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Critical Review of Health Impacts of Wildfire Smoke Exposure

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Abstract

Background: Wildfire activity is predicted to increase in many parts of the world due to changes in temperature and precipitation patterns from global climate change. Wildfire smoke contains numerous hazardous air pollutants and many studies have documented population health effects from this exposure.

Objectives: We aimed to assess the evidence of health effects from exposure to wildfire smoke and to identify susceptible populations.

Methods: We reviewed the scientific literature for studies of wildfire smoke exposure on mortality and on respiratory, cardiovascular, mental, and perinatal health. Within those reviewed papers deemed to have minimal risk of bias, we assessed the coherence and consistency of findings.

Discussion: Consistent evidence documents associations between wildfire smoke exposure and general respiratory health effects, specifically exacerbations of asthma and chronic obstructive pulmonary disease. Growing evidence suggests associations with increased risk of respiratory infections and all-cause mortality. Evidence for cardiovascular effects is mixed, but a few recent studies have reported associations for specific cardiovascular endpoints. Insufficient research exists to identify specific population subgroups that are more susceptible to wildfire smoke exposure.

Conclusions: Consistent evidence from a large number of studies indicates that wildfire smoke exposure is associated with respiratory morbidity with growing evidence supporting an association with all-cause mortality. More research is needed to clarify which causes of mortality may be associated with wildfire smoke, whether cardiovascular outcomes are associated with wildfire smoke, and if certain populations are more susceptible.

Introduction

Wildfires are a global occurrence. Changes in temperature and precipitation patterns from climate change are increasing wildfire prevalence and severity (Westerling et al. 2006; Settele et al. 2014) resulting in longer fire seasons (Flannigan et al. 2013; Westerling et al. 2006) and larger geographic area burned (Gillett et al. 2004). Wildfire smoke contains many air pollutants of concern for public health, such as carbon monoxide (CO), nitrogen dioxide, ozone, particulate matter (PM), polycyclic aromatic hydrocarbons (PAHs), and volatile organic compounds (Naeher et al. 2007). Current estimated annual global premature mortality attributed to wildfire smoke is 339,000 (interquartile range of sensitivity analyses: 260,000-600,000) (Johnston et al. 2012), but the overall impact on public health in terms of respiratory, cardiovascular, and other morbidity effects is unknown. A better synthesis of current knowledge on the health effects of wildfire smoke is needed to guide public health responses.

Wildfire smoke epidemiology is an active area of research (Henderson and Johnston 2012) with new methods uncovering associations that were previously undetectable. Studies of health outcomes associated with wildfire smoke exposure tend to be retrospective and researchers have to rely on administrative health outcome data such as mortality or hospitalization records. Achieving adequate statistical power has been challenging because such severe outcomes are less common, fires tend to be episodic and short in duration, and exposed populations from individual events are often small. Many recent studies have increased statistical power by investigating very high exposure events that last for longer periods, large populations over many years in regions with frequent fires, more common health outcomes such as medication dispensations, or a combination of these methods.

Previous reviews of wildfire health impacts have either not included the full range of health endpoints associated with community exposure to wildfire smoke (Dennekamp and Abramson 2011; Henderson and Johnston 2012) or have summarized the literature without critical analysis of specific studies (Finlay et al. 2011; Liu et al. 2014; Youssouf et al. 2014). Our review follows a modified version of the systematic review methodology outlined in Woodruff and Sutton (2014) to analyze studies critically and only evaluate the strongest evidence.

Methods

We searched PubMed, Web of Science, and PsychInfo to identify scientific papers related to wildfire smoke exposure and relevant health outcomes. We conceptualized wildfires as those within the definition of landscape fires defined in Johnston et al. (2012). Our search strategy (Figure 1) yielded 778 journal articles in PubMed and 1248 journal articles in Web of Science in November 2013. We then selected studies that potentially focused on human health effects related to wildfire smoke based on title and yielded 248 journal articles from PubMed and 217 from Web of Science. After discarding duplicates, 350 articles remained. PsychInfo did not yield any new peer-reviewed journal articles.

After reading abstracts, we removed articles if they assessed only exposure and not associated health effects, reported health surveillance outcomes without analysis of associations with exposure, did not analyze primary or secondary health data, did not adequately describe the exposure assessment or it was not clearly related to wildfire smoke, or were not published fully in English. This yielded 103 studies that we reviewed. We continually searched for new papers and subsequently added 12 more by August 2015. These papers included human experimental studies of woodsmoke, studies of effects on wildland firefighters, and studies whose outcomes were self-reported respiratory symptoms associated with wildfire smoke, but these are not included in this paper.

From the remaining epidemiological studies (N=53), we extracted information and made an expert judgment on the risk of bias for each study based on their sample size, exposure assessment methods, control for potential confounding factors, and use of objective outcome measures (Supplemental Table S1). We deemed studies to have a lower risk of bias if there were no concerns in any of these categories, moderate risk if there were minor concerns in one or more categories, and higher risk if either there were multiple concerns about bias or if one concern was sufficiently large based on our collective judgment.

All evaluation of results from these studies is based on the authors' interpretation of the reported findings in each paper. In this review "significant" means a 95% confidence interval (CI) that does not include the null, "suggestive" means a 95% CI that does include the null but would not with a slightly relaxed criterion such as a 90% CI, and "no association" means that the 95% CI includes the null with no indication of a relationship. We assumed that exposure to smoke from all types of landscape fires were comparable. We use the term wildfire to refer to all types of landscape fires.

Assessing human exposure to wildfire smoke is challenging for many reasons. Wildfires tend to occur in rural areas in which air pollution monitoring networks might be absent or less comprehensive than in cities. The studies we reviewed used various exposure assignment methods such as self-report, assignment to the nearest regulatory air pollution monitor, comparison of fire periods to non-fire periods, and use of satellite data or air quality modeling output. Heterogeneity of exposure assessment methods across studies (Supplemental Table S1 and Table 1) made a quantitative meta-analysis of effect estimates inappropriate. While publication bias could be present in this literature, we could not assess its extent due to the scarcity of studies for each health outcome.

Results

Our review covers the following health outcomes: mortality, respiratory morbidity, cardiovascular morbidity, birth outcomes, and mental health. We further discuss the evidence from toxicological studies and for susceptible population subgroups. Supplemental Table S1 provides more details on reviewed studies.

After review of 53 epidemiological papers, we evaluated 27 as having lower potential for bias, 17 as moderate potential for bias and 10 as higher potential for bias. Of the 10 deemed to have higher risk of bias, four did not adequately adjust for important covariates (Azevedo et al. 2011; Cooper et al. 1994; Prass et al. 2012; Resnick et al. 2015), two were likely underpowered due to small sample size (Cooper et al. 1994; Vedal and Dutton 2006), three used retrospective self-report for exposure assessment with high potential for bias (Ho et al. 2014; McDermott et al. 2005; Marshall et al. 2007), and the exposure assessment in two other studies was not clearly related to smoke from wildfires (Analitis et al. 2012, Caamano-Isorna et al. 2011). The remaining 43 studies deemed to have low to moderate risk of bias are discussed below. More detail on the findings from each study is provided in Supplemental Table S2.

Mortality

Growing evidence from the most recent, adequately statistically-powered studies demonstrates associations between wildfire smoke exposure and all-cause mortality, but more studies are needed to determine whether specific causes of mortality are most affected.

A study of the 1997 southeast Asian wildfire found an increase in mortality in Malaysia associated with a measure of visibility and measured PM_{10} (PM ≤ 10 microns in aerodynamic diameter) both linearly and with various discrete levels of PM_{10} (Sastry 2002). A study of the 2010 heat wave and wildfires in Moscow reported findings of an interaction between high temperatures and high PM_{10} on deaths and that smoke exposure was responsible for about 29% of the 10,859 excess deaths during the 44-day heat wave (Shaposhnikov et al. 2014). A cross-sectional analysis of cardiovascular mortality among people over 65 years in the Brazilian Amazon, where the predominant source of air pollution is from wildfires, found a significant association between the percentage of hours of $PM_{2.5}$ over 25 $\mu g/m^3$ and cardiovascular mortality (Nunes et al. 2013).

The most recent studies of wildfire smoke and mortality take advantage of long time series data and provide growing evidence of significant increases in mortality. A study of 13.5 years of data including 48 days affected by wildfire smoke in Sydney, Australia, demonstrated a significant increase in mortality associated with smoke-affected days (Johnston et al. 2011). An earlier study of mortality in Sydney, using eight years of data, found a suggestive increase in mortality associated with wildfire-related PM_{10} (Morgan et al. 2010). A meta-analysis of data from 2003-2010 in 10 cities in southern Europe found increases in cardiovascular mortality associated with PM_{10} that were stronger on smoke-affected days than on non-affected days, but smoke was not significantly associated with respiratory mortality (Faustini et al. 2015). In Madrid, mortality, but not specifically respiratory or cardiovascular mortality, was associated with PM_{10} on days with advection events associated with biomass burning (Linares et al. 2014). Further multi-year studies in regions regularly affected by wildfire smoke could help clarify if specific causes of mortality are associated with wildfire smoke exposure.

Respiratory Morbidity

Epidemiological studies have demonstrated significant associations between wildfire smoke exposure and declines in lung function among non-asthmatic children (Jacobson et al. 2012 and 2014), and increases in physician visits for respiratory problems (Henderson et al. 2011; Lee et al. 2009; Moore et al. 2006; Mott et al. 2002), respiratory emergency department (ED) visits (Johnston et al. 2014; Rappold et al. 2011; Tham et al. 2009; Thelen et al. 2013) and respiratory hospitalizations (Cancado et al. 2006; Chen et al. 2006; Delfino et al. 2009; Henderson et al. 2011; Ignotti et al. 2010; Martin et al. 2013; Morgan et al. 2010; Mott et al. 2005). Findings for specific respiratory endpoints are reviewed below.

<u>Asthma</u>

Evidence from multiple epidemiological studies demonstrates that wildfire smoke exposure contributes to exacerbations of asthma. Studies have documented increased physician visits (Henderson et al. 2011; Yao et al. 2014), ED visits (Duclos et al. 1990; Johnston et al. 2002; Johnston et al. 2014; Rappold et al. 2011) and hospitalizations (Arbex et al. 2007; Delfino et al. 2009; Martin et al. 2013; Morgan et al. 2010; Mott et al. 2005) for asthma associated with wildfire smoke exposure. Some studies found suggestive increases in asthma ED visits (Smith et al. 1996) and asthma hospital admissions (Johnston et al. 2007); these studies may have lacked statistical power due to short time periods (Smith et al. 1996) or small affected populations (Johnston et al. 2007). Another study did not find a significant increase in ED visits or hospitalizations among a cohort of asthmatic children in the year after large wildfires in San Diego, California compared to the year prior to those fires (Tse et al. 2015).

Four studies demonstrated no significant acute changes in lung function among people with asthma related to PM from wildfires (Jacobson et al. 2012; Jalaludin et al. 2000; Vora et al. 2011; Wiwatanadate & Liwsrisakun 2011), although significant declines in lung function were found among those without asthma (Jacobson et al. 2012) and children without bronchial hyper-reactivity (Jalaludin et al. 2000). One possible explanation for these counter-intuitive findings is increased use of rescue medication in response to elevated levels of smoke among those diagnosed with asthma as was found in one (Vora et al. 2011) of two studies (Vora et al. 2011; Jacobson et al. 2012) that investigated this mechanism.

Other studies documented associations between medication usage for obstructive lung disease and wildfire smoke exposure. Both usage of reliever medication and initiation of oral steroid use were associated with wildfire smoke in a panel study of adults and children in Australia (Johnston et al. 2006). People with asthma reported elevated levels of rescue medication usage during a wildfire in Southern California (Vora et al. 2011). Dispensations of reliever medications were related to metrics of wildfire smoke exposure in British Columbia (Elliott et al. 2013; Yao et al. 2014). Researchers found increases in physician-dispensed short-acting beta-agnoists but not physician-prescribed oral corticosteroids for children with asthma in years after two catastrophic wildfires in southern California compared to the year prior to each wildfire (Tse et al. 2015). An association between visits to hospitals for inhalation therapy and daily mass of air particle sediment collected in four nearby water containers was found during one sugarcane burning season in Brazil (Arbex et al. 2000).

All previously mentioned studies examined exacerbations of asthma, whereas only one study investigated incident asthma related to wildfire smoke. Methodological concerns in that portion of the study suggest a high potential for bias as new diagnoses occurring after, but not during, two large wildfire episodes were included (Tse et al. 2015).

Chronic Obstructive Pulmonary Disease (COPD)

Epidemiological evidence of associations between wildfire smoke exposure and exacerbation of COPD is mounting. Elevated rates of hospitalizations (Delfino et al. 2009; Johnston et al. 2007; Martin et al. 2013; Morgan et al. 2010; Mott et al. 2005), ED visits (Duclos et al. 1990; Johnston et al. 2014; Rappold et al. 2011) and physician visits for COPD (Yao et al. 2014) have been associated with wildfire smoke exposure. Additionally, the findings of increased reliever medication dispensing during wildfire smoke exposure in British Columbia may indicate increases in COPD or asthma exacerbations (Elliott et al. 2013; Yao et al. 2014).

Respiratory Infections

The evidence for associations between wildfire smoke exposure and respiratory infections is inconsistent. Duclos et al. (1990) found a higher rate of ED visits for respiratory infections during major wildfires in California compared to a reference period. Rappold et al. (2011) found a suggestive increase in ED visits for upper respiratory infections in smoke-affected counties in North Carolina during peat fires compared to a reference period and this temporal increase was not found in non-smoke-affected counties. Henderson et al. (2011) and Yao et al. (2014), however, found no association between wildfire smoke exposure and physician visits for upper respiratory infections in British Columbia. Johnston et al. (2007) reported no association between PM predominantly from wildfires and hospitalizations for respiratory infections in Australia.

The evidence suggests, however, an association between wildfire smoke exposure and acute bronchitis and pneumonia. Although Johnston et al. (2014) did not find an association between ED visits for pneumonia and bronchitis associated with wildfire smoke in Australia, most other studies did. Yao et al. (2014) found significant increases in physician visits for lower respiratory infections associated with $PM_{2.5}$ over 10 fire seasons in British Columbia. Rappold et al. (2011) documented increased ED visits for pneumonia and acute bronchitis associated with exposure to smoke from a peat fire. Duclos et al. (1990) found higher rates of hospitalization for bronchitis during a wildfire compared to a reference period. Moreover, Martin et al. (2013) reported associations between days with high levels of bushfire smoke and hospitalizations for pneumonia and acute bronchitis in Newcastle, Australia, although this association was not found in the larger city of Sydney; the authors attribute this to lack of precision in estimates of specific respiratory outcomes. Two studies have documented similar associations between wildfire smoke and background PM with bronchitis and pneumonia (Delfino et al. 2009; Morgan et al. 2010), suggesting that effects of wildfire and urban PM on these outcomes are similar.

Cardiovascular Morbidity

Results from studies of associations between cardiovascular outcomes and wildfire smoke exposure are inconsistent. Many studies of wildfire smoke exposure have found no associations with grouped cardiovascular disease outcomes (Hanigan et al. 2008; Henderson et al. 2011; Johnston et al. 2007; Johnston et al. 2014; Lee et al. 2009; Martin et al. 2013; Moore et al. 2006; Morgan et al. 2010; Rappold et al. 2011; Yao et al. 2014), although a few have documented evidence for specific endpoints. Rates of out-of-hospital cardiac arrests were associated with wildfire-related $PM_{2.5}$ in Australia (Dennekamp et al. 2015; Haikerwal et al. 2015). Hospitalizations but not ED visits for acute myocardial infarctions (MI) were associated with wildfire-related $PM_{2.5}$ during the same fires (Haikerwal et al. 2015). ED visits for congestive heart failure (CHF) were associated with wildfire smoke exposure from a peat fire in North Carolina (Rappold et al. 2011), but only a suggestive association was found for CHF hospitalizations and $PM_{2.5}$ during a wildfire in southern California (Delfino et al. 2009). Johnston et al. (2014) did not find any association between wildfire smoke and ED cardiac failure. Other studies have found no associations between wildfire smoke exposure and CHF (Martin et al. 2013; Morgan et al. 2010) or cardiac dysrhythmias (Delfino et al. 2009; Johnston et al. 2014; Martin et al. 2013). And no associations were found in the one study that investigated angina in relation to wildfire $PM_{2.5}$ (Haikerwal et al. 2015)

Study results are also mixed for ischemic heart disease (IHD). Higher counts of hospitalizations for IHD than expected based on historical data were found in Sarawak, Malaysia during the prolonged very high PM levels of the 1997 Southeast Asian wildfires (Mott et al. 2005). ED visits for IHD were higher on smoke-affected days in Sydney, Australia (Johnston et al. 2014), but two other studies in Australia (Martin et al. 2013; Morgan et al. 2010) and one in California (Delfino et al. 2009) reported no associations for IHD hospital admissions . A study in Darwin, Australia found increased risk of IHD hospitalizations only among the Indigenous population, whereas the results suggested an inverse association among the whole population (Johnston et al. 2007). Researchers also found a positive association between PM_{10} during a wildfire and clinic visits for IHD in a Native American reservation in California (Lee et al. 2009).

Very few studies have investigated other cardiovascular outcomes, making definitive conclusions difficult. Arbex et al. (2010) found increases in hospitalizations for hypertension associated with exposure to total suspended particles over two years within a community seasonally exposed to smoke from sugar cane burning, but there was no clear difference in this finding between burning and non-burning periods, which implies that the relationship may not be due to the source of the particles. Henderson et al. (2011) did not find any relationship between PM_{10} during a wildfire and physician visits for hypertension. One (Delfino et al. 2009) of three (Delfino et al. 2009; Morgan et al. 2010; Johnston et al. 2014) studies to investigate cerebrovascular disease or stroke found a suggestive association with wildfire smoke exposure.

Too few studies and too many inconsistencies in findings exist to determine whether wildfire smoke exposure is associated with specific cardiovascular outcomes, despite evidence that exposure to ambient PM is associated with increased risk of cardiovascular morbidity (Brook et al. 2010).

Birth Outcomes

Corroborative evidence suggests that wildfire smoke exposure effects on birth outcomes are plausible. For example, a growing literature exists on associations between adverse birth outcomes and exposure to ambient air pollution (Woodruff et al. 2010), to wood smoke from household cooking and heating in developing countries (e.g., Lakshmi et al. 2013) and to household heating in developed countries (Gehring et al. 2014). While these exposures are chronic compared to the more acute nature of exposure to smoke from some wildfires, some studies have demonstrated links between wildfire smoke exposure and birth outcomes. Holstius et al. (2012) found lower birth weights, overall and for the second and third trimesters specifically, for babies that gestated during the 2003 southern California wildfires compared to babies from the same region born before or more than nine months after the fires. Jayachandran (2009) found that prenatal smoke exposure from the 1997 Southeast Asian wildfire in the third trimester was the most important predictor of ‘missing’ children from the Indonesian 2000 Census, the only way to estimate early life deaths from the scant data in Indonesia. Pregnant women exposed to very high levels of $PM_{2.5}$ from agricultural burning in the Brazilian Amazon had higher rates of low birthweight babies compared to those exposed to lower levels (Candido da Silva et al. 2014).

Mental Health Outcomes

Although many studies have documented evidence of psychological impairment related to wildfires (e.g. Papanikolaou et al. 2011), few have investigated smoke exposure as a cause.

We found six studies that investigated the association between objective mental health impacts and wildfire smoke exposure, however four of those were deemed to have higher potential for bias (Ho et al. 2014; McDermott et al. 2005; Marshall et al. 2007; Caamano-Isorna et al. 2011). In the two studies that remain, one found no increase in physician visits for mental illness associated with PM during the 2003 wildfire season in British Columbia (Moore et al. 2006) and the other found no increase in mental health hospitalizations during the 1987 California fires compared to a reference period (Duclos et al. 1990).

Toxicological Studies

A major pathway by which PM causes respiratory effects is through pulmonary oxidative stress and inflammation (Nakayama Wong et al. 2011). Systemic responses are the main pathways through which PM is thought to influence cardiovascular health. These are hypothesized to be induced either directly by the movement of pro-inflammatory, pro-coagulation, and pro-oxidant components of PM to the circulation, indirectly as a consequence of the pulmonary changes induced by PM, or through PM-mediated changes in the autonomic nervous system (Brook et al. 2010; Delfino et al. 2010).

In vivo animal studies of wildfire-derived PM exposure compared to controls have demonstrated increased oxidative stress and cell death in mice (Williams et al. 2013), and lower counts of lung macrophages, higher levels of inflammatory cells and cytokines, and greater antioxidant depletion in a study of smoke from a California wildfire in a mouse model (Wegesser et al. 2009; Wegesser et al. 2010). Similarly, increased respiratory inflammation and reduced lung mechanics compared with controls was documented from a mouse study of biomass smoke from sugar cane burning in Brazil (Mazzoli-Rocha et al. 2008). *In vivo* studies in humans have also demonstrated increased inflammatory responses, specifically elevated band neutrophil counts in peripheral blood (Tan et al. 2000) and elevated cytokines (van Eeden et al. 2001) associated with air pollution levels during the 1997 Southeast Asian wildfires.

In vitro studies have documented increased inflammation in rat alveolar macrophages exposed to $PM_{2.5}$ from prescribed fires (Myatt et al. 2011) and in human bronchial epithelial cells exposed to wildfire-derived $PM_{2.5}$ compared to cells exposed to ambient PM (Nakayama Wong et al. 2011). After exposure to wildfire-derived PM, human lung epithelial cells showed declines in glutathione, an important antioxidant (Pavagadhi et al. 2013); mouse peritoneal monocytes showed increased hydrogen peroxide production and oxygen radical generation (Leonard et al. 2007); and mouse macrophages (Franzi et al. 2011), rat macrophages (Myatt et al. 2011), and human lung epithelial cells (Pavagadhi et al. 2013) had increased cell death.

Oxidative stress can also lead to DNA damage. All size fractions of PM extracted from wildfire smoke caused DNA damage in mouse peritoneal monocytes (Leonard et al. 2007). Studies in regions near sugarcane burning in the Brazilian Amazon observed higher numbers of micronucleated cells, a measure of genotoxicity, in buccal cells from children in highly smoke-affected areas compared to children in a control community (Sisenando et al. 2012); however, it is unclear if the higher pollution in the study communities was solely due to agricultural burning because two factories are located in the exposed but not in the control region. Another study found more micronucleated buccal cells in sugarcane workers compared to nearby hospital administrative workers (Silveira et al. 2013), but the authors do not mention any control for other differences in these two populations that could explain this finding.

A recent study demonstrated the potential for early life exposure to wildfire smoke to confer immune effects, measured as reduced cytokine synthesis in peripheral blood cells, lasting into adolescence in Rhesus Macaque monkeys (Miller et al. 2013). Short-term inhalation of wood smoke in general and not specifically from a wildfire can compromise lung immune responses which may be one reason for the observed increased likelihood of lung infections in children exposed to wood smoke (Zelikoff et al. 2002). There is therefore growing evidence to support the theory that incidence of respiratory infections can be increased by exposure to wildfire smoke.

In summary, existing toxicological evidence supports potential respiratory and cardiovascular health effects of wildfire smoke exposure. The body of evidence, however, is relatively small compared to toxicological studies of general PM.

Vulnerable Populations

Few epidemiological studies have investigated whether specific populations are more susceptible to wildfire smoke exposure. Susceptibility factors investigated include those related to lifestage, pre-existing disease, socioeconomic status, and ethnicity. Unless otherwise stated, all subgroup differences are based on observed changes in the magnitudes of point estimates, not on significance tests.

The findings for differential effects by age are inconclusive. A study of PM_{10} exposure in Malaysia from the 1997 Southeast Asian wildfires found higher rates of mortality in people aged 65-74 compared to others; a smaller suggestive effect was found in those 75 and older (Sastry 2002). People 65 years and older had higher rates of respiratory hospitalizations compared to younger adults exposed to biomass burning in the Brazilian Amazon (Ignotti et al. 2010) and wildfire smoke in Australia (Morgan et al. 2010). Such older adults were also found to have higher rates of hospitalization for asthma than their younger counterparts during California wildfires (Delfino et al. 2009), and higher rates of out-of-hospital cardiac arrests and hospitalizations for IHD in Victoria, Australia (Haikerwal et al. 2015).

Other studies, however, have found higher effects for younger adults than older adults. Wildfire PM-related respiratory admissions during Indonesian wildfires exceeded predictions for 40-64 year-olds but not for those 65+ (Mott et al. 2005). Similarly, ED visits for COPD, and pneumonia and acute bronchitis were more strongly associated with peat fire smoke among people under 65 compared to people 65+ in North Carolina (Rappold et al. 2011). Although respiratory physician visits were associated with PM_{10} in people aged 60-70 and 80+ in a British Columbia wildfire, younger adults exhibited stronger associations (Henderson et al. 2011). No differences were found in either of the two studies that investigated differential effects by age for cardiovascular outcomes (Morgan et al. 2010, Henderson et al. 2011).

Children with asthma did not experience increased respiratory symptoms or medication use during Australian wildfires, whereas adults did (Johnston et al. 2006). Similarly, the highest PM-related increase in physician visits for asthma during a wildfire in British Columbia was found for adults (Henderson et al. 2011), as was true for ED visits for asthma on smoke-affected days in Australia (Johnston et al. 2014). Asthma hospitalizations among children ages 0-5 were more strongly associated with wildfire $PM_{2.5}$ exposure than were asthma hospitalizations for both older children and adults under 65 during a California wildfire; but the greatest association was found for people 65+ (Delfino et al. 2009).

Some studies have used previous health care utilization as a measure of pre-existing health conditions. One study found no effect modification by number of physician visits in the previous year (Henderson et al. 2011). In contrast, people 65+ who were hospitalized for any cardiorespiratory outcome in the first half of the year were at increased risk of being hospitalized during the 1997 Southeast Asian fires compared with similar temporal comparisons in previous years without fires (Mott et al. 2005). Pre-existing cardiac or respiratory conditions may plausibly increase vulnerability to wildfire smoke exposure; however, the available evidence is currently inconclusive.

A recent study found that body mass index modified the association of wildfire smoke exposure on exacerbations of asthma, as measured by prevalence of physician-dispensed short-acting beta-agonists for children with asthma in southern California (Tse et al. 2015).

Few studies have investigated how socio-economic status (SES) influences responses to wildfire smoke exposure. Henderson et al. (2011) noted findings of no effect modification by neighborhood SES on associations between wildfire smoke exposure and physician visits in British Columbia, Canada, but detailed results were not presented. In contrast, North Carolina counties with lower SES had higher rates of ED visits for asthma and CHF compared to counties with higher SES affected by peat fire smoke (Rappold et al. 2012). Similarly, in Indonesia, districts with lower food consumption demonstrated larger negative associations between smoke exposure and survival of birth cohorts than those with higher household food consumption (Jayachandran 2009).

To our knowledge only one ethnic subgroup has been studied in relation to differential health outcomes associated with wildfire smoke exposure. Indigenous people in Australia experienced higher rates of hospitalization for respiratory infections (Hanigan et al. 2008), and IHD (Johnston et al. 2007) associated with exposure to bushfire smoke than non-indigenous people. This effect may be explained by underlying health status, access to medical services, or other social characteristics in this group (Martin et al. 2013).

Discussion

Our critical review demonstrated consistent evidence of associations between wildfire smoke exposure with general respiratory morbidity, and exacerbations of asthma and COPD (Table 1). Mounting epidemiological evidence and plausible toxicological mechanisms suggest an association between wildfire smoke exposure and respiratory infections, but inconsistencies remain. Increasing evidence suggests an association between wildfire smoke exposure and all-cause mortality, especially from more recent, higher-powered studies. The current evidence for cardiovascular morbidity from wildfire smoke exposure remains mixed; many studies are inconclusive or negative, but some have demonstrated significant increases for specific cardiovascular outcomes, such as cardiac arrests. Toxicological findings are consistent with cardiac effects through evidence of systemic inflammation and increased coagulability. Most of the other endpoints of interest, including birth outcomes, mental health, and cancer have not been sufficiently studied.

Our review highlights the lack of information about which populations are most susceptible to wildfire smoke exposure. People already diagnosed with asthma or COPD are more susceptible. We found inconsistent evidence of differential effects by age or SES. Two studies have suggested differential effects by Australian indigenous status with no investigation of other ethnic groups.

Many gaps exist in understanding the public health implications of exposure to wildfire smoke. Larger studies with greater statistical power and more spatially-refined exposure assessments are needed to better characterize impacts on mortality, cardiovascular disease, birth outcomes, and mental health effects. Currently, evidence exists of exacerbation, but not incidence, of asthma and COPD from wildfire smoke exposure. In temperate parts of the world, where wildfire smoke exposure is episodic, it is unlikely that changes in asthma incidence would be observed. Studies have not been conducted in populations more chronically exposed to wildfire smoke. Additionally, other health outcomes associated with wildfire smoke exposure have not yet been sufficiently studied, such as otitis media, which has been associated with exposure to secondhand tobacco smoke (Kong and Coates 2009), air pollution from woodsmoke (MacIntyre et al. 2011) and recently wildfire smoke (Yao et al. 2014). Human experimental studies of exposures to wildfire smoke could help clarify biological mechanisms. Very little information exists on health effects associated with measures of pollutants in wildfire smoke other than PM, such as ozone or PAHs. Although this review combined results from studies of various types of fires, it is possible that smoke originating from peat fires, forest fires, grassland fires, and agricultural burning could lead to differential health effects due to different constituents in the smoke. To our knowledge, no studies have yet investigated chronic exposure to wildfire smoke, but many populations in Southeast Asia, Africa, and Latin America are exposed regularly for extended periods (Johnston et al. 2012).

Characterization of the exposure-response function is critical for setting smoke levels for public health warnings or interventions, and it is not yet known whether current levels based on undifferentiated PM sufficiently characterize the effects of wildfire smoke. Four studies (Arbex et al. 2010; Chen et al. 2006; Johnston et al. 2002; Sastry 2002) have attempted to identify effects at different exposure levels, but these studies are hard to compare because of differences in exposure assessment methods, health outcomes, types of fires, and population susceptibilities.

Conclusions

We found consistent evidence of associations between wildfire smoke exposure and respiratory morbidity in general, and specifically for exacerbations of asthma and COPD. Growing evidence suggests associations with respiratory infections and all-cause mortality. More research is needed to determine whether wildfire smoke exposure is consistently associated with cardiovascular effects, specific causes of mortality, birth outcomes, and mental health outcomes. Research into which populations are most susceptible to health effects from wildfire smoke exposure is also needed to inform public health planning for future wildfires.

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Table

Table 1: Findings from epidemiological research studies (N=43) ordered by health outcome

Outcome	Article	Exposure Assessment Type	Direction of Association
Mortality, all	Sastry 2002	Monitored PM	↑↑
	Morgan et al. 2010	Monitored PM	↑↑
	Johnston et al. 2011	Smoky versus non-smoky days	↑↑
	Faustini et al. 2015	Smoky versus non-smoky days	↑↑
	Linares et al. 2014	Monitored PM	↑↑
	Shaposhnikov et al. 2014	Monitored PM	↑↑
Mortality, respiratory	Johnston et al. 2011	Smoky versus non-smoky days	↔
	Morgan et al. 2010	Monitored PM	↔
	Faustini et al. 2015	Smoky versus non-smoky days	↔
	Linares et al. 2014	Monitored PM	↔
Mortality, cardiovascular	Nunes et al. 2013	Modeled PM and satellite data	↑↑
	Faustini et al. 2015	Smoky versus non-smoky days	↑↑
	Johnston et al. 2011	Smoky versus non-smoky days	↑
	Morgan et al. 2010	Monitored PM	↔
	Linares et al. 2014	Monitored PM	↔

Respiratory morbidity			
Lung Function in people	Jacobson et al. 2012	Monitored PM	↓↓
without asthma or	Jacobson et al. 2014	Monitored PM	↓↓
bronchial hyperreactivity	Jalaludin et al. 2000	Monitored PM	↓↓
Physician Visits	Lee et al. 2009	Monitored PM	↑↑
	Henderson et al. 2011	Monitored PM Modeled PM Binary satellite indicator of smoke	↑↑ ↑ ↑
	Moore et al. 2006	Temporal comparison	↑↑
	Mott et al. 2002	Temporal comparison	↑↑
	Lee et al. 2009	Monitored PM	↑↑
ED visits	Rappold et al. 2011	Temporal and spatial comparisons	↑↑
	Tham et al. 2009	Monitored PM	↑↑
	Thelen et al. 2013	Modeled PM	↑↑
	Johnston et al. 2014	Smoky versus non-smoky days	↑↑
Hospitalizations	Morgan et al. 2010	Monitored PM	↑↑
	Henderson et al. 2011	Monitored PM Modeled PM Binary satellite indicator of smoke	↑↑ ↑ ↑
	Johnston et al. 2007	Monitored PM	↑
	Delfino et al. 2009	PM monitoring, statistical modeling, and satellite information	↑↑

	Martin et al. 2013	Smoky versus non-smoky days	↑↑
	Chen et al. 2006	PM monitoring for categorical exposures	↑↑
	Cancado et al. 2006	PM monitoring	↑↑
	Mott et al. 2005	Temporal comparison	↑↑
	Ignotti et al. 2010	% annual hours > 80 $\mu g/m^3$	↑↑
	Tham et al. 2009	Monitored PM	↔
Asthma			
Lung function among people with asthma	Jacobson et al. 2012	Monitored PM	↔
	Jalaludin et al. 2000	Monitored PM	↔
	Vora et al. 2011	Temporal comparison	↔
	Wiwatanadate & Liwsrisakun 2011	Monitored PM	↔
Medications	Elliott et al. 2013	PM monitoring, statistical modeling, and satellite information	↑↑
	Yao et al. 2014	Modeled PM	↑↑
	Tse et al. 2015	Temporal and spatial comparisons	↑↑
	Vora et al. 2011	Temporal comparison	↑↑
	Johnston et al. 2006	Monitored PM	↑↑
	Arbex et al. 2000	Measurement of PM	↑
Physician visits	Henderson et al. 2011	Monitored PM Modeled PM Binary satellite indicator	↑↑ ↑↑ ↑

	Yao et al. 2014	Monitored PM Modeled PM	↑↑ ↑↑
ED visits	Johnston et al. 2002	Monitored PM	↑↑
	Rappold et al. 2011	Temporal and spatial comparisons	↑↑
	Duclos et al. 1990	Temporal comparison	↑↑
	Johnston et al. 2014	Smoky versus non-smoky days	↑↑
	Smith et al. 1996	Temporal comparison	↑
	Tse et al. 2015	Temporal and spatial comparisons	↔
Hospitalizations	Morgan et al. 2010	Monitored PM	↑↑
	Delfino et al. 2009	PM monitoring, statistical modeling, and satellite information	↑↑
	Arbex et al. 2007	PM monitoring	↑↑
	Martin et al. 2013	Smoky versus non-smoky days	↑↑
	Johnston et al. 2007	Monitored PM	↑
	Tse et al. 2015	Temporal and spatial comparisons	↔
COPD			
Physician visits	Yao et al. 2014	Monitored PM Modeled PM	↑↑ ↑↑
ED visits	Rappold et al. 2011	Temporal and spatial comparisons	↑↑
	Duclos et al. 1990	Temporal comparison	↑↑
	Johnston et al. 2014	Smoky versus non-smoky days	↑↑
Hospitalizations	Morgan et al. 2010	Monitored PM	↑↑
	Johnston et al. 2007	Monitored PM	↑↑

	Delfino et al. 2009	PM monitoring, statistical modeling, and satellite information	↑↑
	Martin et al. 2013	Smoky versus non-smoky days	↑↑
	Mott et al. 2005	Temporal comparison[a]	↑↑
Respiratory Infections			
Physician visits	Yao et al. 2014	Monitored PM[b] Modeled PM[b] Monitored PM[c] Modeled PM[c]	↑↑ ↔ ↑↑ ↑↑
	Henderson et al. 2011	Monitored PM[d]	↔
ED visits	Duclos et al. 1990	Temporal comparison[b]	↑↑
	Rappold et al. 2011	Temporal and spatial comparisons[b]	↑
Hospitalizations	Johnston et al. 2007	Monitored PM	↔
Pneumonia and Bronchitis			
ED visits	Rappold et al. 2011	Temporal and spatial comparisons	↑↑
	Johnston et al. 2014	Smoky versus non-smoky days	↔
Hospitalizations	Delfino et al. 2009	PM monitoring, statistical modeling, and satellite information	↑↑
	Morgan et al. 2010	Monitored PM	↑↑
	Martin et al. 2013	Smoky versus non-smoky days	↑
	Duclos et al. 1990	Temporal comparison[e]	↑↑

Cardiovascular morbidity			
Physician visits	Henderson et al. 2011	Monitored PM Modeled PM Binary satellite indicator	↔ ↔ ↔
	Moore et al. 2006	Temporal comparison	↔
	Lee et al. 2009	Monitored PM	↔
	Yao et al. 2014	Monitored PM Modeled PM	↓↓ ↔
ED visits	Rappold et al. 2011	Temporal and spatial comparisons	↔
	Johnston et al. 2014	Smoky versus non-smoky days	↔
Hospitalizations	Morgan et al. 2010	Monitored PM	↔
	Hanigan et al. 2008	PM estimated from visibility data	↔
	Henderson et al. 2011	Monitored PM Modeled PM Binary satellite indicator	↔ ↔ ↔
	Johnston et al. 2007	Monitored PM	↔
	Martin et al. 2013	Smoky versus non-smoky days	↔
CHF			
ED visits	Rappold et al. 2011	Temporal and spatial comparisons	↑↑
Hospitalizations	Delfino et al. 2009	PM monitoring, statistical modeling, and satellite information	↑
	Morgan et al. 2010	Monitored PM	↔

	Martin et al. 2013	Smoky versus non-smoky days	↔
Cardiac Arrest			
Out-of-hospital	Dennekamp et al. 2015	PM monitoring	↑↑
	Haikerwal et al. 2015	Modeled PM	↑↑
ED visits	Johnston et al. 2014	Smoky versus non-smoky days	↔
Acute MI			
ED visits	Haikerwal et al. 2015	Modeled PM	↔
Hospitalizations	Haikerwal et al. 2015	Modeled PM	↑↑
IHD			
Physician visits	Lee et al. 2009	Monitored PM	↑↑
ED visits	Johnston et al. 2014	Smoky versus non-smoky days	↑
	Haikerwal et al. 2015	Modeled PM	↑
Hospitalizations	Mott et al. 2005	Temporal comparison	↑
	Haikerwal et al. 2015	Modeled PM	↑
	Morgan et al. 2010	Monitored PM	↔
	Delfino et al. 2009	PM monitoring, statistical modeling, and satellite information	↔
	Johnston et al. 2007	Monitored PM	↓↓ and ↑↑[f]
	Martin et al. 2013	Smoky versus non-smoky days	↔
Hypertension			
Physician visits	Henderson et al. 2011	Monitored PM	↔
Hospitalizations	Arbex et al. 2010	PM monitoring	↑↑

Cardiac Dysrhythmias/Arrhythmias			
ED visits	Johnston et al. 2014	Smoky versus non-smoky days	↔
Hospitalizations	Delfino et al. 2009	PM monitoring, statistical modeling, and satellite information	↔
	Martin et al. 2013	Smoky versus non-smoky days	↔
Cerebrovascular Disease			
ED visits	Johnston et al. 2014	Smoky versus non-smoky days	↔
Hospitalizations	Delfino et al. 2009	PM monitoring, statistical modeling, and satellite information	↑
	Morgan et al. 2010	Monitored PM	↔
Angina			
Dispensations of fast-acting nitroglycerin	Yao et al. 2014	Monitored PM	↑↑
ED visits	Haikerwal et al. 2015	Modeled PM	↑
Hospitalizations	Haikerwal et al. 2015	Modeled PM	↔
Birth outcomes			
Birth weight	Holstius et al. 2012	Temporal comparison	↓↓
Proportion of cohort surviving	Jayachandran 2009	Satellite data	↓↓
Low birth weight	Candido da Silva et al. 2014	Monitored PM	↑↑

Mental Health			
Physician visits	Moore et al. 2006	Temporal comparison	↔
Hospitalizations	Duclos et al. 1990	Temporal comparison	↔

[a] – asthma and COPD combined

[b] – upper respiratory infections.

[c] – lower respiratory infections

[d] - upper respiratory infections and acute bronchitis combined

[e] – bronchitis alone

[f] – significantly elevated for Indigenous population, but significantly lower risk for whole population

↔ no association

↑ suggestive increase

↑↑ significant increase

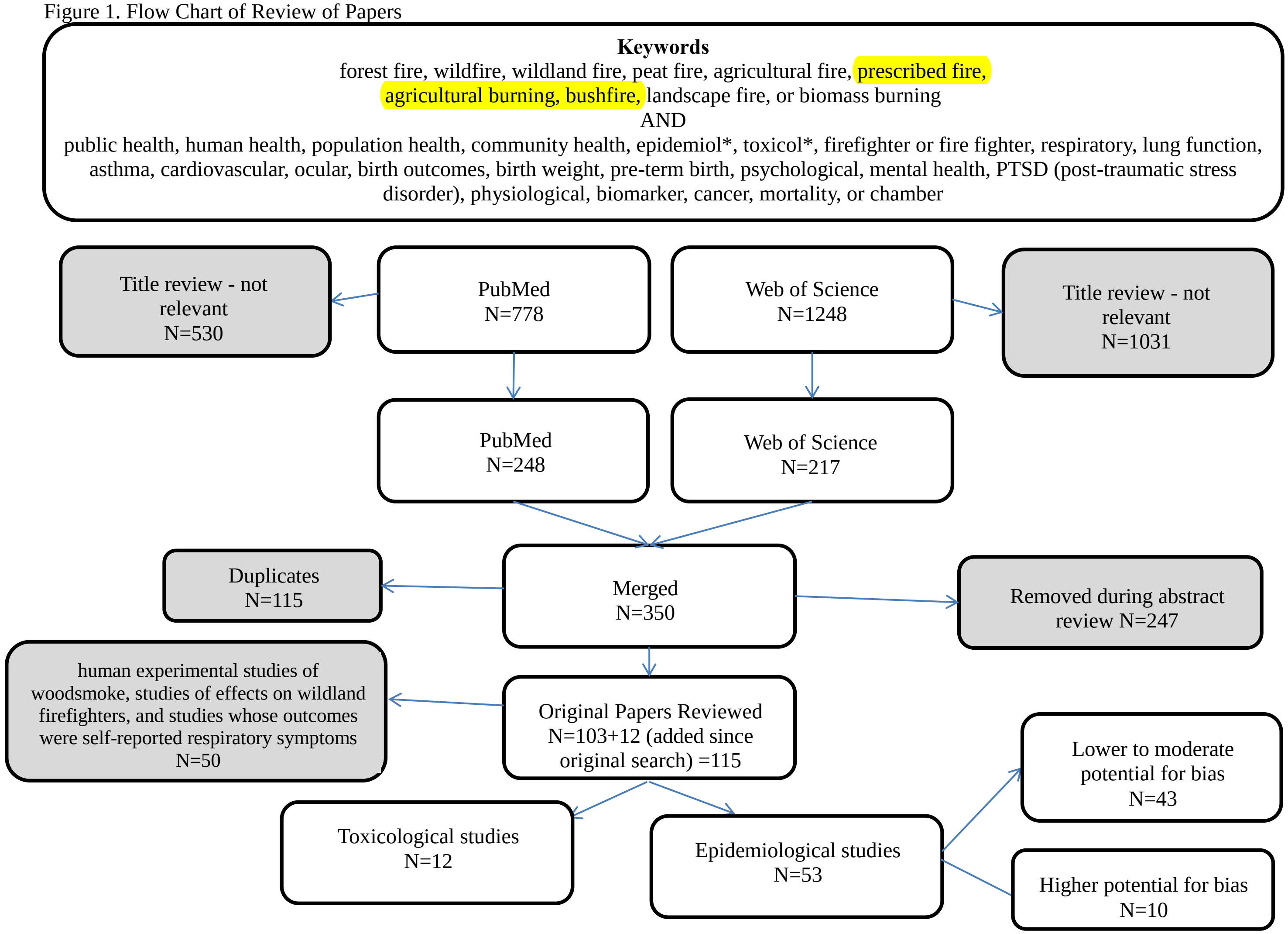
↓↓ significant decrease

Figure Legend

Figure 1. Review of Studies Flow Chart

Figure 1.

Figure 1. Flow Chart of Review of Papers



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Supplemental Material

Critical Review of Health Impacts of Wildfire Smoke Exposure

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Table S1: Assessment of Risk of Bias for all epidemiological studies reviewed (N=53)

Article	Fire Event/Location	Sample Size	Exposure Assessment Method	Exposure Levels	Covariates controlled for	Outcomes	Risk of Bias	Comment on Risk of Bias
Analitis et al. 2011	Athens 1998-2004	1071 days; Average number of deaths per day = 73 and	Categorized days with large fires (fires greater than 30,000,000 m^3), medium fires (1,000,001-30,000,000 m^3), and small fires (10,000 – 1,000,000 m^3), compared to days with no fires	No smoke levels reported	PM, temperature, heat wave day, RH, wind speed, wind direction, day of week, holidays and seasonal and long-term trend.	mortality, all-cause	higher	Exposure assessment method may not be related to smoke exposure; adjustment for black smoke may have attenuated impacts of wildfire-generated smoke
Arbex et al. 2000	Araraquara, Brazil, sugarcane burning season June 1-August 31, 1995	97 days with an average number of hospital visits for inhalation therapy of 22	Gravimetric analysis of particles centrifuged daily from water in receptacles placed at two sites in Araraquara.	12.9 ±7.0 mg sediment per day	Seasonality, temperature, day of week, precipitation	Hospital visits for inhalation therapy	moderate	Exposure assessment method is unique to this study and only yielded the largest particles, as noted by the authors
Arbex et al. 2007	Araraquara, Saõ Paulo State, Brazil, from 23 March 2003 to 27 July 2004	493 days and a total of 640 asthma hospitalizations	TSP from one monitor downtown	Mean TSP = 46.8 ± 26.4 $\mu g/m^3$	long-term trend, temperature, humidity	hospitalizations, asthma	lower	
Arbex et al. 2010	Araraquara, Brazil 23 March 2003 to 27 July 2004	493 days and mean of 2.5 hypertension –related hospital admissions per day	TSP from one monitor downtown	Burning period TSP mean 56.866 ± 25.07 $\mu g/m^3$	long-term trend, temp, RH	hospitalizations, hypertension	lower	
Azevedo et al. 2011	Portugal 2005	350 days	One central monitor	42 days in 2005 had ozone levels over 180 $\mu g/m^3$	Ozone, PM_{10}, SO_2, NO, CO, NO_2, $PM_{2.5}$	Respiratory and cardiovascular hospitalizations	higher	Models not adjusted for temporal trend, seasonality, day of week, or temperature effects. Multipollutant

								models without dealing with collinearity.
Caamano-Isorna et al. 2011	August 2006 Galician Fires	4212 municipality-months (156 municipalities *27 months); did not give average daily doses of each drug per 1000 inhabitants for these municipalities but did for all of Spain: 46.51 for anxiolytics, 22.19 for hypnotics, and 45 for drugs for obstructive airway disease	Number of wildfires within a municipality used to classified municipalities into no exposure (0-3 wildfires), medium exposure (4-10) and high exposure (more than 10).	No air quality exposure assessment	Interaction of exposure and time period, time trend, sex and age by stratification	drug dispensations for anxiolytics and for obstructive airway diseases	higher	Exposure assessment of number of fires in a region may not represent fire smoke exposure and no assessment of air quality
Cançado et al. 2006	Piracicaba in southeast Brazil. From April 1997 through March 1998	306 days; mean daily hospital admissions for children was 2.2 and for elderly was 0.9	PM_{10}, $PM_{2.5}$ and speciated PM information that was used in factor analysis to determine sources	Not reported for the biomass burning factor	long-term trend, day of week, temperature, RH	hospitalization, respiratory	lower	
Candido da Silva et al. 2014	Retrospective cohort of births in cities in Mato Grosso State, Brazil from July 1, 2004 and December 31, 2005	6147 full-term live births	$PM_{2.5}$ from one monitoring station	Average $PM_{2.5}$ levels in 2004 of 21.7 ± 35.2 $\mu g/m^3$ and in 2005 of 18.1 ± 33.7 $\mu g/m^3$	Sex, mother's education, prenatal visits, type of delivery, and age group	Low birth weight	lower	
Chen et al. 2006	July 1 1997 to December 31	1222 days with median of 33	PM_{10} from one of five monitoring sites	Mean daily PM_{10} = 16.11 $\mu g/m^3$, range =	Temperature, seasonality, day	hospitalization, respiratory	lower	

	2000, Brisbane Australia	patients per day admitted to hospital for respiratory disease		4.90 – 60.60	of week, long term trend, influenza			
Cooper et al. 1994	January 1994 Sydney fire (10 day event)	Data only shown in graphical form	hourly average scattering coefficient from a nephelometer used to distinguish before, during and after fires	Only shown in graphical form	None reported	acute asthma hospital presentations	higher	Periods compared had different days of week, did not control for temperature, and not enough information given on methods
Delfino et al. 2009	Southern California 2003	Unit of analysis is ZIP code-day. There were 45 days in the analysis, but does not state number of ZIP codes. Population covered was 20.5 million.	Zip code level $PM_{2.5}$ estimates from spatial interpolations from measured $PM_{2.5}$, light extinction, meteorological conditions and smoke information from MODIS satellite. Missing values were estimated from temporal profiles of continuous PM monitors at closely located sites or light extinction from visibility data, meteorological conditions and smoke info from MODIS. For nonfire periods, spatial interpolations using IDW, kriging or cokriging, but during fire polygons were created to represent the fire densities and $PM_{2.5}$ concentrations in each smoke-polygon were assigned.	During fires modeled mean $PM_{2.5}$ ranged from 42.1 to 76.1 $\mu g/m^3$	Temperature, relative humidity, pressure gradient, fungal spores (asthma only), income, age, race, gender, weekend, county	Hospitalizations for various respiratory and cardiovascular endpoints	lower	
Dennekamp et al. 2015	2006-2007 bushfire season Victoria, Australia	2046 out-of-hospital cardiac arrests	$PM_{2.5}$ from one monitoring station	IQR of $PM_{2.5}$ = 6.1 $\mu g/m^3$	Temperature, relative humidity, month, day of week, and hour of day.	out of hospital cardiac arrest	lower	
Duclos et al. 1990	August 1987, lightning fire in Northern	699 observed ER visits in 2.5 week fire period	temporal comparison of the fire period to two referent periods (one the previous month) and	Not reported	Seasonal and annual trends	emergency department visits and	moderate	Did not control for temperature or RH

	California		one in the previous year at the same time			hospitalizations for respiratory and mental health effects		
Elliott et al. 2013	British Columbia 2003-2010 during fire seasons (April1-September 30)	42456 LHA-days = (29 local health areas (LHAs) * 183 days per year * 8 years); average daily salbutamol dispensations ranged from 4.3 to 103.4 by LHA	$PM_{2.5}$ from one station per LHA, either the one nearest its centroid or its only one. For areas that didn't have $PM_{2.5}$ for the whole period, converted PM_{10} to $PM_{2.5}$ using regressions for the time period with both, or if no $PM_{2.5}$ then the regression from all of the other LHAs. Also dichotomized LHAs as fire affected by using MODIS fire pixels and chose the ones that were regularly impacted by fire.	Maximum concentrations of $PM_{2.5}$ in fire affected LHAs ranged 33.4 to 248.1 $\mu g/m^3$	Temperature, RH, year, month, and day of week	drug dispensations, salbutamol sulfate	lower	
Faustini et al. 2015	Ten cities in Spain, Italy, and Greece	20,087 study days across ten cities; daily mean natural deaths = 36	Smoky days versus non-smoky days classified from NAAP model (derived from AOD and fire plumes)	Smoky days PM_{10} ranged from 8-16 $\mu g/m^3$	Year, month, day of week, holidays, influenza, temperature, Saharan dust	Mortality	lower	
Haikerwal et al. 2015	2006-2007 wildfire episode in Victoria, Australia	457 out-of-hospital cardiac arrests; 2106 ED visits for IHD and 3274 hospital admissions for IHD	$PM_{2.5}$ modeled from a global chemical transport model dynamically downscaled using The Air Pollution Model	PM2.5 mean levels =15.43 $\mu g/m^3$ (IQR = 9.04 $\mu g/m^3$)	Time-stratified case control study controlled for day of week, seasons, time trends and individual covariates, also controlled for temperature and RH	Out-of hospital cardiac arrests, and hospitalizations and ED visits for IHD, acute MI, and angina	lower	
Hanigan et al. 2008	fire seasons, 1996-2005 Darwin, Australia	2410 days; 8279 hospital admissions	model of estimated exposure from visibility data	PM_{10} mean levels during fire period 21.2 ± 8.2 $\mu g/m^3$	RH, temperature, influenza, time trends, indigenous status, holidays	hospitalizations, cardiovascular	lower	

Henderson et al. 2011	July-September 2003 in British Columbia, fire season	281,711 people in cohort with 92 days of observation	(1) TEOM PM10 monitors, people assigned to nearest monitor to their postal address, (2) CALPUFF estimates of PM10 based on fire boundaries, and (3) binary smoke variable based on smoke boundaries from NOAAs fire detection tool: if there was a smoke plume over an area at any point during the day, it was considered exposed	PM_{10} mean levels 29.4 ± 30.7 µg/m^3	Temperature, day of week, week of study	Respiratory and cardiovascular hospitalizations and physician visits	lower	
Ho et al. 2014	2013 south Asian haze crisis	298 respondents	Self-report of perceived pollution standard index (PSI) as dangerous	Highest level pollution standard index of 401 on 0 to 500 scale.	None reported	Impact of Event Scale – Revised Survey, measure of psychological stress	higher	Self-report of exposure
Holstius et al. 2012	2003 Southern California Fires	886,034 births	temporal comparison of before, during and after fires	Not reported	Sex, gestational age, parity, maternal age, maternal education, maternal race, secular trend, season	birth weight	moderate	Not adjusted for maternal smoking
Ignotti et al. 2010	2004-2005 comparison of states in Brazilian Amazon	107 microregions	spatial comparison of % of annual hours with $PM_{2.5}$ > 80 µg/m^3	Assumed a threshold of 80 µg/m^3 based on Oregon standards	Human development index, a measure of education, earned income and longevity; and number of blood counts, an indicator of health service quality	hospitalization, respiratory	moderate	Did not control for meteorology/season and smoke prevalence
Jacobson et al. 2012	August to September 2006, Alta Floresta Brazil	309 children	$PM_{2.5}$ hourly measurements converted to 5-hour, 6-hour, 12-hour and 24-hour averages.	$PM_{2.5}$ mean levels = 24.34 ± 19.25 µg/m^3	age, height, weight, asthma status, passive smoking, use of	lung function	lower	

					medication, temperature, humidity, gender, occurrence of respiratory infections			
Jacobson et al. 2014	August to November 2008, Tangara da Serra, Brazil	234 children	PM_{10} and $PM_{2.5}$ and black carbon from one monitor at the school	PM_{10} mean levels = 62.7 ± 40.7 μg/m^3	Time trends, temperature, humidity	Lung function	lower	
Jalaludin et al. 2000	January 1994 Sydney	32 children for 31 days	PM_{10} from the monitor closest to each child's school	Not reported	Bushfire period, asthma medication usage, time trend, temperature, humidity, pollen counts, alternaria counts	lung function	moderate	Small sample size and duration
Jayachandran 2009	1997 Southeast Asian Fires	67,454 subdistrict-months; average size of birth cohort 95.6 per subdistrict-month	Aerosol index from the Total Ozone Mapping Spectrometer (TOMS) by month interpolated to each spatial subdistrict	Not reported	Subdistrict population, fixed effects for subdistrict and for month, median log of food consumption, rainfall, predicted fertility, fuel use, health facilities	Birth cohort size	moderate	Coarse resolution exposure metric that may not have represented ground-level concentrations well
Johnston et al. 2002	Darwin April 1-October 31 2000, a period of minimal rainfall and	214 days; 256 total asthma presentations	PM_{10} averaged from two monitoring stations	Range of $PM_{10\,=}$ 2.0 to 70 μg/m^3	Influenza, weekends	emergency department visits, asthma	moderate	Did not control for temperature

	almost continuous bushfire activity							
Johnston et al. 2006	seven month period in Darwin, Australia	251 people	$PM_{2.5}$ and PM_{10} from two monitors	PM_{10} mean levels 20 ±6.4 μg/m^3	Temperature, humidity, rainfall, pollen count, spore count, influenza rates, weekends, holidays, temporal autocorrelation	Asthma rescue medication usage; oral steroid medication usage	lower	
Johnston et al. 2007	Darwin, Australia three fire seasons, 2000, 2004, and 2005	2466 emergency admissions	PM_{10} from one monitoring station	Mean PM_{10} = 17.4 μg/$m^{3,}$ range (1.1 to 70)	Day of week, month, year, influenza, temperature, humidity, rainfall, holidays	hospitalizations, asthma	lower	
Johnston et al. 2011	Sydney 1997-2004	284,326 deaths	Categorized days (high smoke days compared to non-smoke days). Days were classified as 'extreme events' based on if the PM_{10} city-wide average from 7 monitoring stations exceeded the 99th percentile for the time series (47.3ug/m3) and the cause of each event was verified to determine days which were due to smoke	Smoke days PM_{10} ranged from 47.3 – 114.8 μg/m^3	Day of week, month, year, influenza, temperature, humidity	mortality	lower	
Johnston et al. 2014	Sydney 1996-2004	630,000 ED presentations for respiratory conditions; 370,000 ED presentations for cardiovascular	Categorized days (high smoke days compared to non-smoke days). Days were classified as 'extreme events' based on if the PM_{10} city-wide average from 7 monitoring stations exceeded the 99th percentile for the time series (47.3μg/m^3) and the cause	Mean PM_{10} on smoke-affected days was 60.5 μg/m^3	Day of week, month, year, influenza, temperature, dew point, holiday	ED visits for respiratory and cardiovascular endpoints	lower	

		conditions	of each event was verified to determine days which were due to smoke					
Lee et al. 2009	Hoopa Valley Indian Reservation Fire of 1999	1882 clinic visits	One PM_{10} monitor and a comparison to the previous year	Weekly average PM_{10} levels ranged from 12.8 to 363.8 $\mu g/m^3$	Residence location (in or near reservation) and # of clinic visits in previous year (both done by stratification), age, sex	Respiratory and physician visits, respiratory	moderate	Did not control for temperature or humidity
Linares et al. 2014	Madrid days with advection from biomass burning from 2004-2009	2192 days	Effect of PM_{10} on mortality on days with advection of biomass burning	Mean PM_{10} on days with advection was 44.2 $\mu g/m^3$	Ozone, temperature, trend, seasonality	Mortality	lower	
Marshall et al. 2007	2003 Southern California Fires	357 respondents	self-reported difficulty breathing because of smoke or ashes	Not reported	Age, gender, race/ethnicity, education, employment status, income	PTSD or depression three months after fires	higher	Retrospective self-report of exposure
Martin et al. 2013	top 99% of days from 1994-2007 in Sydney, Newcastle and Wollongong	3,141,017 non-trauma hospital admissions in Sydney, 273,034 in Wollongong, and 345,736 in Newcastle	Categorized days (high smoke days compared to non-smoke days). Days were classified as 'extreme events' based on if the PM_{10} city-wide average from 7 monitoring stations exceeded the 99th percentile for the time series (47.3ug/m3) and the cause of each event was verified to determine days which were due to smoke	High smoke days Sydney PM_{10} = 67.3 $\mu g/m^3$, range = (47.3 to 114.8)	Day, month, year, temperature, humidity, dew point, influenza, holidays	Respiratory and cardiovascular hospitalization	lower	
McDermott et al. 2005	2003 Canberra, Australia wildfires	222 children	self-reported "saw smoke"	Not reported	None reported	post-traumatic stress disorder reaction index score and Strengths& Difficulties Score (based on	higher	Retrospective self-report of exposure

						emotional problems, conduct problems, and hyperactivity)		
Moore et al. 2006	British Columbia 2003 fires	Studied weekly rates of respiratory physician visits for six weeks in one year compared to ten previous years in two small communities. Population of Kelowna = 146,199. Population of Kamloops = 100,548.	temporal comparison; determined fire affected time periods by PM monitoring at each of two sites (Kelowna and Kamloops)	Graphics appear to demonstrate effects when $PM_{2.5}$ > 50 μg/m^3 only for Kelowna and not Kamloops	LHA population, seasonality by temporal comparison	physician visits for respiratory, cardiovascular or mental health endpoints	moderate	Small sample size, did not control for temperature
Morgan et al. 2010	daily exposure in Sydney 1994-2002	3103 days; average daily all-cause mortality = 56	PM_{10} from 8 monitoring locations, Defined bushfire days as days with city-wide 24hour average PM_{10} greater than the 99th percentile for the study period and verified with newspaper archives and other sources (note that could be bushfires or "fuel-reduction burns") -- and estimated background PM_{10} on bushfire days as the 30-day moving average of PM_{10} when bushfire days are set to missing	Bushfire days range of PM_{10} = 43-117 μg/m^3	Background PM10, temperature, humidity, time trend, day of week, influenza	Respiratory or cardiovascular hospitalization	lower	
Mott et al. 2002	1999 fire near Hoopa Valley National Indian Res, Aug 23-Nov3	289 interviews	temporal comparison	Not reported in tables	Stratified by time period	physician visits, respiratory	moderate	Self-reported outcomes, not adjusted for temperature

Mott et al. 2005	1997 Southeast Asian Fires	Monthly time-series of 35 months used to predict for five months of fire and compare to observed	temporal comparison	Not reported	Stratified by time period	Respiratory and cardiovascular hospitalizations	moderate	Short time series; did not control for temperature effects
Nunes et al. 2013	Brazilian Amazon 2005	107 microareas in the Brazilian Amazon	annual % of hours of $PM_{2.5}$ over 25 μg/m^3	Range of annual % of hours with $PM_{2.5}$ > 25 μg/m^3 = 0.00 – 43.89	controlled for human development index, family health unit, number of intensive care unit beds	Circulatory disease mortality	moderate	potentially insufficient control of regional differences related to mortality such as smoking prevalence
Prass et al. 2012	2001-2005 in Porto Velho, Brazil	60 months	Number of hot spots detected by the NOAA-12 satellite by month	61,154 hot spots over 5 year time period; number by month ranged from 0 to 8,775	Sex, year, month, season	Birth weight	higher	Did not control for temperature or other seasonally varying factors that relate to birth weight; exposure measurement may not relate to smoke exposure
Rappold et al. 2011	2008 peat bog fire in North Carolina, June 1-July14, 2008, but 10-12 June were considered the high exposure period	42 counties (18 exposed); 44 days with three considered high exposure days;	Temporal and regional comparison; AOD to define exposed and unexposed counties, dichotomized to exposed if AOD >1.25 and then if >25% of county area was at AOD 1.25 or higher that day is exposed, but then a county was considered exposed if had 2 days in that exposure category; compared the high exposure days to non-exposure days for each county and then compared exposed to non-exposed counties	not reported	Day of week, stratified by age and sex. Although did not control for temperature, long-term trend or demographical differences between counties, authors note analyses that demonstrated that	Respiratory and cardiovascular ED visits	lower	

					confounding by these variables was not evident			
Resnick et al. 2015	2011 Wallow Fire Albuquerque, NM	Over all time periods there were 4525 cardiovascular ED visits and 4164 respiratory ED visits	Temporal comparison	Mean $PM_{2.5}$ during the fires=31.3 µg/m^3	None reported; stratified by sex and age and time period	Respiratory and cardiovascular ED visits	higher	Did not control for temperature, humidity, day of week, holidays or time trends
Sastry 2002	smoke from the 1997 fires of Indonesia in Malaysia, April-November 1997	52,742 deaths	PM_{10} for Kuala Lumpur for 1996-1997, used visibility data for other locations and other years	Mean daily PM_{10} = 64.2 ±43.0 µg/m^3. Range from 16.2 to 423.9 µg/m^3	Temperature, humidity, long-term trend, seasonality	mortality	lower	
Shaposhnikov et al. 2014	Moscow heat wave and wildfires, summer 2010	Time-series analysis from 2006-2010; Moscow averages about 300 deaths per day	City-average PM_{10}	Not reported	Long-term trend, seasonality, day of week, relative humidity, temperature as an interaction term	mortality	lower	
Smith et al. 1996	January 1994 western Sydney	Average daily asthma attendances at hospitals was 14.1 for control period and 10.7 for fire period	PM_{10} from three monitoring stations	Hourly PM_{10} ranged from 0.0 to 250.0 µg/m^3	Time period (controlled for year and season), temperature, humidity, wind speed, pressure, rainfaull, ozone, NO_2	emergency department visits, asthma	lower	
Tham et al. 2009	January to March 2003, Victoria, Australia	212 days; mean daily respiratory hospital	PM_{10} from one monitoring station in Melbourne, and two others in the Gippsland region of Victoria.	PM_{10} range of 0 to 289 µg/m^3	Day of week, time trend, temperature, humidity	Respiratory hospitalization and emergency department	lower	

		admissions = 48.43				visits		
Thelen et al. 2013	2007 San Diego, whole year including fire	121 days; mean daily ED visits=247.4	HYSPLIT air quality model was run with and without fire emissions estimates to get a way to quantify $PM_{2.5}$ just from wildfires.	Modeled $PM_{2.5}$ of wildfire origin range 0 to 403 $\mu g/m^3$, with corresponding range of RR of 1.0 to 1.41, but they do not give information to understand at what level of exposure the health effects become significant	Temperature, relative humidity, age groups, income categories, day of week	emergency department visits, respiratory	moderate	Did not control for long term trend or seasonality
Tse et al. 2015	Years before and after the 2003 and 2007 southern California wildfires	2195 asthmatic children for the 2003 fires and 2965 asthmatic children for the 2007 fires selected from an ongoing pediatric cohort	ZIP codes were classified as fire affected and not fire-affected, but the method for doing so was not explained in the paper	Not reported	Temporal trends accounted for in using data from a full year before and after	Physician-dispensed short-acting Beta agonists, physician-prescribed oral corticosteroids, ED visits and hospitalizations for asthma, newly diagnosed asthma	moderate	Method of classifying exposure was not made clear; no adjustment for other temporal changes that could affect asthma outcomes such as exposure to tobacco smoke, pollens, temperature
Vedal and Dutton 2006	2002 June Denver - two days, June 9 and June 18	Two days; daily average non-accidental mortality = 35.3	regional comparison	not reported	Investigates temperature and time but just descriptively, not statistically	mortality	higher	Very low power to detect an effect from just two days
Vora et al. 2011	San Diego 2007 5 day firestorm	8 subjects followed for 3 periods of four days	Temporal comparison	Mean morning $PM_{2.5}$ = 71.8 ± 24.5 $\mu g/m^3$	Time periods	lung function and # of rescue medication doses used	moderate	Small sample size and did not control for temperature or humidity or exposure to environmental tobacco smoke
Wiwatana date & Liwsrisak	Chiang Mai, Thailand, August 15,	121 asthmatic subjects followed for	Air quality monitor in city center	$PM_{2.5}$ ranged from 13.19 $\mu g/m^3$to 223.83 $\mu g/m^3$	gender, age, asthma severity, day of week,	Lung function	moderate	Multipollutant models that did not deal with

un 2011	2005 to June 30, 2006	306 days			weight, pressure, temperature, sunshine duration, rain quantity and random effects			collinearity; did not adjust for time trends or seasonality
Yao et al. 2014	British Columbia 2003-2010 fire seasons	89 local health areas; total population over 4 million, April through September for ten years	PM monitoring data for 29 local health areas; modeled PM2.5 from a combination of AOD from MODIS, sum of fire radiative power from MODIS hot spots, and hand drawn smoke plumes from the NOAA Hazard Mapping System for all 89 local health areas	Mean daily measured $PM_{2.5}$ was 5.9±5.2 $\mu g/m^3$; Mean daily measured $PM_{2.5}$ on extreme fire days was 10.2±11.1 $\mu g/m^3$	Temperature, temporal trends	Dispensations of salbutamol and nitroglycerin; physician visits for asthma, upper respiratory infections, lower respiratory infections, otitis media and all cardiovascular diseases	lower	

Table S2: Effect estimates for original epidemiological research studies (N=53), regardless of level of potential bias, ordered by health outcome and type of effect estimate.

Article	Outcome	Lag	Type of Effect Estimate	Effect Estimate	Comment
Mortality, all-cause					
Sastry 2002	mortality, all-cause	one day	RR per 10 µg/m^3 PM_{10}	1.19 (0.98 , 1.41)	
Morgan et al. 2010	mortality, all-cause	one day	RR per 10 µg/m^3 PM_{10}	1.01 (1.00 , 1.02)	derived from reported percent increase; only best lag is reported here
Johnston et al. 2011	mortality, all-cause	one day	OR high smoke versus non-smoke days	1.05 (1.00 , 1.10)	
Faustini et al. 2015	Mortality, natural	0-1 day	RR smoky versus non-smoky days	1.02 (0.99, 1.05)	Derived from reported percent increase
Linares et al. 2014	Mortality, natural	Lag 2	RR per 10 µg/m^3 PM_{10}	1.035 (1.011, 1.060)	
Shaposhnikov et al. 2014	Mortality, non-accidental	Lags 0-6 cumulative	RR per 10 µg/m^3 PM_{10} at different levels of temperature	1.004 (1.001 – 1.008) at T <18°C 1.008 (1.004 – 1.011) at T=22°C 1.014 (1.010 – 1.019) at T=>30°C	Derived from reported percent increase
Analitis et al. 2011	mortality, all-cause	same day	RR large fire versus no fire days	1.50 (1.37 , 1.63)	derived from reported percent increase
Mortality, respiratory					
Analitis et al. 2011	mortality, respiratory	same day	RR large fire versus no fire days	1.92 (1.48 , 2.50)	derived from reported percent increase
Johnston et al. 2011	mortality, respiratory	one day lag	OR high smoke versus non-smoke days	1.09 (0.88 , 1.36)	

Morgan et al. 2010	mortality, respiratory	same day	RR per 10 μg/m^3 PM_{10}	1.00 (0.97 , 1.04)	derived from reported percent increase; only best lag is reported here
Faustini et al. 2015	Mortality, respiratory	0-5	RR smoky versus non-smoky days	0.97 (0.90, 1.03)	Derived from reported percent increase
Linares et al. 2014	Mortality, respiratory	Lag 2	RR per 10 μg/m^3 PM_{10}	No effect reported because it was not statistically significant	
Mortality, cardiovascular					
Analitis et al. 2011	mortality, cardiovascular	same day	RR large fire versus no fire days	1.61 (1.43 , 1.80)	derived from reported percent increase
Johnston et al. 2011	mortality, cardiovascular	one day lag	OR high smoke versus non-smoke days	1.07 (0.98 , 1.18)	
Morgan et al. 2010	mortality, cardiovascular	same day	RR per 10 μg/m^3 PM_{10}	1.01 (0.99 , 1.02)	derived from reported percent increase; only best lag is reported here
Nunes et al. 2013	Mortality, cardiovascular in people 65 years of age and older	NA (cross-sectional comparison)	RR for one unit increase in annual percentage of hours greater than 25 μg/m^3 $PM_{2.5}$	1.01 (p-value reported as 0.035)	Derived from adjusted beta coefficient from multiple linear regression
Faustini et al. 2015	Mortality, circulatory	0-5	RR smoky versus non-smoky days	1.06 (1.10, 1.12)	Derived from reported percent increase
Linares et al. 2014	Mortality, circulatory	Lag 2	RR per 10 μg/m^3 PM_{10}	No effect reported for PM_{10} because it was not statistically significant	
Lung function					
Jacobson et al. 2012	lung function	same day	change in peak expiratory flow (liters/minute) for non-asthmatics associated with $PM_{2.5}$	-0.38 (-0.62 , -0.14)	

Jacobson et al. 2014	Lung function	Lag 3	change in peak expiratory flow (liters/minute) for all children regardless of asthma status with PM_{10}	-0.25 (-0.40, -0.10)	Presented results for all children, but effects were strongest among youngest. Investigated many lags, only presented one here.
Jalaludin et al. 2000	lung function	same day	change in peak expiratory flow rate - children without bronchial hyper-reactivity	-1.03 (-1.95 , -0.11)	calculated from beta and SE - assumed linear model per unit change in PM_{10} based on what was presented in the paper
Respiratory morbidity, all					
Lee et al. 2009	physician visits, respiratory		OR per 10 $\mu g/m^3$ PM_{10}	1.77 (1.51 , 2.09)	this RR is for a unit change in the log of PM_{10}
Henderson et al. 2011	physician visits, respiratory	same day	OR per 10 $\mu g/m^3$ PM_{10}	1.02 (1.01 , 1.03)	presented results are associated with monitored values of PM. Similar results were found using modeled and remotely sensed estimates of smoke exposure.
Moore et al. 2006	physician visits, respiratory		observed compared to 10-year mean	46-78% increase over 10-year mean rates	
Mott et al. 2002	physician visits, respiratory		percent increase in fire year compared to percent increase in non-fire year	11.9% (10.4-13.4) increase in fire year and 8.9% (7.5-10.3) expected from previous year in September, 19.2%(17.2-21.3)in fire year compared to 10.7% (9.1-12.3) increase in previous year	
Lee et al. 2009	physician visits, all respiratory		OR per 10 $\mu g/m^3$ PM_{10}	1.36 (1.24 , 1.50)	this RR is for a unit change in the log of PM_{10}

Rappold et al. 2011	emergency department visits, respiratory	lag0-5 cumulative	RR comparing fire period to reference period	1.66 (1.38 , 1.99)	results presented here are for smoke-affected counties only
Tham et al. 2009	emergency department visits, respiratory	same day	RR per 10 µg/m^3 PM_{10}	1.01 (1.00 , 1.02)	*calculated from 25th-75th range to 10 µg/ m^3
Thelen et al. 2013	emergency department visits, respiratory	cumulative lag exposure kernel centered at same day and with SD of 1 day	OR per 10 µg/m^3 wildfire PM	1.00 (1.00 , 1.01)	original estimates were per unit µg/m^3
Johnston et al. 2014	ED visits, respiratory	Lag 0	OR comparing smoke days to non-smoke days	1.07 (1.04, 1.10)	
Resnick et al. 2015	ED visits, respiratory	NA	RR comparing fire period to pre-fire period	0.83 (0.77, 0.90)	
Tham et al. 2009	hospitalization, respiratory	same day	RR per 10 µg/m^3 PM_{10}	1.00 (0.99 , 1.01)	calculated from 25th-75th range to 10 µg/m^3
Morgan et al. 2010	hospitalization, respiratory	same day	RR per 10 µg/m^3 PM_{10}	1.01 (1.00 , 1.02)	derived from reported percent increase; only best lag is reported here
Henderson et al. 2011	hospitalization, respiratory	same day	OR per 10 µg/m^3 PM_{10}	1.05 (1.00, 1.10)	*only presenting here results associated with monitored values of PM. Similar results were found using modeled and remotely sensed

					estimates of smoke exposure.
Johnston et al. 2007	hospitalization, respiratory	same day	OR per 10 μg/m^3 PM_{10}	1.08 (0.98 , 1.18)	for whole population
Delfino et al. 2009	hospitalization, respiratory	2-day moving average	RR per 10 μg/m^3 $PM_{2.5}$	1.03 (1.01 , 1.04)	This estimate is for the fire period; paper includes estimates for pre-fire and post-fire periods also
Martin et al. 2013	hospitalization, respiratory	same day	OR for high smoke days compared to non-smoke days	1.05 (1.02 , 1.09)	here only reporting the best lag result for Sydney, not other cities
Chen et al. 2006	hospitalization, respiratory	same day	RR comparing highest exposure category (>20 μg/m^3) against the lowest category (<15 μg/m^3), for the bushfire period	1.19 (1.09 , 1.30)	comparing highest exposure category (>20 μg/m^3) against the lowest category (<15 μg/m^3), for the bushfire period
Cancado et al. 2006	hospitalization, respiratory		RR for biomass burning factor from factor analysis	1.52 (1.12, 2.04)	for elderly only; calculated from effect estimate and SE non-exponentiated
Mott et al. 2005	hospitalization, respiratory	NA	observed compared to CI of expected	184 observed and 89.3-174.0 expected	all ages
Ignotti et al. 2010	hospitalization, respiratory		increase in respiratory hospitalizations associated with % annual hours > 80 μg/m^3	0.052 increase (p-value=0.017)	ecological analysis only
Asthma, exacerbations					

Jacobson et al. 2012	lung function	same day	change in peak expiratory flow for asthmatics	-0.18 (-0.66 , 0.31)	
Jalaludin et al. 2000	lung function	same day	change in peak expiratory flow rate - all children	-0.09 (-1.17 , 0.98)	calculated from beta and SE - assumed linear model per unit change in PM_{10} based on what was presented in the paper
Vora et al. 2011	lung function		difference between fires and non-fires	p-values ranged from 0.35 to 0.80 for different lung function metrics	only p-values reported
Wiwatandate & Liwsrisakun 2011	lung function	lag 6	change in peak expiratory flow rate among asthmatic people over age 12	-0.01 (-0.01, 0.00)	Lag 5 was also significant for PM_{10}
Elliott et al. 2013	drug dispensations, salbutamol sulfate	Same day	RR per 10 $\mu g/m^3$ $PM_{2.5}$	1.06 (1.04 , 1.07)	*these dispensations are for both asthma and COPD, but are placed in the asthma section of this table
Yao et al. 2014	drug dispensations, salbutamol sulfate	Mean of same day and previous day	RR per 10 $\mu g/m^3$ $PM_{2.5}$	1.04 (1.03 – 1.06)	Estimate from modeled $PM_{2.5}$; similar results for modeled $PM_{2.5}$
Tse et al. 2015	Physician-dispensed Beta-agonists	NA	Compared total for year after fires to year before fires	$p < 0.05$	
Tse et al. 2015	Physician-prescribed oral coriticosteroids	NA	Compared total for year after fires to year before fires	$p >= 0.05$	
Arbex et al. 2000	Hospital visits for inhalation therapy	Moving average of days 1-5	RR per 10 mg sediment weight	1.09 (1.00 – 1.19)	

Caamano-Isorna et al. 2011	drug dispensations for obstructive airway diseases		high exposure regions post-fire compared to no exposure regions pre-fire	1.18 (1.01, 1.35)	calculated from percent increase; presenting only results for male pensioners, also sig increase for women pensioners; *these dispensations are for both asthma and COPD, but are placed in the asthma section of this table
Vora et al. 2011	# of rescue medication doses used		only significance values presented for difference between fires and non-fires	p=0.03	
Johnston et al. 2006	rescue medication usage	one day	OR per 10 $\mu g/m^3$ PM_{10}	1.01 (0.99, 1.04)	
Johnston et al. 2006	oral steroid medication usage	one day	OR per 10 $\mu g/m^3$ PM_{10}	1.54 (1.01, 2.34)	
Henderson et al. 2011	physician visits, asthma	same day	OR per 10 $\mu g/m^3$ PM_{10}	1.06 (1.03 , 1.11)	*only presenting here results associated with monitored values of PM. Similar results were found using modeled and remotely sensed estimates of smoke exposure.
Yao et al. 2014	physician visits, asthma	Mean of same day and previous day	RR per 10 $\mu g/m^3$ $PM_{2.5}$	1.06 (1.04 – 1.08)	Estimate from modeled $PM_{2.5}$; similar results for modeled $PM_{2.5}$

Johnston et al. 2002	emergency department visits, asthma	Same day	RR per 10 μg/m^3 PM_{10}	1.20 (1.09 , 1.34)	
Rappold et al. 2011	emergency department visits, asthma	Lag 0-5 cumulative	RR comparing fire period to reference period	1.65 (1.25 , 2.17)	results presented here are for smoke-affected counties only; see paper for comparison to non-smoke affected counties
Duclos et al. 1990	emergency department visits, asthma	NA	observed/expected	1.4 (p-value<0.001)	
Smith et al. 1996	emergency department visits, asthma		difference in difference calculation	0.0067 (-0.0007, 0.0141)	temporal comparison of week of fire to same week a year before - presented difference in proportion of all visits that were for asthma for fire weeks compared to previous year minus the same difference for weeks surrounding the fire of both years and found no significant effect
Johnston et al. 2014	ED visits, asthma	Lag 0	OR comparing smoke days to non-smoke days	1.23 (1.15, 1.30)	
Resnick et al. 2015	ED visits, asthma	NA	RR comparing fire period to pre-fire period	1.73 (1.03-2.77)	this estimate is for ages 65+, non-significant findings for other ages; also found higher effects on women than men

					for asthma
Tse et al. 2015	ED visits, asthma among children with asthma	NA	Compared total for year after fires to year before fires	$p >= 0.05$	
Morgan et al. 2010	hospitalizations, asthma	same day	RR per 10 μg/m^3 PM_{10}	1.05 (1.02 , 1.08)	15-64 year-olds; derived from reported percent increase; only best lag is reported here
Johnston et al. 2007	hospitalizations, asthma	same day	OR per 10 μg/m^3 PM_{10}	1.14 (0.90 , 1.44)	for whole population
Delfino et al. 2009	hospitalizations, asthma	2-day moving average	RR per 10 μg/m^3 $PM_{2.5}$	1.05 (1.02 , 1.08)	This estimate is for the fire period; paper includes estimates for pre-fire and post-fire periods also
Arbex et al. 2007	hospitalizations, asthma	5-day moving average	RR per 10 units of TSP	1.12 (1.05 , 1.18)	calculated from percentage increase
Martin et al. 2013	hospitalizations, asthma	same day	OR for high smoke days compared to non-smoke days	1.12 (1.05 , 1.19)	here only reporting the best lag result for Sydney, not other cities
Tse et al. 2015	hospitalizations, asthma among children with asthma	NA	Compared total for year after fires to year before fires	$p >= 0.05$	
Asthma, new diagnoses					
Tse et al. 2015	newly diagnosed asthma	NA	Compared total for year after fires to year before fires	Decline in new asthma diagnoses post-fire ($p < 0.05$)	

Chronic obstructive pulmonary disease (exacerbations)					
Yao et al. 2014	physician visits, COPD	Mean of same day and previous day	RR per 10 µg/m^3 $PM_{2.5}$	1.02 (1.00 – 1.03)	Estimate from modeled $PM_{2.5}$; similar results for modeled $PM_{2.5}$
Rappold et al. 2011	emergency department visits, COPD	Lag 0-5 cumulative	RR comparing fire period to reference period	1.73 (1.06 , 2.83)	results presented here are for smoke-affected counties only; see paper for comparison to non-smoke affected counties
Duclos et al. 1990	emergency department visits, COPD	NA	observed/expected	1.3 (p-value =0.02)	
Johnston et al. 2014	ED visits, COPD	Lag 0	OR comparing smoke days to non-smoke days	1.12 (1.02, 1.24)	
Morgan et al. 2010	hospitalizations, COPD	lag 2	RR per 10 µg/m^3 PM_{10}	1.04 (1.01 , 1.06)	Only analyzed COPD for 65+; similar findings for lags 0 through 3, but presented largest finding here at lag 2; derived from reported percent increase; only best lag is reported here
Johnston et al. 2007	hospitalizations, COPD	same day	OR per 10 µg/m^3 PM_{10}	1.21 (1.00 , 1.47)	for whole population; 1.98 (1.10,3.59) for Indigenous
Delfino et al. 2009	hospitalizations, COPD	2-day moving average	RR per 10 µg/m^3 $PM_{2.5}$	1.04 (1.00 , 1.08)	Ages 20-99; This estimate is for the fire period; paper includes estimates for pre-fire and post-fire

					periods also
Martin et al. 2013	hospitalizations, COPD	same day	OR for high smoke days compared to non-smoke days	1.13 (1.05 , 1.22)	here only reporting the best lag result for Sydney, not other cities
Mott et al. 2005	hospitalizations, COPD	NA	observed compared to CI of expected	255 observed, 152.4-250.2 expected	all ages
Respiratory infections					
Henderson et al. 2011	Physician visits, acute upper respiratory infections	same day	OR per 10 μg/m^3 PM_{10}	0.99 (0.47 , 1.98)	Calculated from effect found for 30 unit change in PM_{10}; *only presenting here results associated with monitored values of PM. Similar results were found using modeled and remotely sensed estimates of smoke exposure.
Yao et al. 2014	physician visits, upper respiratory infections	Mean of same day and previous day	RR per 10 μg/m^3 $PM_{2.5}$	1.03 (1.02 – 1.05)	Estimate from measured $PM_{2.5}$; results from modeled $PM_{2.5}$ was null and not reported in tabular form.
Yao et al. 2014	physician visits, lower respiratory infections	Mean of same day and previous day	RR per 10 μg/m^3 $PM_{2.5}$	1.03 (1.00 – 1.05)	Estimate from modeled $PM_{2.5}$; similar results for modeled $PM_{2.5}$
Rappold et al. 2011	emergency department visits, upper respiratory infections	Lag 0-5 cumulative	RR comparing fire period to reference period	1.68 (0.94 , 3.00)	results presented here are for smoke-affected counties only; see paper for

					comparison to non-smoke affected counties
Duclos et al. 1990	hospitalizations, upper respiratory infections	NA	observed/expected	1.5 (p-value<0.001)	
Johnston et al. 2007	hospitalizations, upper respiratory infections		OR per 10 µg/m^3 PM_{10}	Effect Estimate not reported.	
Pneumonia and bronchitis					
Rappold et al. 2011	ED visits for pneumonia and acute bronchitis	Lag 0-5 cumulative	RR comparing fire period to reference period	1.59 (1.07 , 2.34)	results presented here are for smoke-affected counties only; see paper for comparison to non-smoke affected counties
Johnston et al. 2014	ED visits, pneumonia and bronchitis	Lag 0	OR comparing smoke days to non-smoke days	1.02 (0.95, 1.10)	
Delfino et al. 2009	hospitalizations for acute bronchitis and bronchiolitis	2-day moving average	RR per 10 µg/m^3 $PM_{2.5}$	1.10 (1.02 , 1.18)	Acute bronchitis and bronchiolitis; This estimate is for the fire period; paper includes estimates for pre-fire and post-fire periods also
Delfino et al. 2009	hospitalizations for pneumonia	2-day moving average	RR per 10 µg/m^3 $PM_{2.5}$	1.03 (1.01, 1.05)	Pneumonia; This estimate is for the fire period; paper includes estimates for pre-fire and post-fire periods also

Morgan et al. 2010	hospitalizations for pneumonia and acute bronchitis	lag 1	RR per 10 µg/m^3 PM_{10}	1.03 (1.02 , 1.06)	pneumonia and acute bronchitis for 65+ attributable to bushfire days; derived from reported percent increase; only best lag is reported here
Martin et al. 2013	hospitalizations for pneumonia and acute bronchitis	lag 2	OR for high smoke days compared to non-smoke days	1.26 (1.03, 1.55)	best lag for Newcastle; non-significant findings for Sydney and Wollongong
Duclos et al. 1990	hospitalizations for bronchitis	NA	observed/expected	1.2 (p-value = 0.03)	bronchitis
Duclos et al. 1990	hospitalizations for pneumonia	NA	observed/expected	1.0 (p-value = 0.4)	pneumonia
Cardiovascular disease, all					
Yao et al. 2014	Dispensations of fast-acting nitroglycerin for angina	Mean of same day and previous day	RR per 10 µg/m^3 $PM_{2.5}$	1.03 (1.01 – 1.05)	Effect for extreme fire days; RR was null for all days
Henderson et al. 2011	physician visits, cardiovascular	same day	OR per 10 µg/m^3 PM_{10}	1.00 (0.99 , 1.01)	*only presenting here results associated with monitored values of PM. Similar results were found using modeled and remotely sensed estimates of smoke exposure.
Moore et al. 2006	physician visits, cardiovascular			data not shown	

Lee et al. 2009	physician visits, all circulatory illness		OR per 10 µg/m^3 PM_{10}	1.13 (0.94 , 1.37)	this RR is for a unit change in the log of PM_{10}
Yao et al. 2014	physician visits, cardiovascular	Mean of same day and previous day	RR per 10 µg/m^3 $PM_{2.5}$	Null; data only shown graphically	
Rappold et al. 2011	emergency department visits, cardiovascular	Lag 0-5 cumulative	RR comparing fire period to reference period	1.13 (0.95 , 1.35)	results presented here are for smoke-affected counties only; see paper for comparison to non-smoke affected counties
Johnston et al. 2014	ED visits, COPD	Lag 0	OR comparing smoke days to non-smoke days	1.00 (0.96, 1.04)	
Morgan et al. 2010	hospitalizations, cardiovascular	lag 2	RR per 10 µg/m^3 PM_{10}	1.01 (0.99 , 1.01)	derived from reported percent increase; only best lag is reported here
Hanigan et al. 2008	hospitalizations, cardiovascular	same day	RR per 10 µg/m^3 PM_{10}	0.97 (0.91 , 1.02)	
Henderson et al. 2011	hospitalizations, cardiovascular	same day	OR per 10 µg/m^3 PM_{10}	1.00 (0.96 , 1.05)	*only presenting here results associated with monitored values of PM. Similar results were found using modeled and remotely sensed estimates of smoke exposure.
Johnston et al. 2007	hospitalizations, cardiovascular		OR per 10 µg/m^3 PM_{10}	data not shown	

Martin et al. 2013	hospitalizations, cardiovascular		OR for high smoke days compared to non-smoke days	data not shown	
Resnick et al. 2015	ED visits, all cardiovascular	NA	RR comparing fire period to pre-fire period	1.08 (1.00, 1.16)	
Congestive Heart Failure					
Rappold et al. 2011	emergency department visits, congestive heart failure	Lag 0-5 cumulative	RR comparing fire period to reference period	1.37 (1.01 , 1.85)	results presented here are for smoke-affected counties only; see paper for comparison to non-smoke affected counties
Morgan et al. 2010	hospitalizations, congestive heart failure	lag 2	RR per 10 µg/m^3 PM_{10}	1.00 (0.99 , 1.01)	derived from reported percent increase; only best lag is reported here
Delfino et al. 2009	hospitalizations, congestive heart failure	2-day moving average	RR per 10 µg/m^3 $PM_{2.5}$	1.02 (0.99 , 1.04)	This estimate is for the fire period; paper includes estimates for pre-fire and post-fire periods also
Martin et al. 2013	hospitalizations, congestive heart failure	lag 3	OR for high smoke days compared to non-smoke days	1.05 (0.96 , 1.14)	here only reporting the best lag result for Sydney, not other cities
Cardiac Failure					
Dennekamp et al. 2015	out of hospital cardiac arrest	48-hour	OR per 10 µg/m^3 $PM_{2.5}$	1.04 (1.00 , 1.08)	OR derived from reported percent increase in IQR $PM_{2.5}$
Johnston et al. 2014	ED visits, Cardiac failure	Lag 0	OR comparing smoke days to non-smoke days	1.05 (0.95, 1.17)	
Ischemic heart disease					
Johnston et al. 2014	ED visits, Ischemic heart disease	Lag 2	OR comparing smoke days to non-smoke days	1.07 (1.00, 1.15)	Non-significant at other lags (0,1, and 3 days)

Morgan et al. 2010	hospitalizations, ischemic heart disease	same day	RR per 10 μg/m^3 PM_{10}	1.00 (0.99 , 1.02)	derived from reported percent increase; only best lag is reported here
Delfino et al. 2009	hospitalizations, ischemic heart disease	2-day moving average	RR per 10 μg/m^3 $PM_{2.5}$	1.01 (0.99 , 1.02)	This estimate is for the fire period; paper includes estimates for pre-fire and post-fire periods also
Johnston et al. 2007	hospitalizations, ischemic heart disease	same day	OR per 10 μg/m^3 PM_{10}	0.82 (0.68 , 0.98)	for whole population; 1.71 (1.14,2.55) for Indigenous population
Martin et al. 2013	hospitalizations, ischemic heart disease	lag 2	OR for high smoke days compared to non-smoke days	1.03 (0.98 , 1.08)	here only reporting the best lag result for Sydney, not other cities
Mott et al. 2005	hospitalizations, ischemic heart disease	NA	observed compared to CI of expected	109 observed when 51.5-91.5 expected	results for ages 19-39 only significant age group
Lee et al. 2009	physician visits, coronary artery disease		OR per 10 μg/m^3 PM_{10}	1.48 (1.11 , 1.97)	this RR is for a unit change in the log of PM_{10}
Resnick et al. 2015	ED visits, ischemic heart disease	NA	RR comparing fire period to pre-fire period	1.17 (0.89, 1.55)	
Hypertension					
Henderson et al. 2011	physician visits, hypertension	same day	OR per 10μg/m^3 PM_{10}	1.00 (0.98 , 1.01)	Calculated from effect found for 30 unit change in PM_{10}; *only presenting here results associated with monitored values of PM. Similar results were found using modeled and remotely sensed estimates of smoke exposure.

Arbex et al. 2010	hospitalizations, hypertension	3-day moving average	RR per 10 μg/m^3 TSP	1.13 (1.06 , 1.20)	burning season estimate was 30% higher than non-burning season; calculated from percent increase
Resnick et al. 2015	ED visits, hypertensive disease	NA	RR comparing fire period to pre-fire period	1.08 (0.97, 1.20)	
Cardiac dysrhythmias					
Johnston et al. 2014	ED visits, arrhythmias	Lag 0	OR comparing smoke days to non-smoke days	0.97 (0.89, 1.06)	
Delfino et al. 2009	hospitalizations, dysrhythmias	2-day moving average	RR per 10 μg/m^3 $PM_{2.5}$	0.99 (0.96 , 1.02)	This estimate is for the fire period; paper includes estimates for pre-fire and post-fire periods also
Martin et al. 2013	hospitalizations, arrhythmia	lag 2	OR for high smoke days compared to non-smoke days	0.96 (0.88 , 1.04)	here only reporting the best lag result for Sydney, not other cities
Cerebrovascular disease					
Johnston et al. 2014	ED visits, cerebrovascular disease	Lag 0	OR comparing smoke days to non-smoke days	0.99 (0.91, 1.08)	
Resnick et al. 2015	ED visits, cerebrovascular disease	NA	RR comparing fire period to pre-fire period	1.69 (1.03, 2.77)	This estimate is just for ages 20-64; non-significant findings for 65+ and for 0-19
Delfino et al. 2009	hospitalizations, cerebrovascular disease and stroke	2-day moving average	RR per 10 μg/m^3 $PM_{2.5}$	1.02 (1.00 , 1.04)	This estimate is for the fire period; paper includes estimates for pre-fire and post-fire periods also

Morgan et al. 2010	hospitalizations, stroke	lag 2	RR per 10 µg/m^3 PM_{10}	1.01 (0.99 , 1.03)	just stroke, converted from percentage increase
Birth outcomes					
Holstius et al. 2012	birth weight	NA	decline in birth weight associated with gestation during fires compared to gestation not during fires	7.0 g lower [95% confidence interval (CI): –11.8, –2.2]	only presenting results for full pregnancy, not divided by trimester
Breton et al. 2011	birth weight	NA		not yet published	these findings have not yet been published, therefore we cannot publish the estimates
Jayachandran 2009	cohort size	NA	proportion of cohort surviving compared to normal cohort due to exposure to fire smoke during last three months of pregnancy	0.97 (0.94, 0.99)	calculated from log effect estimate and SE
Candido da Silva et al. 2014	Low birth weight	NA	OR of low birth weight associated with PM2.5 during second and third trimester for highest exposed quartile compared to lowest exposed quartile	1.51 (1.04, 2.17)	*Only presented second trimester results
Prass et al. 2012	Birth weight	NA	effect of monthly number of satellite detected hot spots on mean monthly birth weight in boys	-0.004485, p-value = 0.0431	Did not find an effect of monthly hot spots on monthly birth weight for girls
Mental Health					
McDermott et al. 2005	post-traumatic stress disorder reaction index score	NA	t-test for comparing scores for those who reported seeing smoke to those who reported not seeing smoke	t=1.63, p=0.11	p-value calculated from reported t-test and degrees of freedom

McDermott et al. 2005	Strengths& Difficulties Score (based on emotional problems, conduct problems, and hyperactivity)	NA	t-test for comparing scores for those who reported seeing smoke to those who reported not seeing smoke	t=3.76, p=0.0003	p-value calculated from reported t-test and degrees of freedom
Marshall et al. 2007	PTSD or depression three months after fires	NA	OR for those who reported difficulty breathing because of fires compared to those who did not	2.09 (1.10, 3.98)	
Caamano-Isorna et al. 2011	drug dispensations for anxiolytics	NA	high exposure regions post-fire compared to no exposure regions pre-fire	1.21 (1.10, 1.33)	calculated from percent increase; presenting only results for male pensioners, also sig increase for male non-pensioners; only significant for medium exposure regions compared to non-exposed
Moore et al. 2006	physician visits, mental illness	NA		data not shown	
Duclos et al. 1990	hospitalizations, mental health	NA	observed/expected	1.1 (p-value=0.4)	
Ho et al. 2014	Impact of Event Scale – Revised Survey, measure of psychological stress	NA	Chi-squared	Those who perceived lower PSI values as dangerous were more likely to have higher IES-R stress values (p = 0.047)	

*effect estimates for symptoms are not included in this table because of their varied nature.

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