnaire-assessed health problems, such as itchy eyes (OR=2.26), irritated eyes (OR=2.38),

4	Mirabelli et al (2009)	Yes	Fire smoke significantly raised the risk of eye irritations (PR= 1.81; 95% CI: 1.39-2.34), itchy/watery eyes (PR= 2.12; 95% CI: 1.38-2.94), and any eye symptoms (PR= 1.47; 95% CI: 1.17-1.86)
5	Viswanathan et al (2006)	Yes	Increased doctor visits for eye irritation during the highest fire period
Physical strength and overall health			
1	Frankenberg et al. (2005)	Yes	People living in fire smoke affected areas had substantially poorer general health among middle-age women and older adults; older subjects in exposed areas 10% more likely to report cough and "fair or poor health" than those in non-exposed areas
RESPIRATORY		**Contacts with hospital or clinic**	
1	Azevedo et al. (2011)	No	No significant association between days with? O_3 >100 μg/m^{3}with respiratory disease admissions
2	Smith et al. (1996)	No	No difference in percentage of ED visits due to asthma between during-fire period with similar period in previous year
3	Johnston et al. (2002)	Yes	Significant 20% increase in asthma ED visits (95% CI: 1.09-1.34) per 10 μg/m^3 increase in PM_{10}; asthma ED visits 2.39 times higher (95% CI: 1.46-3.90) on days with PM_{10} >10μg/m^3 compared with days with PM_{10} <10μg/m^3
4	Rappold et al. (2012)	Yes	Risk for asthma ER visits increased 66% (95% CI: 285%-117%) per 100 μg/m^3 increase in $PM_{2.5}$ at lag 0; counties with the lowest SES ranking have 85% higher risk for asthma and 124% higher risk for CHF than counties with the highest SES ranking per 100 μg/m^3 increase in $PM_{2.5}$ when exposed to fire smoke.
5	CDC (1999)	Yes	ED visits increased 91% for asthma, and 132% for bronchitis with acute exacerbation during fire period compared to non-fire period
6	CDC (2008)	Yes	Respiratory ED visits increased during the 5-day fire period compared with the previous 20 weekdays; visits in 6 hospitals increased from 48.6 to 72.6/day for dyspnea and 21.7 to 40.7/ day for asthma comparing the fire periods with non-fire periods
7	Chen et al. (2006)	Yes	During the study period 452 of X days were "fire days"; PM_{10} was significantly associated with respiratory hospital admissions (RR = 1.09-1.16 for lags 0, 1, 3, 5 for PM_{10}> 20μg/m^3 compared to <20μg/m^3); association stronger during fire episodes than non-fire periods (fire periods RR=1.09-1.19; non-fire periods RR=1.09-1.13)
8	Delfino et al. (2009)	Yes	Association between $PM_{2.5}$ and hospital admissions strongest during fires, compared to before and after fires. Compared with pre-fire periods, heavy smoke periods ($PM_{2.5}$ on average 70μg/m^3 higher than pre-fire period) is associated with 34%

			increase in asthma; for 10 μg/m^3 increase in $PM_{2.5}$ association with respiratory admissions strongest for people 65-99y (10.1%, 95%CI: 3.0%-17.8%) and 0-4y (8.3%, 95%CI: 2.2%-14.9%)
9	Dohrenwend et al, (2013)	Yes	significant increase during fire period compared with pre-fire period in average visit counts for dyspnea (increased 3.2 visits per day) and asthma (increased by 2.6 visits per day).
10	Duclos, (1990)	Yes	Visits of asthmatics increased 40% and COPD increased 30% during fire period compared with reference period; patients with laryngitis 1.6-2.2 times more likely to visit doctors during fire period compared with reference period; visits of persons with sinusitis and upper respiratory infections also increased
11	Emmanuel, (2000)	Yes	A 30% increase in outpatient attendance during the period affected by smoke compared to non-haze period; 100μg/m^3 increase in PM_{10} during fire (from 50 to 150μg/m^3) was associated with 12%, 19%, and 26% increases in upper respiratory tract problems, asthma, and rhinitis, respectively.
12	Hanigan et al. (2008)	Yes	Significant positive relationship between respiratory diseases and PM_{10}; increase of 10μg/m^3 in PM_{10} associated with a 4.81% (95%CI: -1.04%, 11.01%) increase in total respiratory admissions; indigenous Australians (with disadvantaged SES and high risk of chronic diseases) are more vulnerable.
13	Henderson et al. (2011)	Yes	Based on measurements from TOEMS, a 30 μg/m^3 increase in PM_{10} was associated with a 5% increase in all respiratory physician visits, a 16% increase in asthma-related physician visits, and a 15% increase in hospital admissions for respiratory conditions. Based on CALPUFF model, a 60 μg/m^3 increase in PM_{10} was associated with 11% increase in for respiratory conditions, and 4% increase in asthma-related physician visits. Based on SMOKE, the presence of smoke plume was associated with 21% increase in asthma-related physician visits. People 30-40y are affected the greatest for respiratory diseases, while people 40-50y are most affected for CVD diseases. No differences by sexes, socioeconomic status, or pre-existing sensitivity
14	Ignotti et al. (2010)	Yes	A 1% increase in annual hours of $PM_{2.5}$ >80μg/m^3 associated with 5%, 8% and 10% increases in hospital admissions for the intermediate age group, children, and the elderly, respectively
15	Lee et al. (2009)	Yes	15% increase in total clinic visits during a 12-week fire period in 1999 compared with the same period in 1998 (control period); significantly association between clinic visits for asthma and headaches and PM_{10} during fire period compared to non-fire period;
16	Martin et al (2013)	Yes	Significant 5% increase (OR=1.05, 95%CI=1.02-1.09) in respiratory admissions on smoke event days compared with control days
17	Moore et al. (2005)	Yes	In Kelowna: Doctor visits for respiratory health problems increased 46%, 54%, and78% during the second, fourth and fifth week of intense forest fires compared to aggregated rate from 1993 to 2002; in Kamloops: doctor visits were not significantly different from the aggregate rate from 1993 to 2002.
18	Morgan et al. (2010)	Yes	A 10μg/m^3 increase of PM_{10} from bushfires was associated with increase in hospital admissions: 1.24% (95% CI: 0.22%-2.27%) for respiratory disease at lag 0 for all ages, 2.31% (95% CI: 0.69%-3.96%) for respiratory disease at lag 2 for

			people >65 y, 3.80% (95% CI: 1.40%-6.26%) for COPD at lag 2 for people >65 y, and 5.02% (95% CI: 1.77%-8.37%) for asthma at lag 0 for people 15-64 y; more significant associations for elderly populations in respiratory-related diseases
19	Mott et al. (2002)	Yes	Clinic visits for respiratory illness increased by 52% during the weeks of the fire compared with the same period in 1998; weekly PM_{10} positively correlated with weekly counts of clinic visits in the same periods in1998 and 1999 (r=0.74); use of high-efficiency particulate air filters (OR=0.54), and ability to recall public service announcements (OR=0.25), were both associated with lower odds of self-reported lower respiratory tract health effects
20	Mott et al. (2005)	Yes	Compared with the same period in previous years, COPD hospitalization during fire period increased 50% among people 40-64y and 42% among people >65 y; asthma hospitalizations increased 83% among people 40-64y and 22% among people 19-39y during fire periods Aug. 1- Oct. 31, 1997
21	Schranz et al. (2010)	Yes	ED visits 5.8% higher during the fire than a few days after the fire; significant increase in complaint of shortness of breath (6.5% vs. 4.2% p = 0.028) and smoke exposure (1.1% vs. 0%/ = 0.001) following the fires; patients with significant cardiac or pulmonary histories not more likely to present to the ED during fires.
22	Viswanathan et al. (2006)	Yes	Significant increase in asthma visits associated with elevated $PM_{2.5}$ and PM_{10} during fire period compared with pre- and post-fire period
23	Aditama (2000)	Yes	In Jambi, respiratory disease doctor visits increased 51% during haze period compared to non-haze period; 70% of respiratory patients had worse symptoms during haze period.
24	de Mendonça (2006)	Yes	Per unit increase of area of forests burned in each district, the number of in-patient treatments of respiratory ailments caused only by fire significantly increased 29.07%
25	Johnston et al. (2007)	Yes (in sub population)	Positive but non-significant relationship between PM_{10} (10μg/m^3 increase) and admissions for respiratory diseases (OR 1.08 95%CI 0.98-1.18); the relationship was stronger in the Indigenous subpopulation (OR1.17 95% CI 0.98-1.40); Indigenous population with ischemic heart disease have greatest risk at a lag of 3 days (OR 1.71 95%CI 1.14-2.55)
26	Crabbe (2012)	Yes	Respiratory admissions increased 2.5% per 10μg/m^3 of PM_{10} at 1-day lag (95%CI: 1.000-1.051); respiratory admissions significantly increased 9.1% per 10μg/m^3 of FPM at 1-day lag (95%CI: 1.023-1.163).
27	Frankenberg et al. (2005)	Yes	Substantial negative effect on respiratory health
28	Mascarenhas et al. (2008)	Yes	Significant positive correlation between $PM_{2.5}$ concentrations and asthma-related ER visits (r=0.59), and incidence of respiratory disease-related ER visits was higher among children <10 y. Positive relationship between the 7-day moving average $PM_{2.5}$ level and the number of asthma-related ER visits.
29	Shusterman et al. (1993)	Yes	Half of the total visits were smoke-related disorders

30	Tham et al. (2009)	Yes	Significant association between PM_{10} (when increased from 25th to 75th percentile of its levels) and respiratory ED attendances; hospital admissions, but not ED visits, were associated with increases in O_3 from the 25th to 75th percentile.
31	Thelen et al (2013)	Yes	Risk for respiratory ER visits increased 50% during peak fire PM concentrations compared to non-fire conditions (not clear whether significant or not)
32	do Carmo et al. (2010)	Yes	Every 10μg/m^3 increase in particulate exposure was associated with 2.9% and 2.6% increase in primary care visits for respiratory disease in children on the 6th and 7th days following exposure, respectively
33	Rappold et al. (2011)	Yes	Compared with counties not exposed to fire smoke, exposed counties' ED visits for respiratory diseases increased 66% (95% CI: 1.38-1.99), for asthma increased 65% (95% CI: 1.25-2.17), for COPD increased 73% (95% CI: 1.06-2.83), pneumonia and acute bronchitis increased 59% (95% CI: 1.07-2.34), for heart failure-related condition increased 37% (95% CI: 1.01-1.85); females have greater increase in "asthma, pneumonia, acute bronchitis, and URI"; males have greater increase in COPD; people <65 have greater increase in asthma, COPD, pneumonia, and acute bronchitis compared to people ≥65
		Medication Dispensation or Use	
34	Elliott et al. (2013)	Yes	Per 10 μg/m^3 increase in $PM_{2.5}$ level, the dispensation of salbutamol (use of ventolin) in populations exposed to forest fires significantly increased 6% (95% CI: 4%-7%). This result suggests a potentially higher respiratory morbidity during the fire events.
35	Vora et al. (2011)	Yes	The subject used significantly more rescue medication during the wildfires (2.6 ±2.0 does per day) than before the fires (0.94 ±1.3 does per day) which was associated with $PM_{2.5}$ values.
36	Caamano-Isorna et al., (2011)	Yes	Significant increase in consumption of drugs for obstructive airway diseases for both men (increased 10.29%) and women (increased 12.09%)
		Symptoms	
37	Johnston et al. (2006)	Yes	Significant associations between minor asthma symptoms and 1) per 10 μg/m^3 increase in PM_{10}: interval rate ratio =(1.240, 1.317); and 2) per 5 μg/m^3 increase in $PM_{2.5}$: OR=(1.042, 1.076))
38	Vora et al. (2011)	Yes	25% of the subjects showed increases in sputum eosinophil counts and increased airways inflammation during fire period compared with pre- and post-fire periods
39	Sutherland et al. (2005)	Yes	Peak air pollution events significantly elevated (worsened) symptom scores compared to before the air pollution event (baseline score=20; spike-day score=21.5; p=0.0002)
40	Kolbe and Gilchrist (2009)	Yes	About 70.4% of the survey respondents reported to have at least one respiratory or depression symptoms during fire

41	Kunii et al. (2002)	Yes	98.7% of survey respondents reported an exacerbation of symptoms during fire; 91.3% of them had respiratory symptoms during fire; 13.1% of the respondents self-assessed their health problems as severe
42	Künzli et al. (2006)	Yes	Self-reported indoor smoke exposure lasting for >6 days was significantly associated with all 17 of the questionnaire-assessed health problems, such as dry cough (OR=2.24-2.67), and wheezing (OR=2.15-2.29). The risk of having medication, visiting a doctor, and missing school for health problems increased 82%, 33% and 59%, respectively; associations are stronger for children without asthma when exposed to smoke
43	Mirabelli et al. (2009)	Yes	Strongest impact of fire smoke on students with the lowest quartile of airway size; for students in that quartile with exposure to fire smoke more than 6 days, fire smoke significantly raised the risk of wheezing (prevalence ratio (PR)=31.5; 95% CI: 4.14-239), morning dry cough (PR= 4.49; 95% CI: 1.83-11.0), night-time dry cough (PR= 4.55; 95% CI: 1.86-11.1)
44	Wiwatanadate and Liwsrisakun (2011)	Yes	In non-asthmatic population, SO_2 (lag4) was significantly positively associated with night-time PEFR (general linear mixed model regression coefficient range 0.88-1.00 in 11 models). PM_{10} (lag5) marginally associated with nigh-time PEFR (coefficient=0.02, 95% CI: 0.00-0.04).
45	Jalaludin et al. (2010)	Yes (in sub population)	Significant association between PM_{10} and PEFR for children with no bronchial hyper-reactivity: one $\mu g/m^3$ increase in PM_{10} would lead to a decrease of 0.10 in PEFR. No significant association was found (p=0.86) in general populations.