Science of the Total Environment 781 (2021) 146739

Contents lists available at ScienceDirect

Science of the Total Environment

journal homepage: www.elsevier.com/locate/scitotenv

Review

Short-term health effects from outdoor exposure to biomass burning emissions: A review

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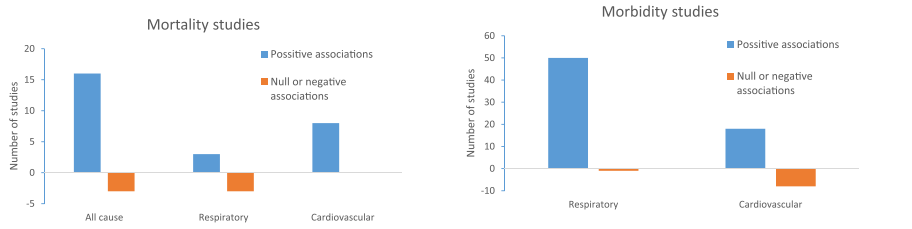
HIGHLIGHTS

- A systematic review on the health effects of BB emissions in the framework of the WHO activities on air pollution
- PM2.5 and PM10 originating from BB were associated with all-cause and cardiovascular mortality.
- High risk of respiratory morbidity on smoky days
- High risk of bias related to poor estimation of BB exposure and lack of adjustment for important confounders

GRAPHICAL ABSTRACT



ARTICLE INFO

Article history:
Received 6 November 2020
Received in revised form 20 March 2021
Accepted 21 March 2021
Available online 26 March 2021

Editor: Philip K. Hopke

Keywords:
Wildfires
Agricultural fires
Domestic heating
Health effects
Epidemiological studies
PM emissions

ABSTRACT

Biomass burning (BB) including forest, bush, prescribed fires, agricultural fires, residential wood combustion, and power generation has long been known to affect climate, air quality and human health. With this work we supply a systematic review on the health effects of BB emissions in the framework of the WHO activities on air pollution. We performed a literature search of online databases (PubMed, ISI, and Scopus) from year 1980 up to 2020. A total of 81 papers were considered as relevant for mortality and morbidity effects. High risk of bias was related with poor estimation of BB exposure and lack of adjustment for important confounders. PM10 and PM2.5 concentrations originating from BB were associated with all-cause mortality: the meta-analytical estimate was equal to 1.31% (95% CI 0.71, 1.71) and 1.92% (95% CI −1.19, 5.03) increased mortality per each 10 μg m^{-3} increase of PM10 and PM2.5, respectively. Regarding cardiovascular mortality 8 studies reported quantitative estimates. For smoky days and for each 10 μg m^{-3} increase in PM2.5 concentrations, the risk of cardiovascular mortality increased by 4.45% (95% CI 0.96, 7.95) and by 3.30% (95% CI −1.97, 8.57), respectively. Fourteen studies evaluated whether respiratory morbidity was adversely related to PM2.5 (9 studies) or PM10 (5 studies) originating from BB. All found positive associations. The pooled effect estimates were 4.10% (95% CI 2.86, 5.34) and 4.83% (95% CI 0.06, 9.60) increased risk of total respiratory admissions/emergency visits, per 10 μg m^{-3} increases in PM2.5 and PM10, respectively. Regarding cardiovascular morbidity, sixteen studies evaluated whether this was adversely related to PM2.5 (10 studies) or PM10 (6 studies) originating from BB. They found both positive and negative results, with summary estimates equal to 3.68% (95% CI −1.73, 9.09) and 0.93% (95% CI −0.18, 2.05) increased risk of total cardiovascular admissions/emergency visits, per 10 μg m^{-3} increases in PM2.5 and PM10, respectively. To conclude, a significant number of studies indicate that BB exposure is associated with all-cause and cardiovascular mortality and respiratory morbidity.

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https://doi.org/10.1016/j.scitotenv.2021.146739

Contents

1. Introduction

Biomass burning (BB) including accidental wildfires, agricultural fires for land clearing and land-use change, residential wood combustion, and power generation has long been known to affect both climate and human health. Humans are responsible for about 90% of BB with only a small percentage of natural fires contributing to the total amount of vegetation burned (Cole, 2001). Although BB is a relatively small source category with a global contribution to mortality by ambient fine particles (PM2.5) and ozone (O_3) of about 5%, its areal range is large (e.g. in South America and Africa) and is the main source of air pollution in large parts of Canada, Siberia, Africa, South America and Australia (Lelieveld et al., 2015). Biomass burning is also widespread in Southeast Asia (e.g. Indonesia and Malaysia) for agricultural use, residential energy production and waste burning (Lelieveld et al., 2015; Koppmann et al., 2005; bin Abas et al., 2004) and in Europe for residential heating (Olsen et al., 2020).

Biomass burning is considered a significant emission source of gaseous pollutants and particulate matter (PM) Active trace gases (e.g. sulphur dioxide (SO_2), nitrogen oxides (NOx), ammonia (NH_3)) released from BB are major precursors of secondary aerosols and tropospheric O_3 in the atmosphere. The combustion of biomass generates gross greenhouse gases GHG emissions, such as methane (CH_4) and carbon dioxide (CO_2) roughly equivalent to the combustion of fossil fuels (IPCC, 2014: 877). Particles emitted from and formed in BB plumes are typically a mixture of black carbon (BC), brown carbon (BrC) and organic carbon (OC) (Zhang et al., 2020; Reid et al., 2005) and can also contain inorganic material (ash) and heavy metals from contaminated biomass fuel (Olsen et al., 2020). Most of the particle emissions arising from BB fall in the PM2.5 fraction (Reid et al., 2005).

Smoke particles have major direct and indirect effects on climate due to light absorption and scattering and their role as cloud condensation nuclei (CCN). While GHG and BC emitted from fires absorb radiation and have a warming effect, the influence of solar radiation scattering by organic aerosol and the production of CCN has an indirect cooling effect on the Earth's lower atmosphere (IPCC, 2013). The large uncertainty that BB emissions have on the radiative budget arises from the variability in emission factors, fuel type and moisture, combustion phase, aerosol particle size, chemical composition, aging, wind conditions, and the high uncertainty related to both measurement methodologies and atmospheric models (Mallet et al., 2017; Andreae and Merlet, 2001).

Studies suggest that BB has increased on a global scale over the last 100 years, and modelling calculations indicate that global warming will lead to more frequent and larger fires (Liu et al., 2009). The Intergovernmental Panel on Climate Change (IPCC) states that energy produced by BB is considered to be 'carbon neutral' since emissions are subsequently eliminated by future growth (IPCC, 2006). However, neutrality of BB CO_2 emissions is increasingly controversial, as it is based on the assumption of high-speed regrowth/foresting (Holm et al., 2020; Cherubini et al., 2011). To reduce emissions of CO_2 from fossil fuel burning and meet CO_2 emission reduction targets, many countries intend to or already have substituted fossil fuels by biomass in existing power plants (Johnston and van Kooten, 2015), and policies have been adopted (i.e. feed-in tariffs, a premium on market prices and tradable renewable energy certificates) for the use of biomass for domestic heating. As a result, an increase in BB emissions occurred in the last decade (Viana et al., 2016; EEA, 2017), and probably will continue in the near future. A review by Sigsgaard et al. (2015), concluded that anthropogenic BB emissions are increasing in the last years in Europe in contrast to emission form other sources, negatively affecting respiratory and, possibly, cardiovascular health in Europe. As a response to fuel poverty, wood/BB is considered as a cheap form of fuel when gathered locally. For example, a recent study in Greece documented a 30% increase in winter PM, and a 2.5-fold increase in biomass combustion markers and a 20–30% decrease in fuel oil tracers coinciding with the economic crisis (Amaral et al., 2016).

To calculate BB impact on air quality and eventually on human health it is important to identify good tracers of BB emissions. Potassium has been extensively used as an inorganic tracer to apportion BB contributions to ambient PM (Chen et al., 2017) due to the presence of potassium oxide and salts, in wood smoke. However, potassium is not a unique tracer of BB as it has other sources, such as sea salt and soil dust. On the other hand, levoglucosan is one of the most abundant organic compounds associated with PM in wood smoke and can be considered a tracer for BB emissions (Simoneit et al., 1999; Simoneit, 2002).

Previous reviews have reported positive associations between wildfire smoke exposure and respiratory health effects, specifically exacerbations of asthma and chronic obstructive pulmonary disease. In contrast to respiratory health risks, the data on cardiovascular effects are mixed (Reid et al., 2016a). These previous reviews did not calculate the combined estimates of the health effects from exposure to BB emissions. We found only one review that examined heterogeneity of effect in studies conducted in North America (Kondo et al., 2019). With this work we intend to supply a systematic review and meta-analysis on the short-term health effects from outdoor exposure to BB emissions in the framework of the activities carried by WHO to expanding the knowledge base about impacts of air pollution on health as suggested by the World Health Assembly WHA68.8 resolution in 2015 and the roadmap on air pollution A69/18 adopted in 2016. This review contributes to the agenda of the technical activities of the WHO air pollution programme. Additionally, this report will contribute to the update of the WHO Global Air Quality Guidelines (AQGs); a global project coordinated by the WHO Regional Office's European Centre for Environment and Health (ECEH) in Bonn (Germany), including participation from all WHO Regions and WHO headquarters.

2. Materials and methods

This systematic-review with meta-analysis was conducted following the protocol for systematic reviews and meta-analyses on air pollutants and health effects established by the WHO Global Air Quality Guidelines (AQGs). The review panel came from an international collaboration of research groups from Spain and Italy. Screening of studies, data extraction, data synthesis and studies results were performed by the Spanish group while the meta-analysis was performed by the Italian group. WHO experts and external advisors were solicited for their critical analysis and consolidated expertise in the field. The review protocol was registered at the International Prospective Register of Systematic Reviews (PROSPERO) with registration number CRD42018099286 on 20 July 2018. The results were structured and presented in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA).

2.1. Information sources and search strategy

A literature search of online databases (PubMed, ISI, and Scopus) from 1980 up to 2020 was performed using the following terms: *Biomass burning, Biomass combustion, Wood burning, Wood combustion, Wood smoke, Wood fire, Forest fires, Wildfire(s), Wildland fire, Biomass, Domestic heating, Residential heating, Vegetation fire, Agricultural fire, Agricultural burning, Harvest fires, Open-area burning, Stubble burning, Brown carbon, Levoglucosan,* in combination with the terms *Health effects, Mortality, Morbidity, Hospital, Admissions, Admission Visits, Respiratory, Cardiovascular, Circulatory, Cerebro-vascular, Cardio-pulmonary, Asthma, Rhinitis, Pregnancy, Allergy, Cross over, Long time series, Cohort.* In addition, references of the retrieved articles were examined to identify further relevant articles. Previously published reviews and reports were also consulted and studied (WHO, 1999; Fowler, 2003; Naeher et al., 2007; Hutton et al., 2006; EPA, 2008; Dennekamp and Abramson, 2011; Benmantnia et al., 2014; Youssouf et al., 2014; WHO, 2015; Liu et al., 2015; Sigsgaard et al., 2015; Reid et al., 2016a; Black et al., 2017, Chen et al., 2017; Kondo et al., 2019; Olsen et al., 2020).

2.2. Data management

Conference papers and articles written in other language than English were excluded. Exposure studies concerning indoor exposure from residential biomass burning (cooking, heating), occupational exposure (firefighters), toxicological studies and studies on long-term health effects were also excluded. A sifting process identified (from study titles, abstracts and the full paper) those studies suitable for inclusion in the review. Studies with small sample size (e.g. Vora et al., 2011; Cooper et al., 1994), studies that did not specify the method used to assess BB exposure (Hashim et al., 1998) and source apportionment studies where the BB source was not well separated from other combustion sources like fossil fuel combustion were excluded from this review (Ostro et al., 2007; Wilhelm et al., 2012). Studies focusing on public health advisories and behavioural change (e.g.: Kolbe and Gilchrist, 2009) were also excluded.

2.3. Risk of bias

In the present review the critical issues were related to:

- Assessment of BB exposure: in epidemiologic studies differences in the accuracy of the estimates of exposure for various components can impact the results, shifting to association of health outcome with one component with less error than another one, but highly correlated in reality. The way selected by different studies to determine what type or fraction of pollutant arising from BB emissions to be used for health studies varies widely and in some cases the error might be large.
- Identification of BB emissions. Biomass covers a wide variety of materials (agro-wastes, wood, and grass, pellets among others), and the composition of the emissions when fired also widely varies. All these factors might influence greatly in the inter-comparability of different studies.
- Confounder adjustment. Several factors other than PM concentrations (such as weather conditions and other air pollutants) might confound the temporal association between BB and health outcomes.

2.4. Meta-analysis

We grouped studies with common health outcomes such as all-cause mortality, respiratory and cardiovascular mortality, respiratory diseases, asthma. Due to statistical heterogeneity between studies in terms of study design (location, age groups, BB sources, BB exposure assessment, and different confounding factors) a random-effects meta-analysis was conducted, of the association between different exposure metrics and mortality and morbidity. Only studies that adjusted for important confounders such as weather conditions including temperature and seasonality were considered for meta-analysis. All effects are expressed as % change of risk, and corresponding 95% confidence intervals, per fixed increases in exposure: YES vs NO for the "smoke" indicator, 10 μg m^{-3} for PM10 and PM2.5.

2.5. Selection process

We used the following Population, Exposure, Comparison, Outcome, Study Design (PECOS) statement: In any population including subgroups of susceptible individuals, what is the association of BB emissions with health outcomes (Mortality: cardiovascular, respiratory, cerebrovascular; Morbidity: cardiovascular; respiratory) observed in outdoor short-term exposure. A total of 81 original articles were considered as relevant for obtaining data on mortality and morbidity effects of the BB (Fig. 1). Two experts participated in the selection of the papers reaching agreement for all reviewed studies.

3. Results

3.1. Mortality studies

A total of 19 studies have evaluated the effects of BB emissions on mortality. Table 1 reports information on the study area, population, methodology used for BB exposure and key findings of each study. Most of these studies have investigated the impact of vegetation fires or forest fires, while 5 source apportionment studies (Berger et al.,

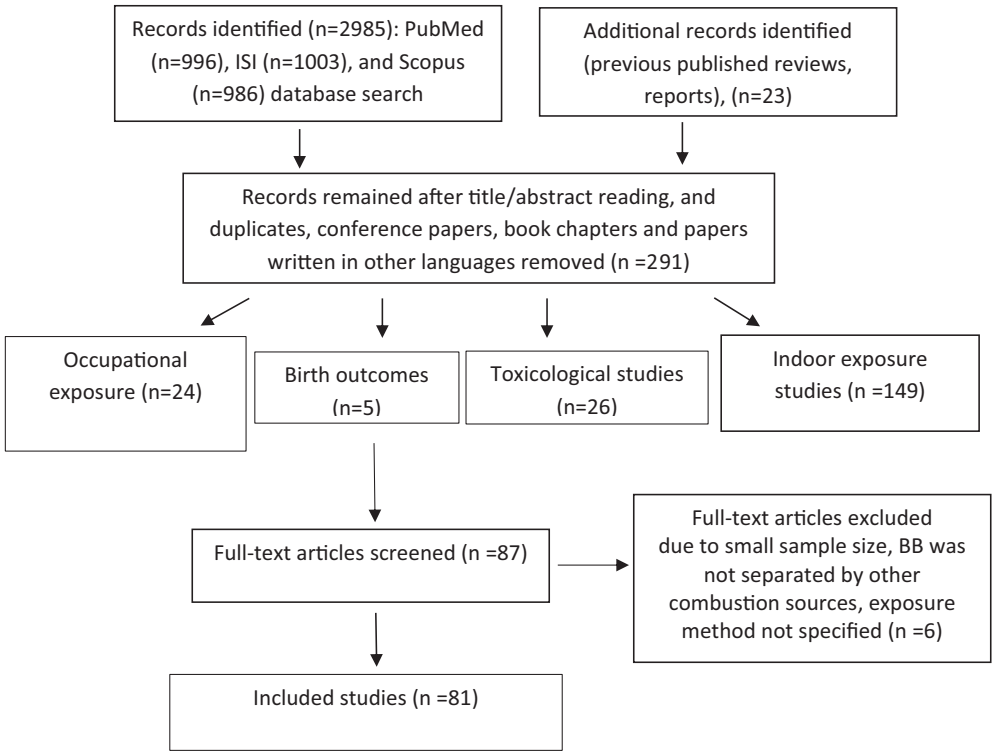

Fig. 1. PRISMA flow diagram representing the identification, screening and selection process performed in the current systematic review and meta-analysis.

2018; Thurston et al., 2016; Park et al., 2014; Ito et al., 2006; Mar et al., 2006) have studied the effect of BB without distinguishing the type of source: wildfires, residential heating. Nine of the 19 studies were conducted in US, six in Europe, one in Brazil, two in Australia, and one study in Malaysia, Asia. No study was conducted in Africa. The majority of the studies take advantage of long time series data and provide growing evidence of significant increases in mortality. Only two studies (Hanninen et al., 2009; Vedal and Dutton, 2006) have investigated the short-term effect of wildfires on mortality during a short-time period (2-week period).

Different methods were used to assess the exposure to BB emissions. Most of the studies have used PM data from ground monitoring sites and examined their variation and peak concentrations during wildfires. e.g. Morgan et al. (2010) identified smoky days as days with city-wide 24 h average PM10 concentrations greater than the 99th percentile for the study period; Faustini et al. (2015) used smoke surface concentration maps. Xi et al. (2020), and Nunes et al. (2013) have used satellite data and atmospheric models to determine the exposure to PM during fire events. Four studies, all from US (Thurston et al., 2016; Park et al., 2014; Ito et al., 2006; Mar et al., 2006) are source apportionment studies that used factor based receptor models to identify and quantify the contribution of BB to PM ambient concentrations. In all studies mortality data came from governmental agencies or bureaus.

3.1.1. All-cause mortality

Most of the studies (16 out of 19) found positive associations between BB and all-cause mortality (even though in some cases not statistical significant) and only three studies (Thurston et al., 2016; Zu et al., 2016; Vedal and Dutton, 2006) did not report any association. Out of the 19 studies listed in Table 1, 11 reported quantitative estimates of associations with mortality suitable for a meta-analysis and at the same time properly adjusted for weather conditions. Fig. 2 presents the study-specific effect estimates, together with summary estimates from a random-effects meta-analysis, of the association between different exposure metrics and all-cause mortality. All effects are expressed as % change of risk, and corresponding 95% confidence intervals, per fixed increases in exposure: YES vs NO for the "smoke" indicator, 10 μg m^{-3} for PM10 and PM2.5.

The results of the meta-analysis are consistent with an association of each exposure metric with daily all-cause mortality (Fig. 2). Only four studies provided quantitative and comparable effect estimates for the 2-level exposure variable: Presence vs absence of smoke attributed to forest fires. They all showed an increased mortality on smoky days, as reflected by the meta-analytical estimate, equal to 2.61% (95% CI 1.02, 4.20) increased risk of all-cause deaths on smoky days compared to smoke-free days.

Similarly, daily mean PM10 and PM2.5 concentrations originating from BB were associated with all-cause mortality: the meta-analytical estimate of the three eligible studies (one on a short forest fire and two source apportionment studies) focussing on PM2.5 is equal to 1.92% (95% CI−1.19, 5.03) increased mortality per each 10 μg m^{-3} increase of PM2.5. The corresponding effect of PM10 (as a summary of five eligible studies) is 1.31% (95% CI 0.91, 1.71). Given that BB emissions mostly fall in the PM2.5 fraction (Reid et al., 2005) we combined studies on PM2.5 and PM10 and calculated the combined risk estimate (see figure in Appendix A). The risk estimate is equal to 1.32% (95% CI 0.93, 1.72).

3.1.2. Cause-specific mortality

Out of the 19 studies listed in Table 1, six have investigated the associations between BB exposures (smoke indicator, PM10, PM2.5) and respiratory mortality but only three have reported quantitative estimates; two studies used smoke presence as exposure type and one used PM2.5. This prevented us to calculate a meta-analytical estimate of effect. Specifically, Doubleday et al. (2020) estimated that wildfire events were associated with a 9.0% (95% CI 0.0, 18.0) increase in the risk of same-day respiratory mortality. Faustini et al. (2015) reported that respiratory mortality was up to 3.90% higher on smoky days. The study by Analitis et al. (2012) found the highest effects on respiratory mortality: 16.2%, (95% CI 1.3, 33.4) increased risk for medium-sized fires in Greece. Morgan et al. (2010) and Johnston et al. (2011) reported null findings for respiratory mortality in Sidney. Both studies suggested

Table 1
Summary of studies investigating impact of BB emissions on mortality.

Location	Authors	Study period	Population	Health outcomes	Exposure assessment	Findings
Washington, US	Doubleday et al., 2020	June 1–September 30, 2006–2017	All ages and age stratified sub-groups	All-cause, cardiovascular, respiratory, cerebrovascular mortality	PM2.5 from fixed monitoring network. Wildfire smoke days were defined as days with PM2.5 >20.4 μg m^{-3}	All-cause mortality increased by 1.0% (95% CI −1.0, 4.0) greater on wildfire smoke days compared to non-wildfire smoke days. Respiratory mortality increase by 9.0% (95% CI 0.0, 18.0), COPD mortality increased by 14.0% (95% CI 2.0, 26.0).
253 counties, US	Xi et al., 2020	2008–2012	Dialysis patients	Mortality	PM2.5 estimated using Community Multiscale Air Quality, CMAQ model with and without fire emissions.	All-cause mortality increased by 4% on the same day (RR = 1.04; 95% CI 1.01, 1.07) and by 7% cumulatively over 30 days following exposure (RR = 1.07; 95% CI 1.01, 1.12) for a 10 μg m^{-3} increase in wildfire PM2.5.
California, US	Berger et al., 2018	2002–2011	All ages	All-cause, cardiovascular, IHD mortality	PM2.5 source apportionment using factor based receptor model (Positive Matrix Factorization model).	All-cause mortality 2 days after exposure (lag2) (% change in odds per IQR width increase in BB-PM2.5 = 0.8, 95% CI 0.2, 1.4), cardiovascular (% changelag2 = 1.3, 95% CI 0.3, 2.4), and IHD (% changelag2 = 2.0, 95% CI 0.6, 3.5).
Helsinki, Finland	Kollanus et al., 2016	2001–2010	All ages	All-cause, cardiovascular mortality	PM2.5 estimated from satellite data and atmospheric chemistry models	Cardiovascular mortality increased by12.4%, (95% CI 0.2, 26.5) for 10 μg m^{-3} increase in wildfire PM2.5
Metropolitan areas, US	Thurston et al., 2016	1982–2004	Adults	IHD mortality	Source apportionment of PM2.5 using absolute principal component analysis (APCA)	IHD mortality HR 1.00, 95% CI 0.99, 1.01.
Boston, New York, US	Zu et al., 2016	July 2002	All ages	All causes, respiratory, cardiovascular mortality	PM2.5 obtained from fixed monitoring sites. Peak PM2.5 related to fires	Daily mortality rates were unaffected by marked increases in PM2.5 concentrations
Mediterranean cities	Faustini et al., 2015	2003–2010	All ages	All causes, respiratory, cardiovascular mortality	PM10 obtained from fixed monitoring sites. Fire-smoke obtained from satellite data. Forest-fire affected days when smoke concentrations >8 μg m^{-3}	All-cause mortality increased by 1.78%, (95% CI −0.91, 4.53), cardiovascular mortality by 6.29%, (95% CI 1.00, 11.85) on smoky days. All-cause mortality increased up to 1.10%, respiratory mortality up to 3.90%, cardiovascular mortality up to 3.42%, for 10 μg m^{-3} increase in wildfire PM10 and after controlling for Saharan dust
Madrid, Spain	Linares et al., 2015	Jan. 2004 –Dec. 2009	All ages and aged ≥75	Mortality	PM10, PM2.5 obtained from fixed monitoring sites. BB influenced air mass advection occurred on 56 days	PM10, rather than PM2.5, were associated with an increase in natural cause mortality on days with biomass advection particularly in the ≥75 year age group (RR all ages1.035, 95% CI 1.011, 1.060; RR ≥ 75 years 1.066 95% CI 1.031, 1.103).
Moscow, Russia	Shaposhnikov et al., 2014	2006–2010	All ages	Mortality	PM10 and O_3 obtained from fixed monitoring sites	Interactions between high temperatures and air pollution from wildfires contributed to more than 2000 deaths. RR 1.004, 95% CI 1.001, 1.008 at T < 18 °C; 1.008, 95% CI 1.004, 1.011 at T 22 °C; 1.014, 95% CI 1.010, 1.019 at T > 30 °C and for a 10 μg m^{-3} increase in PM10
Phoenix, Arizona, US	Park et al., 2014	Feb. 9, 1995–Dec. 31, 1997	Elderly (>65)	Cardiovascular mortality	PM2.5 source apportionment using factor based receptor model (Positive Matrix Factorization).	Increase in daily mortality counts per 5th-to-95th percentile of BB-PM2.5 for lags 0, 1, 2, 3 and 5 in the range 0.10–0.26.
Brazilian Amazon	Nunes and Ignotti, 2013	2005	Elderly, different age groups >65	Cardiovascular, cerebrovascular mortality	PM2.5 from satellite observations, using Coupled Aerosol and Tracer Transport Mode model. BB affected days when PM2.5 > 25 μg m^{-3}	Correlation between BB-PM2.5 exposure and cardiovascular disease, acute myocardial infarction and cerebrovascular disease mortality rates were 33%, 39% and 6% respectively. No association for cerebrovascular disease mortality.
Athens, Greece	Analitis et al., 2012	1998–2004	All ages	Mortality	Black smoke obtained from fixed monitoring sites. Forest fires were classified by size Forest fire affected days when forest fire burned more than 10000m^2	Medium sized fires: increase of 4.9% (95% CI 0.3, 9.6) in all-cause mortality, 6.0% (95% CI −0.3, 12.6) in cardiovascular mortality and 16.2%, (95% CI 1.3, 33.4) in respiratory mortality. Large fire: all-cause mortality increase 49.7%, (95% CI 37.2, 63.4), cardiovascular mortality 60.6%, (95% CI 43.1, 80.3) and respiratory mortality 92.0% (95% CI 47.5, 150.0).
Sydney, Australia	Johnston et al., 2011	1994–2007	All ages	Mortality	PM10 obtained from fixed monitoring sites. Smoke-day was considered when PM10 > 99th percentile	Mortality increased by 5% (RR 1.05, 95% CI 1.00, 1.10) for days affected by smoke haze from bushfires. Cardiovascular mortality increase 10% (RR 1.10, 95% CI 1.00, 1.20) per 10 μg m^{-3} increase in PM10.
Sydney, Australia	Morgan et al., 2010	1994–2002	All ages	All causes, respiratory, cardiovascular	PM10 obtained from fixed monitoring sites. Smoke-day was considered when PM10 > 99th percentile	Positive associations for 10 μg m^{-3} PM10 increases from bush fires, with small increase in all-cause (RR 1.008, 95% CI 0.99, 1.019)

(continued on next page)

Table 1 (*continued*)

Location	Authors	Study period	Population	Health outcomes	Exposure assessment	Findings
				mortality (and hospital admissions)		and cardiovascular mortality (RR 1.008, 95% CI 0.992, 1.023). No associations for respiratory mortality.
11 Finnish provinces	Hanninen et al., 2009	Aug. 26–Sep. 8, 2002	All ages	Mortality	PM10, PM2.5 obtained from fixed monitoring sites before, during and after fire event	Mortality increased by 2% (RR 1.021, 95% CI 0.93, 1.12) per 10 μg m^{-3} increase of PM2.5 during fires.
Washington US	Ito et al., 2006	1988–1997	All ages	Total non-accidental, cardiovascular, and cardiorespiratory mortality	PM2.5 source apportionment using factor analysis based receptor models	Mortality excess risk estimate of 2% (95% CI −1.48, 5.48)
Phoenix, Arizona, US	Mar et al., 2006	1995–1997	All ages	Non-accidental and Cardiovascular mortality	PM2.5 source apportionment using factor analysis based receptor models	Mortality excess risk estimate of 1% (95% CI −9.29, 11.29). Cardiovascular mortality median excess risk estimate 8.3% among 8 methods.
Denver, US	Vedal and Dutton, 2006	2 days June 2002	All ages	All-cause and cardiorespiratory mortality	PM10, PM2.5 from fixed monitoring sites	No perceptible increases in daily natural and cardiorespiratory mortality.
Kuala Lampur, Malaysia	Sastry, 2002	Apr.–Nov. 1997	All ages and different age sub-groups	Natural-cause mortality	PM10 obtained from fixed monitoring sites. Exposure to forest fires when PM10 > 210 μg m^{-3}	Mortality increased by 0.8% (all ages) per 10 μg m^{-3} increase in PM10

CI: confidence intervals, HR: hazard ratio, IHD: ischemic heart disease, COPD: chronic obstructive pulmonary disease, RR: relative risk, IQR: interquartile range.

that the lower number of daily deaths ascribed to respiratory causes in Sydney compared to deaths from cardiovascular diseases, may have limited the power of their analysis to find an effect on respiratory mortality.

Eight studies have reported quantitative estimates for cardiovascular mortality (Fig. 3). For studies on PM10 we could not obtain a summary estimate as only two studies were identified. For smoky days and for each 10 μg m^{-3} increase in PM2.5 concentrations, the

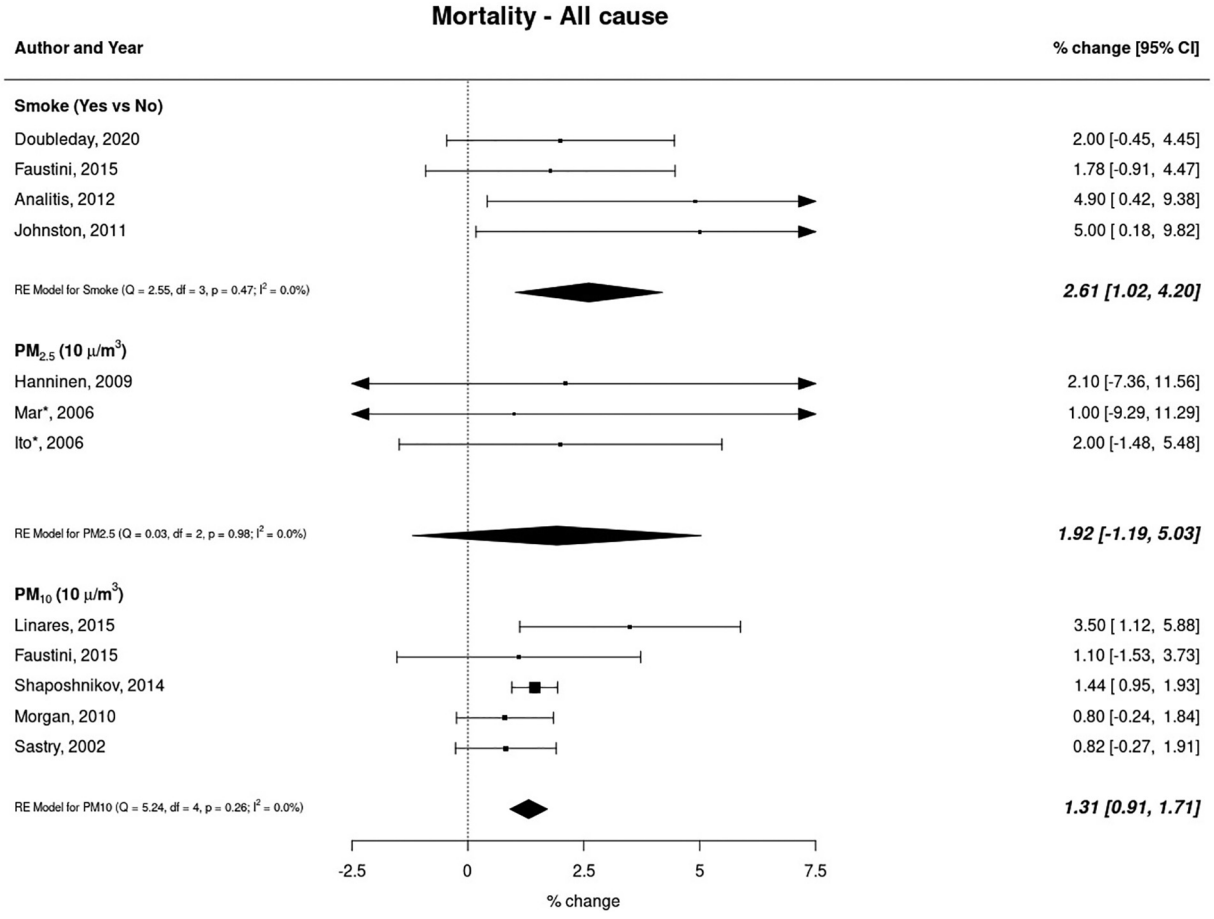

Fig. 2. Random-effects meta-analysis of associations between exposures to BB and all-cause mortality. Squares represent study-specific effect estimates, with size of the square proportional to its weight in the meta-analysis; Diamonds represent meta-analytical effect estimates. *Estimates were transformed from 5th–95th increases of PM2.5.

risk of cardiovascular mortality increased by 4.45% (95% CI 0.96, 7.95) and by 3.303.30% (95% CI -1.973, 8.57), respectively. By combining studies on PM2.5 and PM10 we calculated a combined risk estimate of 2.70% (95% CI −0.20, 5.60) (see Appendix A).

The increased all-cause and cardiovascular mortality risks are somewhat higher than the risk typically reported for short-term exposure to outdoor PM (e.g. C. Liu et al., 2019 reported increased risks of mortality from 0.4 to 0.7% per 10 μg m^{-3} of PM2.5 or PM10). These discrepancies might be explained by the much higher PM concentrations during smoky days compared to non-smoky days. For instance, Linares et al. (2015) and Johnston et al. (2011) reported high PM levels that practically tripled the permitted WHO values for PM10 and PM2.5 (observed concentrations of up to 150 μg m^{-3} for PM10 and up to 71 μg m^{-3} for PM2.5).

3.2. Morbidity studies

A total of 63 studies were identified in the literature on the impact of BB emissions on cardiorespiratory morbidity (Table 2). The majority of the studies focused on the impact of wildfires, four source apportionment studies (Ostro et al., 2016; Gass et al., 2015; Gent et al., 2009; Janssen et al., 2002) did not specify the type of BB source, and Sarnat et al. (2008) referred to a mixed source of forest fires and residential wood burning (in Appendix A we categorized studies by BB source type: forest fires/agricultural fire/mix, residential heating). Two studies (Mnatzaganian et al., 2015; Golshan et al., 2002) investigated the effects of agricultural BB. Pope et al. (2017) investigated the impact of fireplace emissions on emergency department visits while Sarigiannis et al. (2015) examined the association between lung cancer risk and outdoor exposure to BB for domestic heating. Most of the studies examined short periods with intense BB emissions that usually covered less than six months while 17 studies studied long-term data series typically >1 year. The majority of the studies, 31 in total, were conducted in the US followed by studies that took place in Australia (15 studies). Six studies were conducted in Asia, five in Brazil, three in Canada and three in Europe. No studies were set in Africa.

The most commonly used method for assessing outdoors exposure to BB emissions was the use of measurements from fixed monitoring sites, followed by satellite-based methods, and air quality chemistry models to calculate PM2.5 exposure during fire events. Only three studies used specific compounds such as levoglucosan and PAHs concentrations as BB tracers (Weichenthal et al., 2017; Croft et al., 2017; De Oliveira Alves et al., 2015). Five studies were source apportionment studies using factor based receptor models to determine the contribution of BB to ambient PM concentrations. In five studies the impact of fires was determined by the statistical association of fire occurrence with cardiorespiratory morbidity.

The monitoring data usually covered pre-, during- and post-fire periods. Most of the studies determined "exposed period" based on the start/end dates of fire events but did not specify how the start/end days were identified. Some studies used thresholds of air monitoring data to categorize days; for example, in the study of Liu et al. (2017) the smoke exposure was defined as >2 consecutive days with daily wildfire PM2.5 concentrations >20 μg m^{-3}. Another study assigned high PM10 exposure when PM10 concentrations exceeded 40 μg m^{-3}, and low exposure when PM10 levels were below 10 μg m^{-3} (Johnston et al., 2002). Ignotti et al. (2010) considered high exposure periods when the annual hours of PM2.5 exceeded 80 μg m^{-3}.

3.2.1. Respiratory diseases

In general, emergency visits for respiratory symptoms (asthma, bronchitis, dyspnea, pneumonia and COPD symptoms) increased with BB-exposure. Only one study (Mueller et al., 2020) did not find associations between BB exposure and respiratory morbidity in Thailand. Out

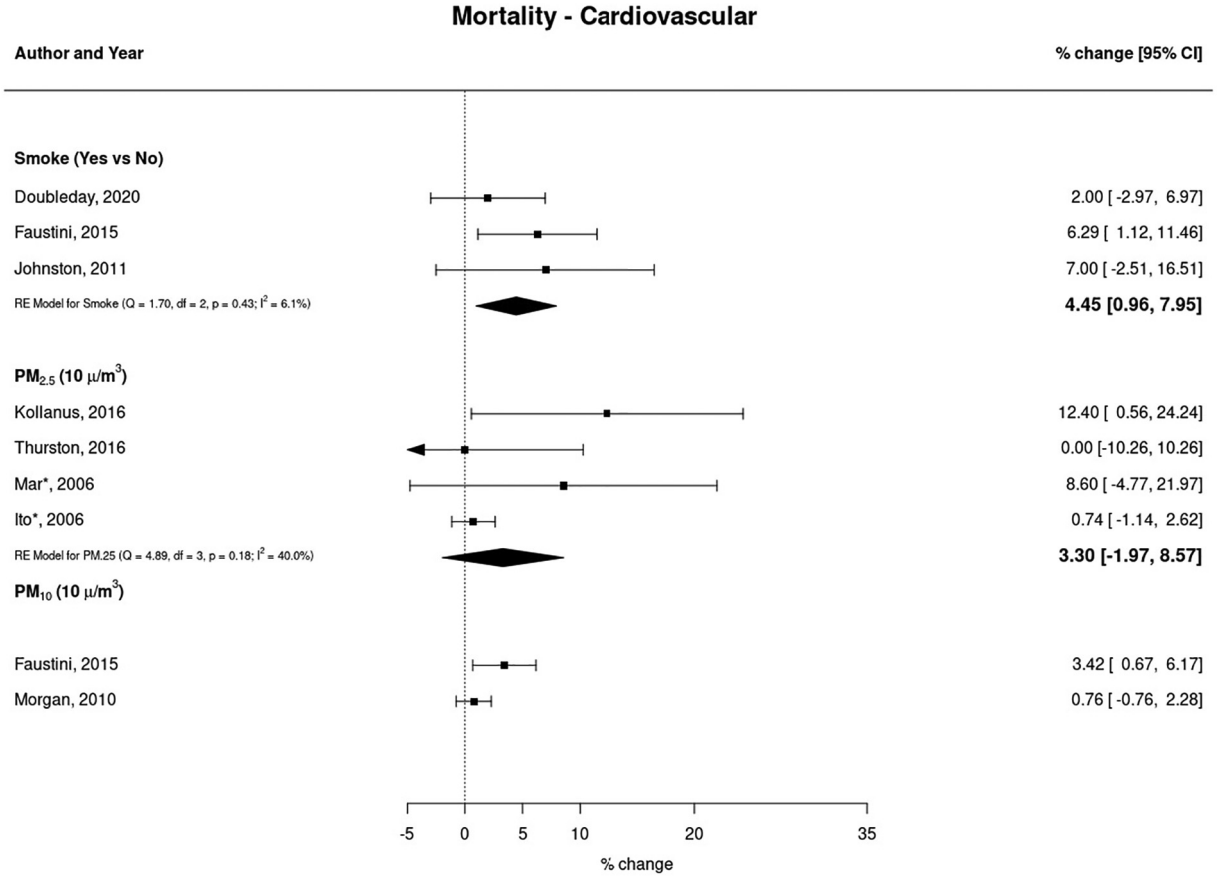

Fig. 3. Study-specific estimates of association between exposures to BB and cardiovascular mortality. *Estimates were transformed from 5th–95th increases of PM2.5.

Table 2
Summary of studies investigating impact of BB emissions on morbidity.

Location	Authors	Study period	Population	Health outcomes	Exposure assessment	Findings
Thailand	Mueller et al., 2020	2014–2017	All ages	R-, C-, IHD-hospital visits	PM10, PM2.5 from monitoring sites. Peak PM10, PM2.5 associated to BB	No associations were found for BB affected days.
Colorado, US	Stowell et al., 2019	May–August 2011–2014	All ages	R-, C-EDVs	BB-PM2.5 from ground measurements, chemical transport models, and remote sensing data	Per 1 μg m^{-3} increase in BB-PM2.5, associations were observed for asthma (OR 1.081, 95%CI 1.058, 1.105)) and combined respiratory disease (OR 1.021, 95%CI 1.012, 1.031).
692 counties, US	DeFlorio-Barker et al., 2019	2008–2010	Elderly >65 years	R, C, Asthma HAs	BB-PM2.5 from monitoring sites and atmospheric chemistry model. BB affected day when BB-PM2.5 > 5 μg/m^3	Asthma risk 6.9% 95%CI 3.71,10.11 on smoke days Increased risk for R and C HA was similar on smoke and non-smoke days
California, US	Reid et al., 2019	May 6–September 26, 2008	All ages	R-EDVs, R-HAs	PM2.5, O3 from fixed monitoring sites, satellite data, chemical transport models.	Asthma EDVs relative risk RR = 1.112 and 95% CI (1.087, 1.138) for a 10 μg m^{-3} increase in PM2.5
San Diego US	Hutchinson et al., 2018	August 16–December 15, 2007	All ages (0–64) and different age sub-groups	C, R, COPD, Asthma	BB-PM2.5 from smoke emissions and dispersion models. BB exposure as 5-day exposure to fire vs pre-fire periods	Respiratory diagnoses increased by 34% during BB exposure (rate ratio, RR 1.34, 95% CI 1.18, 1.52), and for asthma increased by 112% (RR 2.12; 95% CI 1.57, 2.86) associated with a 10 μg m^{-3} increase in PM2.5.
8 California air basins, US	Wettstein et al., 2018	May 1–September 30, 2015 wildfire season	All ages, age- and sex-stratified groups	EDVs	BB-PM2.5 and smoke from the National Oceanic and Atmospheric Administration (NOAA) Hazard Mapping System (HMS) Fire and Smoke product.	Cardiovascular ED visits were elevated across all lags, with the greatest increase on smoke days and among those aged ≥65 years at lag 0 (RR 1.15, 95% CI 1.09, 1.22). Cerebrovascular ED visits were associated with smoke, especially among those 65 years and older, RR 1.22 (95% CI 1.00, 1.49),
4 cities, US	Krall et al., 2017	1999–2009, 2004–2010, 2001–2007, 2006–2009.	All ages	R -EDVs	PM2.5 source apportionment based on receptor models	RR 1.006 (95% CI, 1.003, 1.010) for Atlanta, 1.008 (95% CI: 0.996, 1.019) for Birmingham, 1.007 (95% CI: 0.999, 1.016) for St. Louis, and 1.001 (95% CI 0.989, 1.013) for Dallas associated with an IQR increase in lag 2 PM2.5 from BB.
Western US (561 counties)	Liu et al., 2017	2004–2009	All ages	C-HAs, R-HAs	BB-PM2.5 from chemical transport model. BB exposure defined as ≥2 consecutive days with BB-PM2.5 >20 μg m^{-3}	Increase in risk of R-HA 7.2% (95% CI 0.25, 15) i during smoke wave days. No association for C-HA
Phoenix, US	Pope et al., 2017	Nov. 1–Jan. 31, 2008–2012	All ages divided in adults and children	Asthma-related EDV (A-EDVs)	PM2.5 obtained from fixed monitoring sites. BB exposure when PM2.5 >35 μg m^{-3}	Elevated estimates of A-EDVs risk among adults on lag days 2 (RR 1.19, 95% CI 1.06, 1.34) and 3 (1.20, 95% CI 1.05, 1.37).
Washington, US	Gan et al., 2017	1 July–31 October 2012	All ages	R-HAs	BB-PM2.5 from ground measurements, chemical model, hybrid model	COPD odds ratio OR 1.11, (95% CI 1.03, 1.18) using ground monitoring data, OR 0.99, (95% CI 0.93, 1.05) using a chemical model, and OR 1.09, (95% CI 1.03,1.15) using hybrid model
British Columbia, Canada	Weichenthal et al., 2017	2014–2015	All ages and elderly (>65)	MI-HAs	PM2.5 from fixed monitoring sites. Levoglucosan was used as BB tracer.	Increase of 5 μg m^{-3} in 3-day mean PM2.5 associated with an increased risk of MI among elderly (>65) OR: 1.06, 95% CI 1.03, 1.08. High biomass contribution OR = 1.19, 95% CI 1.04, 1.36; Mid biomass contribution OR = 1.08, 95% CI 1.06, 1.09; Low biomass contribution OR = 1.04, 95% CI 1.03, 1.06 (per 5 μg m^{-3} PM2.5 increase)
Rochester, NY, US	Croft et al., 2017	Winters 2011–2013	Adults	Inflammation, coagulation, thrombosis biomarkers	PM2.5 Delta-C, a tracer of BB and other PM2.5 air pollutants	Each 0.13 μg m^{-3} increase in Delta-C was associated with an increase in fibrinogen levels (1.009, 95% CI 1.0023, 1.0159)
Colorado, US	Alman et al., 2016	Jun. 5–Jul. 6, 2012	All ages	R-EDV (for asthma COPD) and C-EDV and	Modelled and measured PM2.5 from fixed monitoring sites.	Positive associations between lag 0 PM2.5 and asthma/wheeze (1 h max OR 1.01, 95% CI 1.00, 1.01, per 10 μg m^{-3}); 24 h mean (OR 1.04, 95% CI 1.02, 1.06, per 5 μg m^{-3}), and COPD (1 h max OR 1.01, 95% CI 1.00, 1.02, per 10 μg m^{-3}); 24 h mean OR (1.05, 95% CI 1.02, 1.08, per 5 μg m^{-3}). C-EDV results were consistent with no association
Victoria, Australia	Haikerwal et al., 2016	Dec. 2006–Jan. 2007	All ages	EDV for asthma and COPD	PM2.5 from monitoring sites. BB by smoke models. BB exposure when PM2.5 >50 μg m^{-3}.	An IQR increase in PM2.5 levels of 8.6 μg m^{-3} was associated with an increase in EDVs for asthma by 1.0196, 95% CI 1.0002, 1.039 on the day of exposure. No association with COPD.
California, US	Ostro et al.,	Jan. 2005–Oct.	All ages	C-EDVs and R-EDVs	Source apportionment of PM2.5 by	Excess risk, ER per IQR 2.7 μg m^{-3}, for

Table 2 (*continued*)

Location	Authors	Study period	Population	Health outcomes	Exposure assessment	Findings
	2016	2009		Dysrhythmia, IHD, MI, HF, Asthma	factor based receptor model PMF. K used as a marker for BB.	R-EDVs (ER: 2.6%, 95% CI 0.8, 4.4), asthma (ER:1.8%, 95% CI −0.0, 3.6), COPD (ER: 0.9 95% CI: 1.0, 2.9), pneumonia (ER: 2.1, 95% CI: −0.8, 5.1) IHD (ER: 1.0%, 95% CI: −1.5, 3.5) MI (ER:2.4%, 95% CI: −1.8, 6.8). No association for HF and C-EDVs.
San Diego, US	Reid et al., 2016b	May 6–Sep. 15, 2008	All ages, different age sub-groups	C-,R-EDV, C-,R-HA, IHD, cerebrovascular, asthma, COPD, pneumonia	PM2.5 obtained from fixed monitoring sites. BB exposure estimated by exposure prediction modelling	Increase in risk for R-HA and asthma-HA (RR 1.018, 96% CI 1.007, 1.029 and 1.07, 95% CI 1.05, 1.10, per 5 μg m^{-3} increase) and asthma-EDVs (RR 1.06, 95% CI 1.05, 1.07), COPD-EDVs, *RR 1.02, 95% CI 1.01, 1.04 per 5 μg m^{-3} increase). No associations were found for C-HAs or C-EDVs
North Carolina, US	Tinling et al., 2016	5 May 5–15 June 2011	All ages, age and sex stratified groups	R and C ED visits	Wildfire PM2.5 was obtained from modelled predictions from the Smoke Forecasting System.	Relative risk associated with a 10 μg m^{-3} increase in 24-h PM2.5 for respiratory/other chest symptoms 1.06 (1.00–1.13), upper respiratory infections 1.13 (1.05–1.22), 'all-cause' cardiac outcomes 1.06 (1.00–1.13).
Melbourne, Australia	Dennekamp et al., 2015	Jul. 2006–Jun. 2007	>35 yr and sex stratified sub-groups	Out-of-hospital cardiac arrests (OHCA)	PM10, PM2.5 from fixed monitoring site. BB exposure by chemical transport models and pollutants peaks during fires	Among men during the fire season, greater increases in OHCA were observed with IQR increases in the 48-h lagged PM2.5 (1.081, 95% CI 1.023, 1.141 per 6.1 μg m^{-3}), PM10 (1.111, 95% CI 1.015, 1.215, per 13.7 μg m^{-3}).
Atlanta, Georgia, US	Gass et al., 2015	2002–2010	Children	EDVs for paediatric asthma	Source apportionment of PM2.5 using receptor models	Biomass burning was associated to EDVs for paediatric asthma for a cumulative 8-day exposure, but it was not statistically significant.
Maui, Hawaaii, US	Mnatzaganian et al., 2015	Apr. 2011–Apr. 2012	All ages	EDVs, HAs, and prescription fills for acute R illnesses	Burn and no burn sugar cane days	Higher incidence of respiratory distress in smoke-exposed regions when greater amounts of acres were burned than in non-smoke exposed regions ($P = 0.015$, OR 1.024, 95% CI 1.012, 1.048)
Porto Velho, Brazilian Amazon	De Oliveira Alves et al., 2015	Aug–Oct. 2011 and Nov. 2011–Mar. 2012	All ages	Lifetime lung cancer risk	Source apportionment study using levoglucosan as BB tracer. PAHs were used to calculate the lifetime lung cancer risk.	The BB factor is found to be the dominating aerosol source, having 75.4% of PM10 loading. The estimated lung cancer risk calculated during the dry season largely exceeded the WHO health-based guideline
Athens, Greece	Sarigiannis et al., 2015	Winter 2012–2013	All ages	Lung cancer risk	PM10, PM2.5, PM1 from monitoring sites. Levoglucosan and PAHs used as BB tracers	High lung cancer risk attributable to PAHs on PM emitted from BB
New Mexico, US	Resnick et al., 2015	Wallow fire of 2011	All ages	R-EDVs (R, asthma) and C-EDVs	PM2.5 obtained from fixed monitoring sites. BB exposure when PM2.5 > 15μgm^{-3}	increased risk of EDVs for >65 yr for asthma (RR 1.017, 95% CI 1.10, 1.029), for diseases of the veins, lymphatic and circulatory system (RR 1.016, 95% CI 1.010, 1.024), and for all-causes C (1.011, 95% CI 1.010, 1.012).
Sydney, Australia	Johnston et al., 2014	Forest fires 1996–2007	All ages and children (<15)	R-EDVs and C-EDVs	PM2.5, PM10 from fixed monitoring sites. Smoke event when PM10 or PM2.5 >99th percentile for the study period	Fire smoke associated with (lag 0) increases in ED attendances for all non-trauma conditions (OR 1.03, 95% CI 1.02, 1.04), respiratory conditions (OR 1.07, 95% CI 1.04, 1.10), asthma (OR 1.23, 95% CI 1.15, 1.30), and COPD (OR 1.12, 95% CI 1.02, 1.24). IHD-ED increased at a lag 2 (OR 1.07, 95% CI 1.01, 1.15) while arrhythmias had an inverse association (OR 0.91, 95% CI 0.83, 0.99)
81counties, US	Le et al., 2014	6–8 July 2002	Elderly >65	HA	PM2.5 from fixed monitoring sites. BB exposure during haze vs non haze period	49.6% increase in respiratory HA (95% CI, 29.8, 72.3) and 64.9% increase in cardiovascular HA (95% CI, 44.3–88.5) when the smoke plume was present
Brazilian Amazon	do Carmo et al., 2013	2004–2009	Children	HAs	PM2.5 were estimated by Coupled Chemistry Aerosol and Trace Gases Transport Model	Increases of 10 μg m^{-3} in PM2.5 exposure was associated with 5.6% increase HAs
Southern California, US	Dohrenwend et al., 2013	Wildfires in 2007	All ages	R-EDVs	Number of visits compared 2 weeks before and during the wildfires.	Dyspnoea complaints increased by 3.2 visits per day. During the fire the diagnoses of asthma increased significantly by 2.6 patients per day.
3 cities, Australia	Martin et al., 2013	1994–2007	All ages	R-HAs and C-Has	PM10, PM2.5 from fixed monitoring sites. BB exposure when PM10 or PM2.5 >99th percentile	BB were associated with 6% (OR 1.06, 95% CI 1.02, 1.09) increase in R-HA, 13% increase in COPD-HA (OR1.13, 95% CI 1.05, 1.22) and 12% increase in asthma-HA

(*continued on next page*)

Table 2 (*continued*)

Location	Authors	Study period	Population	Health outcomes	Exposure assessment	Findings
						(OR1.12, 95% CI 1.05, 1.19), non-statistically significant association for pneumonia.
Victoria, Australia	Dennekamp et al., 2011	Nov. 1, 2006–Mar. 31, 2007	Age >35	Out-of-hospital cardiac arrests (OHCA)	PM10, PM2.5, from fixed monitoring sites. Smoke event was considered when PM10 or PM2.5 >99th percentile	An IQR increase of 6.0 μg m^{-3} in PM2.5 was significantly associated with an increased a lag 2 risk of an OHCA of 4.52% (RR 1.05, 95% CI 1.005, 1.09); 5.57 for PM10 (IQR 11.7 μg m^{-3}, 6.57%, RR 1.065, 95% CI 1.004, 1.13) and 35.7% for CO (IQR 0.3 ppm RR 1.357, 95% CI 1.090, 1.689) For NO_2, SO_2, and O_3, the associations with OHCA risk were non-significant.
British Columbia, Canada	Henderson et al., 2011	Jul. 1–Sep. 30, 2003	All ages, sex, age and socio-economic status subgroups	R-, C-EDVs, R-, C-HAs (R all causes and asthma)	PM10 from fixed monitoring sites. BB-PM10 from dispersion model, and smoke plumes from satellite data	IQR increase of 30 μg m^{-3} in PM10 was associated with an increased R-EDVs, OR 1.05, 95% CI 1.03, 1.06, asthma visits, OR 1.16, 95%, 1.09, 1.23. HAs (OR 1.15, 95% CI 1.00, 1.29). No association with all-causes cardiovascular problems
42 North Carolina counties, US	Rappold et al., 2011	Jun. 2008	Adults	R-, C-EDVs	Aerosol Optical Depth, AOD by satellite. BB exposure when AOD ≥ 1.25	Cumulative RR for asthma (1.65, 95% CI, 1.25, 2.1), COPD (1.73, 95% CI 1.06, 2.83), pneumonia and acute bronchitis (1.59, 95% CI 1.07, 2.34), C-EDVs (1.23, 95% CI 1.06, 1.43), heart failure (1.37, 95% CI 1.01, 1.85)
São Paulo Brazil,	Arbex et al., 2010	Mar. 2003 to Jul. 2004	Hospital patients	HA for hypertension	TSP from monitoring sites	Hypertension HAs increase during the harvest period (RR 1.013, 95% CI 1.056, 1.199) that was almost 30% higher than during non-harvest periods (1.090, 95% CI 1.040, 1.143).
Brazilian Amazon Region	Ignotti et al., 2010	2004–2005	All ages and age stratified groups	R-HA	PM2. from fixed monitoring sites. BB exposure when PM2.5 >80 μg m^{-3}	The association of the exposure indicator (AH%) with R-HA was higher for the elderly than for other age groups. For each 1% increase in the exposure indicator there was an increase of 8% in child R-HA, and a 10 and 5% for elderly, and the intermediate age group
Sydney, Australia	Morgan et al., 2010	1994–2002	All ages	R-HA (COPD, asthma, pneuminia), (and mortality)	PM10 from monitoring sites. Smoke-day when PM10 >99th percentile for the study period	A 10 μg m^{-3} increase in BB- PM10 was associated with R-HA RR 1.012, 95% CI 1.002, 1.023, COPD-HA RR 1.038, 95% CI 1.014, 1.062, Asthma RR 1.050, 95% CI 1.018, 1.084, pneumonia RR1.028, 95% CI 1.002, 1.055). No association was found for C-HA and cardiac failure
San Diego, US	Schranz et al., 2010	14–26 Oct. 2007	All ages	R-EDVs	PM2.5 concentrations from fixed monitoring sites	R-EDVs post fires 19.8% vs.5.2% in baseline period. Shortness of breath (6.5% vs. 4.2%) and smoke exposure (1.1% vs. 0%)
Southern California, US	Delfino et al., 2009	Oct. 2003	All ages	R-HA and C-HA	BB-PM2.5 from measured PM2.5, light extinction, and smoke from satellite images.	Per 10 μg m^{-3} BB-PM2.5, acute bronchitis HA across all ages increased by 9.6% (RR 1.10, 95% CI 1.02, 1.18), COPD HA for ages 20–64 by 6.9% (RR 1.07, 95% CI 1.01, 1.13), and pneumonia HA for ages 5–18 years by 6.4% (RR 1.06, 95% CI 1.02, 1.14).
New Haven, Connecticut, US	Gent et al., 2009	2000–2003	Children with asthma (age 4–12)	Symptoms and medication use (MU)	PM2.5 source apportionment (K was used as a BB tracer)	There was no associations between BB and asthma symptoms and medication. Inhaler short, acting (OR 1.0, 95% CI 0.96, 1.03); shortness of breath (OR 1.05, 95% CI 0.95, 1.17), chest tightness (OR 1.06, 95% CI 0.95, 1.18).
Southern California, US	Mirabelli et al., 2009	Oct. 2003	(Age 16–19)	Respiratory diseases atopy and bronchial hyper-responsiveness	Webmail questionnaire was used to assess smoke exposure	Increase respiratory symptoms with increasing frequency of wildfire smoke exposure. Respiratory symptoms were stronger in the lowest quartile of the lung function ratio (e.g., fire smoke 6+ days: Prevalence ratio, R 1.038, 95% CI 1.020, 1.072).
3 cities in Victoria, Australia	Tham et al., 2009	2002–2003	All ages	R-HAs, R-EDVs.	24 h average daily PM10 concentrations (derived from hourly maximum values).	The strongest associations were observed between PM10 and daily R-EDV (RR 1.018, 95% CI 1.004, 1.033). No significant associations were identified with HA.
Darwin, Australia	Hanigan et al., 2008	Apr.–Nov. 1995–2005	All ages	R-HA, C-HA	BB-PM10 exposure determined using visibility data, and predictive peaks of PM10 were mapped against wildfire records	An increase of 10 μg m^{-3} in same-day estimated PM10 was associated with RR 1.05, 95% CI 1.01, 1.11 increase in total R-HA. A stronger association was found for indigenous people than non-indigenous (RR 1.15, 95% CI 1.04, 1.28

Table 2 (*continued*)

Location	Authors	Study period	Population	Health outcomes	Exposure assessment	Findings
						vs. 1.007, 95% CI 1.08, 1.10). No association was found for C-HAs
Atlanta, Georgia, US	Sarnat et al., 2008	Nov. 1998 and Dec. 2002	All ages	R-EDVs and C-EDVs	PM2.5 source apportionment using receptor models and tracer approach	R-EDVs C-EDVs (RR 1.033, 95% CI 1.02, 1.044).
Sao Paulo State, Brazil	Arbex et al., 2007	Mar. 23, 2003–Jul. 27, 2004	All ages	Asthma HAs (A-HAs)	TSP from fixed monitoring sites. BB exposure defined as days with sugar cane fires	For a variation of 10 μg m^{-3} in TSP concentration, A-HAs increased by 10% (RR 1.10, 95% CI 1.03, 1.17) on non-fire days and 13% (RR 1.13, 95% CI 1.02, 1.24), on fire days.
Copenhagen, Denmark	Andersen et al., 2007	Jan. 1, 1999–Dec. 2004	Elderly (age >65) Children (5–18)	R-HAs and C-HAs in the elderly, and asthma (A-HA) in children	Source apportionment of PM10 chemical speciation data from fixed monitoring sites	C-HA (RR 1.040, 95% CI 1.009, 1.072, one-pollutant model) and R-HA (RR 1.084, 95% CI 1.034, 1.136; RR 1.072, 95% CI 1.003, 1.145, for one and two-pollutant models) in elderly are significantly related to the identified BB- PM10 (IQR 5.4 μg m^{-3}).
Darwin, Australia	Johnston et al., 2007	Fires 2000, 2004, 2005	All ages, indigenous population	R-HAs and C-HAs R = COPD, asthma, resp. infections, IHD	Daily PM10 concentrations. Smoke-day was considered when PM10 >99th percentile	R-HA (OR 1.08, 95% CI 0.98, 1.18, per each 10 μg m^{-3} PM10), COPD-HA (OR 1.21, 95% CI 1.00, 1.47), asthma (OR 1.14, 95% CI 0.90, 1.44). No relationship with C-HA
Brisbane, Australia	Chen et al., 2006	1 July 1997–31 Dec. 2000	All ages	R-HA	PM10 obtained from fixed monitoring sites. BB days when bushfires occurred	An increase of PM10 from low (<15 μg m^{-3}) to high level (>20 μg m^{-3}), is accompanied by an increase of 19% in R-HA for wildfire days (RR 1.19, 95% CI 1.09, 1.30) vs. 13% for background days (RR 1.13, 95% CI 1.06, 1.23).
Darwin, Australia	Johnston et al., 2006	Fire seasons of 2000, 2004, 2005	Adults and children	Asthma symptoms (AS), medication (MU-A), asthma attacks (AA), medical services (UMS)	PM10, PM2.5 from fixed monitoring sites. Smoke-day when PM10, or PM2.5 >99th percentile	Positive associations between an increase in PM10 concentrations of 10 μg m^{-3} with AS (IRR 1.240, 95% CI 1.106, 1.39) and MU (IRR 1.035, 95% CI 1.004,1.06). Similar results were obtained for increases of 5 μg m^{-3} of PM2.5 (AS IRR 1.150, 95% CI 1.07, 1.23; MU IRR 1.181, 95% CI 1.076, 1.296). No association with AA and UMS
Southern California, US	Künzli et al., 2006	Oct. 2003	School students (17–18) and (6–7)	Respiratory diseases, medication usage, physician visits	Questionnaire to estimate BB exposure. PM10 from fixed sites during fire activity.	6 days or more of fire smell is associated with more than 4-fold higher rates of eyes symptoms, 3 fold dry cough and sneezing 2 for cold, sore throat, wet cough, medication use, physician visits. Associations tended to be strongest among those without asthma.
British Columbia, Canada	Moore et al., 2006	3-weeks in 2003	All ages	Physician visits for R, C, and mental illness	PM2.5, PM10 from fixed monitoring network. BB-PM2.5 when >10 μg m^{-3} for ≥3 days	A 46% to 78% increase in physician visits for R illness in Kelowna, British Columbia. The lack of a similar effect in Kamloops is likely due to the population being exposed to lower levels of PM2.5. Effects on visits for C diseases or mental disorders were not seen in either community
Vilnius, Lithuania	Ovadnevaitė et al., 2006	Aug.–Sep. 2002	All ages	R diseases, bronchial asthma	PM10, from monitoring sites. Peak PM10 attributed to fires.	R diseases and exacerbation of the bronchial asthma during the fire period were up to 20 times higher in comparison to periods with no fires.
San Diego, US	Viswanathan et al., 2006	Sep.–Nov. 2003,	All ages	EDVs for asthma, R problems, eye irritation and smoke inhalation	Surveillance data before, during, and after the fire. Air pollution data from fixed monitoring sites	Increase in R-EDVs (asthma, other R problems), but not for chest pain; total examinations did not increase. Quantitative data is not supplied.
Kuching, Malaysia	Mott et al., 2005	Jan. 1 1995–Dec. 31, 1998	All ages	All-R (COPD, asthma) and aa-C-HA	Comparison of health outcomes in the wildfire or post-fire period	Increase R-HA specifically for COPD and asthma patients. Persons >65 years with previous HA for C-R disease, R disease or COPD were significantly more likely to be re-HA during the follow-up period in 1997 than in the follow-up period in the pre-fire years
Denver, US	Sutherland et al., 2005	Jun.–Jul. 2002	Adults ≥40 years with COPD	R symptoms	PM10, PM2.5, CO from fixed monitoring site. Days of elevated PM attributed to fires	Increase in R-symptom index on two days of elevated PM2.5 (63 μg m^{-3}) relative to control days (14 μg m^{-3}). Index was higher on spike days vs non spike days.
Sydney, Australia	Jalaludin et al., 2000	January 1994	Children	R symptoms	PM10 were categorized into bushfire and non-bushfire	Association between the BB-PM10 and prevalence of evening wet cough (OR 1.23, 95% CI 1.10, 1.37 per 10 g m^{-3} increase in PM10)
Isfahan, Iran	Golshan et al., 2002	Oct. 2000	1–80 years, sex sub-groups	Asthma, lung function, respiratory symptoms	PM10 obtained from fixed sites. BB exposure when fire occurred	Increased prevalence of respiratory symptoms and various asthma indicators, decreased lung function post-rice stubble

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Table 2 (*continued*)

Location	Authors	Study period	Population	Health outcomes	Exposure assessment	Findings
						burning period relative to period prior to burning
Darwin, Australia	Johnston et al., 2002	Apr. 1–Oct. 31, 2000	Adults and Children	Asthma EDVs	PM10 from fixed monitoring sites. Smoke-day when PM10 or PM2.5 >99th percentile	Increased asthma EDVs associated with PM10, (adjusted rate ratio 1.20; 95% CI, 1.09–1.34 per each 10 μg m^{-3} PM10 increase), especially when PM10 >40 μg m^{-3} (adjusted rate ratio 2.39, 95% CI 1.46, 3.90) compared with days when PM10<10 μg m^{-3}
14 cities, US.	Janssen et al., 2002	1985–1994	All ages	COPD-, C-, pneumonia-HA	BB-PM10 obtained from fire emissions and air quality data.	COPD risk increase 1.4%, pneumonia risk increase 2.3%, C risk increase 2.7%
Indonesia	Kunii et al., 2002	Sep. 2–Oct. 7, 1997	All ages, age sub-groups	R diseases	PM10, PAHs from fixed monitoring sites.	A restrictive respiratory functional pattern was found in 68% of responders
Singapore	Emmanuel, 2000	1997	All ages	R diseases	PM10, PM2.5, from fixed monitoring sites. PM10 peaks attributed to forest fires	Increase in PM10 levels from 50 μg m^{-3} to 150 μg m^{-3} was significantly associated with increase of 12% of upper R tract illness, 19% asthma and 26% rhinitis
Sydney, Australia	Jalaludin et al., 2000	Jan. 1994	Children	PEFR	PM10 from fixed monitoring sites	No significant association between mean PM10 in the fire period and PEFR. Children without bronchial hyperactivity had a significant negative association between PEFR and PM10
Central Florida, US	Sorensen et al., 1999	Jun.–Jul. 1998	All ages	R-EDVs and R-HAs	Surveillance system monitored patient visits during wildfires	EDVs increased substantially for asthma (91%), bronchitis with acute exacerbation (132%), and chest pain (37%). EDVs visits for painful respiration decreased (27%).
Sydney, Australia	Smith et al., 1996	Jan. 1994	All ages	Asthma EDVs	PM10 concentrations, O_3, NO_2 from fixed sites.	No increase in asthma visits with PM10 increase during episode of exposure to bushfire emissions
Singapore	Chew et al., 1995	Sep.–Oct. 1994	Children <12 years	Asthma EDVs	BB-PM10 when PM10 20% higher than the year's average moving trend during fire evens.	Increased asthma EDVs with PM10 during fire episodes. No information when compared with no fire periods.
California, US	Duclos et al., 1990	Aug. 15–Sep. 15, 1987	All ages	R and non-R-EDVs	PM10 from fixed monitoring sites. BB exposure during fire vs non-fire periods.	Asthma (observed/expected ratio OIE = 1.4), COPD (OIE = 1.3) and EDVs were higher during the fires period than non-fires period.

CI, confidence intervals, HAs, hospital admissions; EDVs, emergency department visits; C, cardiovascular; R, respiratory; RR, relative risk; OR, odds risk; MI, myocardial infarction; COPD, chronic obstructive pulmonary disease; IHD, ischemic heart disease, HF, heart failure; CF, cardiac failure; PEFR, peak expiratory flow rates.

of the 63 studies reported in Table 2, 22 provided quantitative estimates suitable for meta-analysis. Specifically, as with mortality studies, also morbidity studies focused on three BB-related exposures: presence vs absence of smoke, daily mean PM10 and PM2.5 concentrations on smoky days (or originating from BB sources). The outcomes mostly related to BB exposures were total respiratory hospitalizations/emergency visits, asthma admissions (in the general population or in children), pneumonia and COPD. Figs. 4–6 present the study-specific effect estimates, together with summary estimates from a random-effects meta-analysis, of the association between the three different exposure metrics and the four aforementioned outcomes, respectively. All studies included in the meta-analysis controlled for critical covariates such as temperature.

Seven studies investigated whether the risk of respiratory admissions/visits was higher on smoky days compared with smoke-free days (Fig. 4). All found positive associations, with higher rates of hospitalizations on smoky days. The highest risk was reported by Hutchinson et al. (2018) in San Diego, where respiratory diagnoses increased by 34% during exposure to wildfires. The summary estimate of the seven studies, therefore, showed a high risk of respiratory morbidity on smoky days by 10.52% (95% CI 3.87, 17.18). Fourteen studies evaluated whether respiratory morbidity was adversely related to PM2.5 (nine studies) or PM10 (five studies) concentrations originating from BB. All found positive associations (Fig. 4). The pooled effect estimates were 4.10% (95% CI 2.86%, 5.34%) and 4.83% (95% CI 0.06, 9.60) increased risk of respiratory admissions/emergency visits, per 10 μg m^{-3} increases in PM2.5 and PM10, respectively. The combined effect estimate for both PM2.5 and PM10 studies was 4.65% (95% CI 2.70, 6.59), see figure in Appendix A.

Twenty-one different studies focussed on asthma in the general population, providing consistent evidence of association of short-term exposure to BB and asthma (Fig. 5A). When comparing day with and without smoke from BB sources, the risk of asthma was 38.26% (95% CI 7.91, 68.60) higher on smoky days. Nine papers were published on the adverse effects of BB-related PM2.5 on asthma: all of them provided positive effect estimates, ranging between 2% and 15% increased risk. Overall, asthma risk increased by 9.19% (95% CI 5.71, 12.68) per 10 μg m^{-3} increases in PM2.5. Similarly, six studies investigated the relationship between daily PM10 (from BB sources) and asthma admissions/visits: the pooled estimate was 10.35% (95% CI 4.44, 16.26) increased risk of asthma per 10 μg m^{-3} increases in PM10. The combined effect estimate for both PM2.5 and PM10 studies was 9.59% (95% CI 6.53, 12.24), see figure in Appendix A.

The results on the association of BB exposure and asthma in children are more conflicting, as they are based on a small number of studies; only six provided quantitative estimates suitable for a meta-analysis and at the same adjusted for important confounders such as weather conditions and in the case of asthma influenza (Fig. 5B). No study used presence-absence of smoke as the main exposure, and only two focussed on PM10. Four papers were published on evidence for association between PM2.5 exposure (from BB sources) and asthma occurrence in children: two showed positive associations (Alman et al., 2016; Gass et al., 2015); one a flat relationship (Delfino et al., 2009; wildfires), and one a negative estimate (Gent et al., 2009). Combining PM2.5 and PM10 studies, paediatric asthma risk increased by 3.52% (95% CI −2.13, 9.18) per 10 μg m^{-3} increase in PM2.5 or PM10.

There is little evidence on the association between daily exposure to BB and hospitalizations (or emergency room visits) for pneumonia (Fig. 6A). Two studies used presence-absence of smoky days as the relevant exposure, and found clear evidence of higher pneumonia rates on smoky days. Four studies investigated the relationship with PM2.5 and

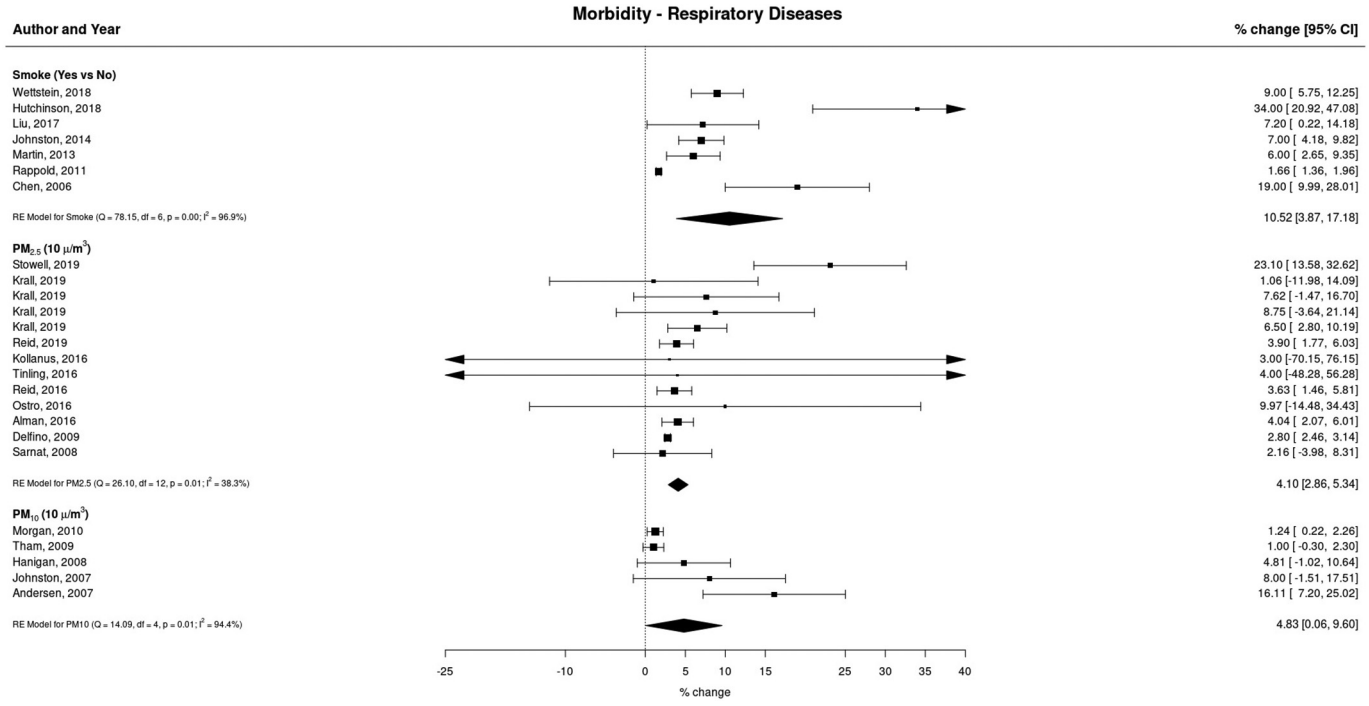


Fig. 4. Random-effects meta-analysis of associations between exposures to BB and respiratory morbidity. Squares represent study-specific effect estimates, with size of the square proportional to its weight in the meta-analysis; Diamonds represent meta-analytical effect estimates.

two studies focussed on PM10. All studies found positive estimates. By combining PM2.5 and PM10 studies we found a pooled estimate of 1.72% (95% CI −0.09, 3.53) increased risk per 10 μg m^{-3} increase in PM2.5 or PM10.

Sixteen studies investigated the relationship between COPD admissions and BB exposures and provided quantitative estimates suitable for a meta-analysis (Fig. 6B): four used presence-absence of smoke as main exposure, nine focused on daily PM2.5 concentrations and four on PM10 from BB sources. In general, the studies comparing days with vs those without smoke provided consistent results of a harmful effect of BB, with COPD admission rates increasing by 13.33% (95% CI 7.31, 19.34) on smoky days compared with smoke-free days, on average. Results on PM exposure were mixed, with most of them showing positive associations. In summary, COPD rates increased by 3.92% (95% CI −1.133, 6.70) and 3.95% (95% CI 1.65, 6.24) per 10 μg m^{-3} increases in BB-related PM2.5 and PM10, respectively.

3.2.2. Cardiovascular diseases

Most studies have shown increases in all-cause or specific cardiovascular morbidity as consequence of BB exposure (e.g. Weichenthal et al., 2017, Sarnat et al., 2008 among others). However, a number of studies reported no increase in hospital admissions or emergency department visits for cardiovascular events during wildfires or residential BB. Liu et al. (2017) found, in a large number of Western US counties a marginally negative estimate between PM2.5 and cardiovascular hospital admissions. Similarly, Stowell et al. (2019) and Alman et al. (2016) in Colorado, US found negative estimates between cardiovascular outputs and wildfire PM2.5. An Australian study by Hanigan et al. (2008) noted decrease in infections and medical visits for cardiovascular problems with the increase in PM10 during bush fires.

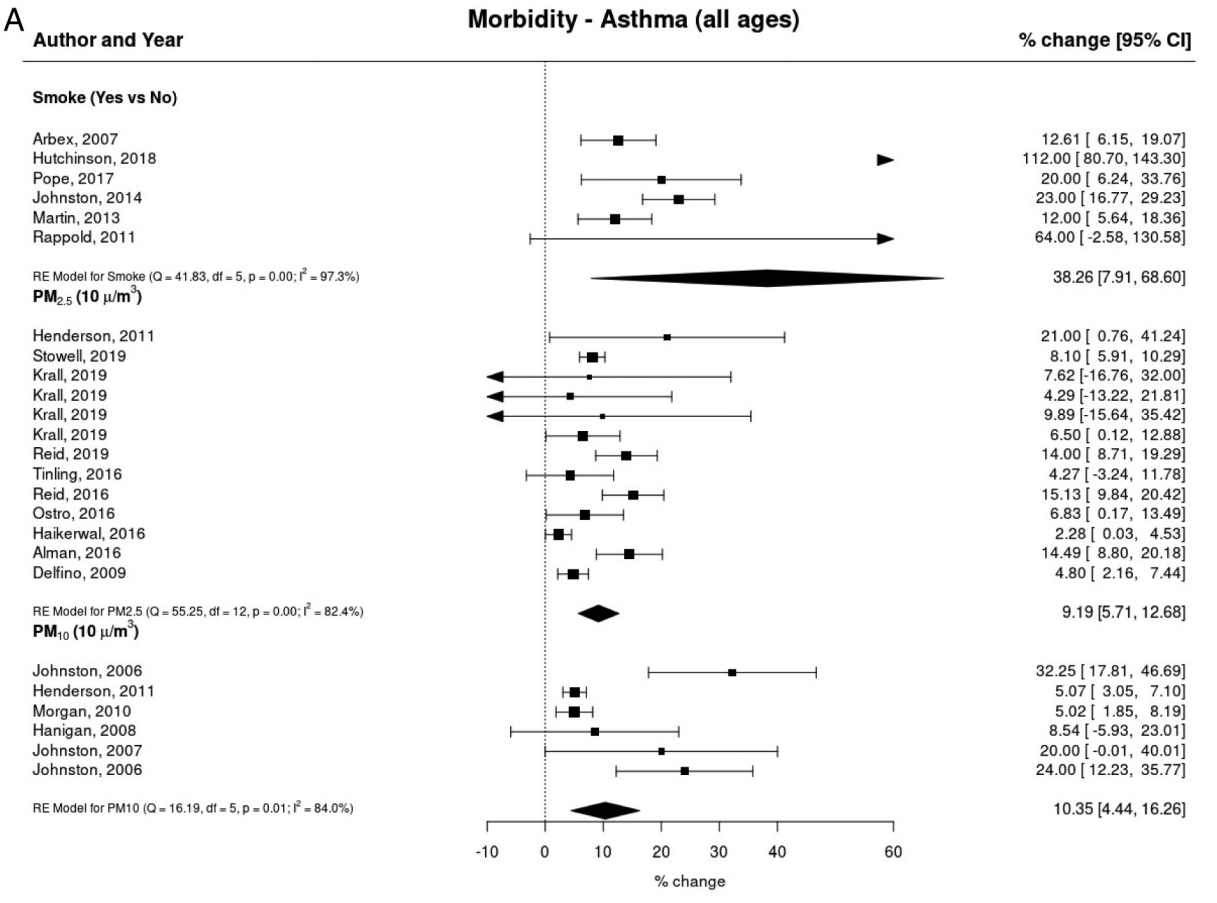
Out of the 63 studies, 23 provided quantitative estimates of associations between cardiovascular morbidity outcomes, with a specific focus on IHD, and BB exposures (presence-absence of smoke, PM2.5, PM10) suitable for meta-analysis, Fig. 7. Seven studies investigated the relationship between presence of smoke and peaks in daily cardiovascular admissions/visits; two of these studies found negative effect estimates. The pooled estimate showed a higher risk of cardiovascular morbidity on smoky days by 4.84%, (95% CI −0.44, 10.11). Sixteen studies evaluated whether cardiovascular morbidity was adversely related to PM2.5 (ten studies) or PM10 (six studies) concentrations originating from BB. They found both positive and negative results, with summary estimates equal to 3.68% (95% CI −1.73, 9.09) and 0.93% (95% CI −0.18, 2.05) increased risk of total cardiovascular admissions/emergency visits, per 10 μg m^{-3} increases in PM2.5 and PM10, respectively.

The results of the association between IHD morbidity and BB exposure were generally consistent with those of total cardiovascular morbidity, showing higher rates of IHD on smoke days compared with smoke-free days, while the effect of BB-related PM2.5 or PM10 was less evident.

The combined effect estimates for respiratory morbidity and especially for asthma are larger than the ones reported in the literature on PM health effects (e.g. Atkinson et al., 2014 reported 4% increase in risk for asthma due to exposure to PM2.5). Most studies reporting large estimates are small size studies (e.g. one month) that observed high differences in PM exposure on smoke vs no-smoke days. For example, in the study of Rappold et al. (2011) PM2.5 concentrations exceed 200 μg m^{-3} during a wildfire much higher than background PM2.5 levels.

3.3. Studies in sub-population groups

A number of studies have assessed whether specific populations are more susceptible to wildfire smoke exposure than the general population (see Table A1 in Appendix A). Health effects from BB were related to age, pre-existing health conditions, gender, ethnicity and socioeconomic status. Larger positive associations between wildfire smoke and cardiorespiratory morbidities were observed for the elderly (Weichenthal et al., 2017) middle-aged and older adults (i.e. Henderson et al., 2011; Johnston et al., 2014; Mott et al., 2005) compared to other age groups. A study of PM10 exposure in Malaysia from the 1997 Southeast Asian wildfires found higher rates of mortality among people 65–74 years old compared to others; a smaller suggestive effect was found among those ≥75 years old (Sastry, 2002). Similarly Kunii et al. (2002) for 'the 1997 haze disaster in Indonesia', mostly



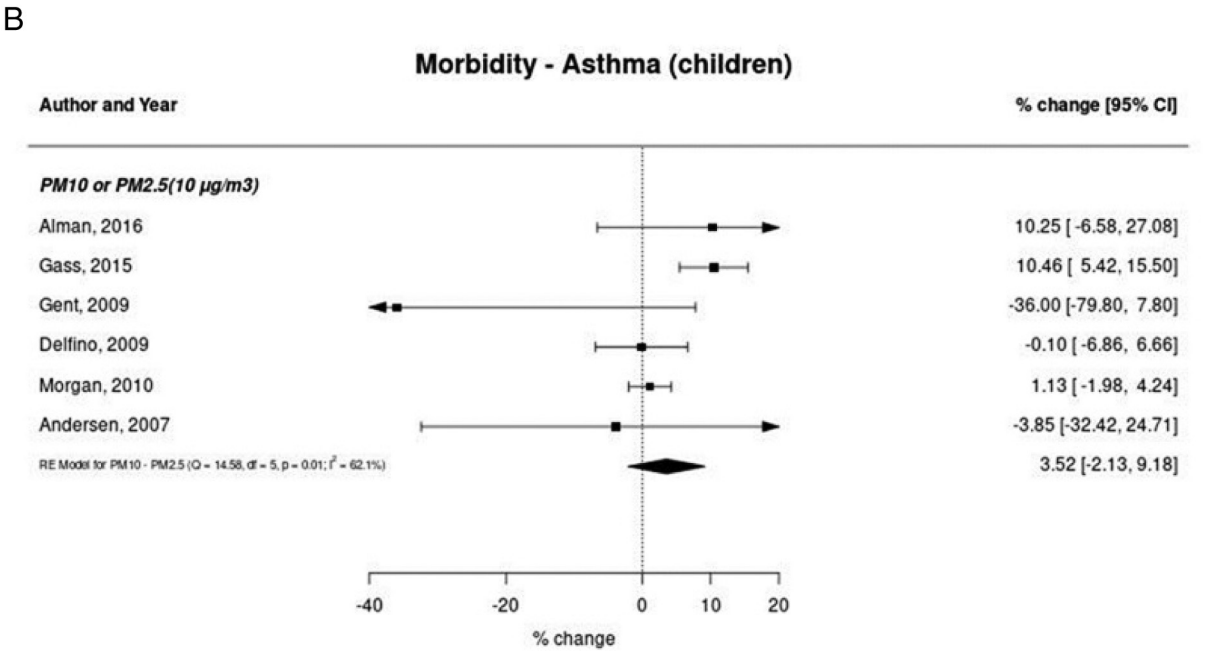
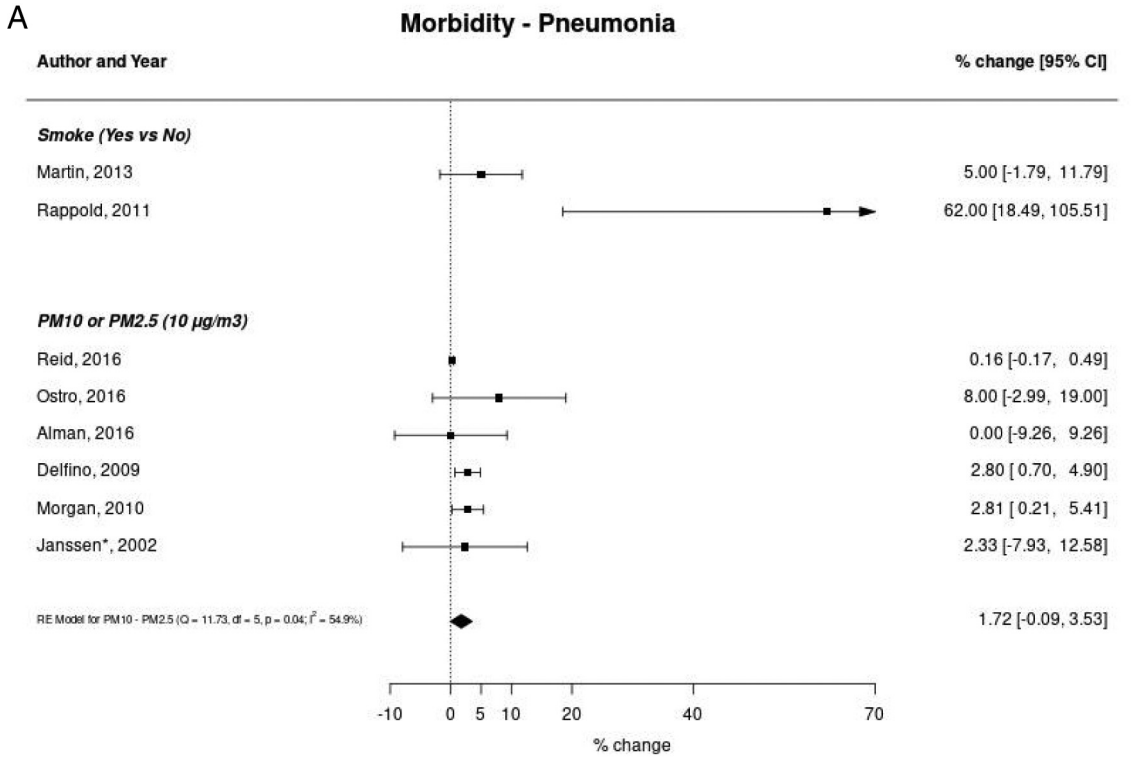


Fig. 5. Random-effects meta-analysis of associations between exposures to BB and asthma hospitalizations/emergency visits for general population (A) and among children (B). Squares represent study-specific square proportional to its weight in the meta-analysis; Diamonds represent meta-analytical effect estimates.



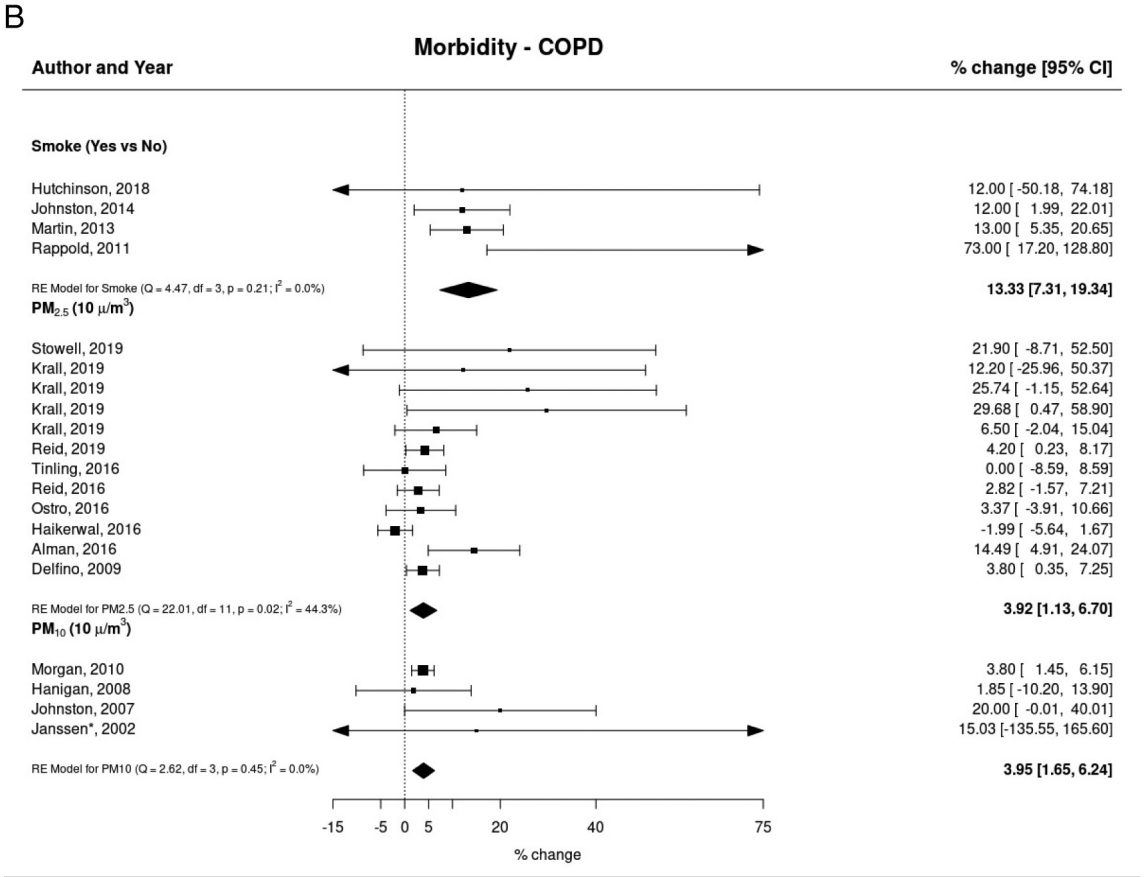
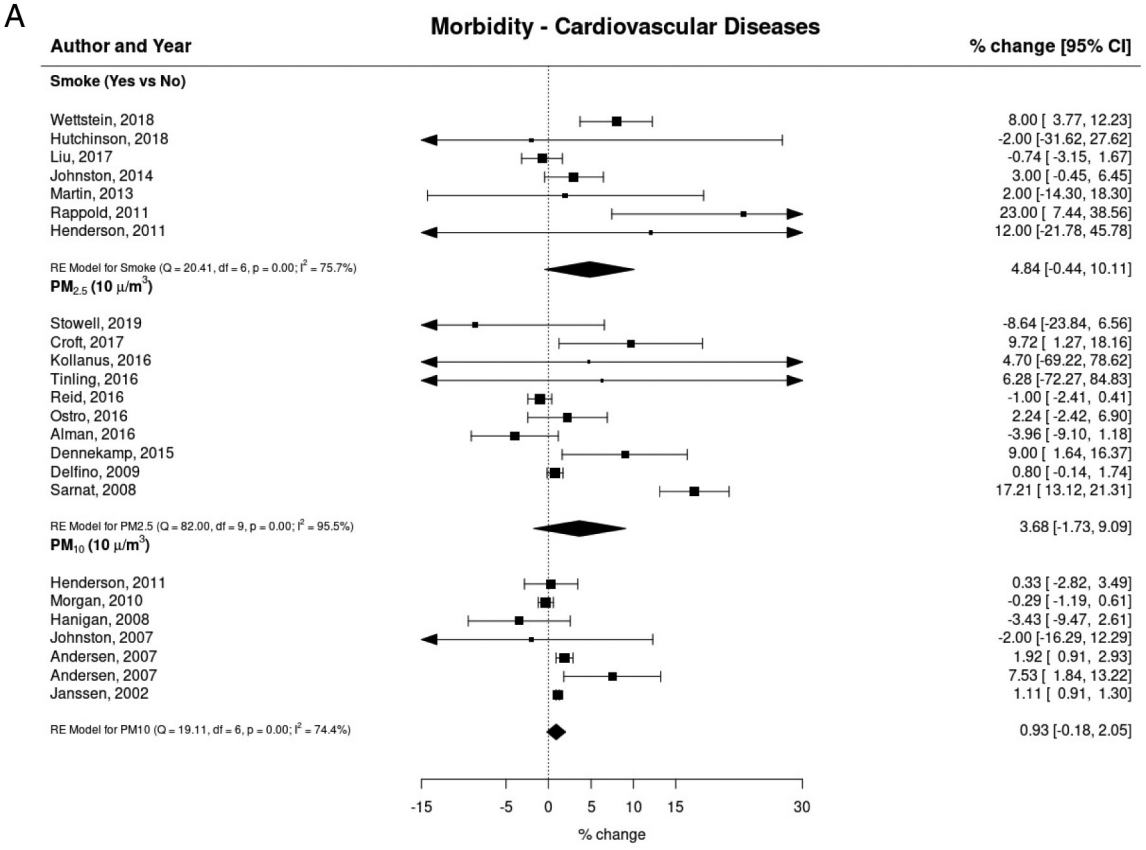


Fig. 6. Random-effects meta-analysis of associations between exposures to BB and pneumonia (A) and COPD (B). Squares represent study-specific effect estimates, with size of the square proportional to its weight in the meta-analysis; Diamonds represent meta-analytical effect estimates.



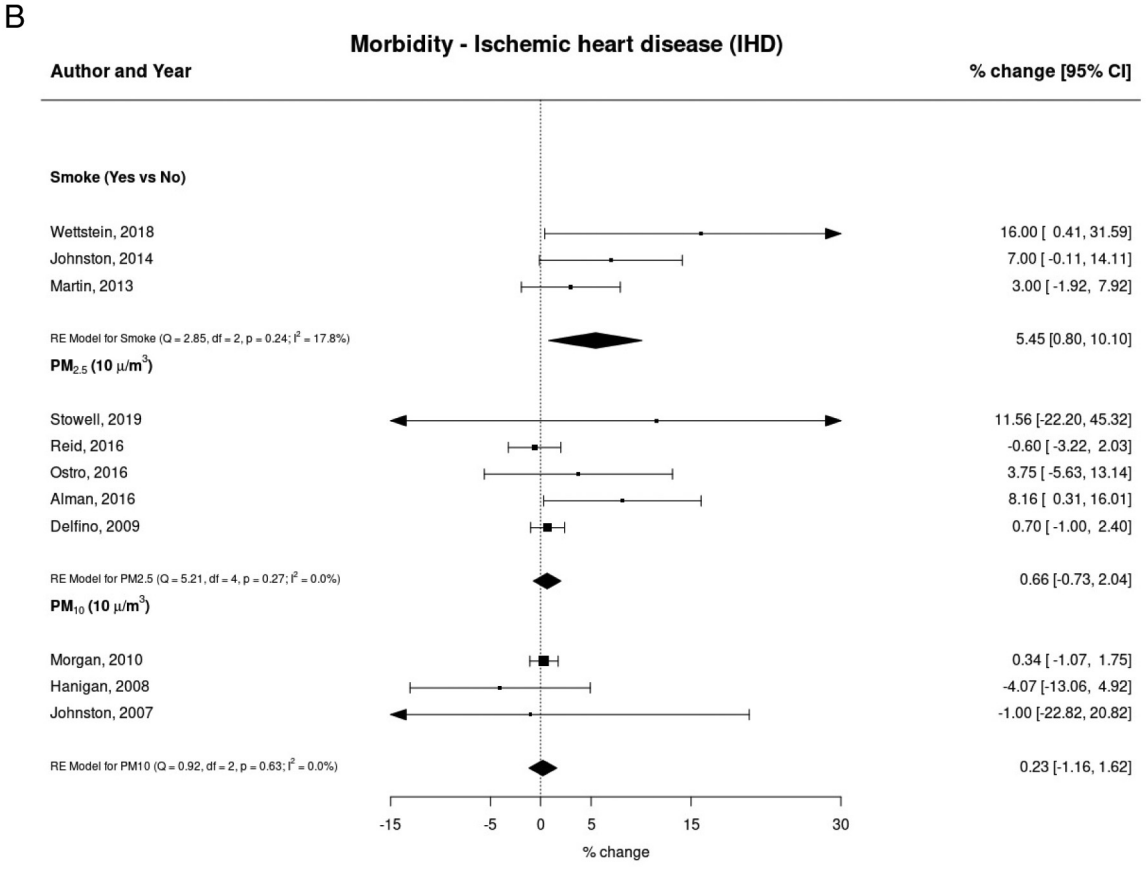


Fig. 7. Random-effects meta-analysis of associations between exposures to BB and cardiovascular (A) and ischemic heart disease morbidity (B). Squares represent study-specific effect estimates, with size of the square proportional to its weight in the meta-analysis; Diamonds represent meta-analytical effect estimates. *Estimates were transformed from 5th–95th increases of PM2.5.

caused by land cleaning fires and forest fires, evidenced that individuals aged >60 years suffered a serious deterioration of overall health compared to other groups.

There was also some evidence of increased vulnerability to adverse health effects from BB exposure among children. Risk of increased respiratory- symptoms, asthma, and hospital contacts associated with wildfire smoke was higher for children (Stowell et al., 2019; Hutchinson et al., 2018; Tinling et al., 2016; Ignotti et al., 2010) compared with other age groups However, a recent study by Pope et al. (2017) did not find any association between elevated wood smoke originated PM2.5 and hospital admissions among children when compared to adult age groups. Similarly, children with asthma did not experience increased respiratory symptoms or medication use during Australian wildfires (Johnston et al., 2006). Johnston et al. (2014) in Sydney for age-specific analyses, found no associations present in children (<15 years) for any respiratory and cardiovascular outcome, although a non-significant trend towards a positive association was seen with childhood asthma.

Men and women may have different health risks when exposed to wildfire smoke. In general, risks for respiratory and asthma-related symptoms (Stowell et al., 2019; Haikerwal et al., 2016; Reid et al., 2016b; Rappold et al., 2011) and for cardiovascular diseases (Jones et al., 2020; Tinling et al., 2016) in relation to wildfire smoke were greater in women than men However, Dennekamp et al. (2015) found a significant association between wood smoke exposure and out-of-hospital cardiac arrests for men but not for women. Henderson et al. (2011) did not find differences in wildfire effect estimates between men and women and respiratory and cardiovascular hospitalisations.

As for ethnic factors, 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 hospitalisation 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).

Pre-existing cardiac or respiratory conditions may plausibly increase vulnerability to wildfire smoke exposure; however, the available evidence is currently inconclusive. Mott et al. (2005) found that people ≥65 years old who were hospitalised for any cardiorespiratory outcome in the first half of the year were at increased risk of being hospitalised during the 1997 Southeast Asian fires compared with similar temporal comparisons in previous years without fires. On the contrary, Schranz et al. (2010) report that patients with significant cardiac or pulmonary diseases were no more likely to visit the emergency department during the 2007 fires in California. In an Australian study, no adverse association was observed between wildfire related PM10 and lung function (peak expiratory flow) in children with reported history of wheezing in the previous period of the 1997 bushfires in Sydney, Australia (Jalaludin et al., 2000). Likewise, children with asthma did not experience increased respiratory symptoms wildfires (Johnston et al., 2006; Künzli et al., 2006; Mirabelli et al., 2009).

Socioeconomic status (SES) was another factor that may influence health effects from BB exposure. Jones et al. (2020) reported that low SES may have increased the risk of out-of-hospital cardiac arrest due to BB exposure during wildfires whereas Henderson et al. (2011) did not observe differences between different SES groups.

3.4. Biomass burning exposure, pregnancy and birth weight

Some studies have demonstrated links between wildfire smoke exposure and birth outcomes (Table S2 in Appendix A). Studies details and effect estimates are given in Table A2 in the Appendix A. Abdo et al. (2019) investigated the association between wildfire exposure and pre-term birth and birth weight in Colorado, US for the period 2007–2015. Exposure to wildfire PM2.5 over the full gestation and during the second trimester were positively associated with pre-term birth, while exposure during the first trimester was associated with decreased birth weight. 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 >9 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 PM2.5 from agricultural BB in the Brazilian Amazon had higher rates of low birthweight babies compared to those exposed to lower levels (Cândido da Silva et al., 2014). On the contrary, Prass et al. (2012) using "heat spots" as a proxy for exposure to smoke from forest fires did not find significant evidence of an association between birth weight and the number of forest fires.

4. Discussion

4.1. Exposure assessment methods

Methods used to assess BB exposure is a crucial element for future studies as direct associations between BB emissions and health outcomes need to be further investigatedPM10 was the most commonly studied, but the majority of US studies focused on PM2.5 exposure. Nevertheless, results have to be equivalent since most PM arising from BB falls in the PM2.5 fraction (Reid et al., 2005). Epidemiological studies in this review have used several methods to estimate BB exposure. Most of the studies used PM measurements from surface air pollutant monitors and compared fire and no fire affected days (e.g. Johnston et al., 2014), satellite observations (e.g. Rappold et al., 2011), atmospheric chemistry models (e.g. Liu et al., 2017) or combinations of the previous methods (Dennekamp et al., 2015). Less studies used BB-PM tracers such as levoglucosan (Weichenthal et al., 2017) or Delta-C (Croft et al., 2017). A number of studies used Positive Matrix Factorization (PMF), Constrained physical receptor model (COPREM), and other factor analysis based receptor models to quantify the daily contribution of BB-PM (e.g. Thurston et al., 2016; Mar et al., 2006). Although all above mentioned methods are useful to assess BB exposure, each method presents limitations. PM data from surface monitoring sites have sparse spatial coverage, while satellite observations often provide data of smoke concentrations in the entire atmospheric column, and they might incorrectly identify some aerosol plumes as clouds (fires produce smoke as thick as some clouds). Atmospheric modelling for smoke simulation is quite challenging because it is difficult to account for the high temporal variability of smoke plumes (Y. Liu et al., 2019). Regarding source apportionment studies they have a limitation in their ability to examine the long lag structure of PM2.5 sources and constituents because chemical speciation data were only available for every 3rd or 6th day. Discrepancies in defining and assessing BB exposure made difficult obtaining consistent results from the review. Indeed, Gan et al. (2017) found different risk estimates for COPD, depending on the method used to estimate BB exposure; odds ratio equal to 1.11, (95% CI 1.03, 1.18) using ground monitoring data, OR = 0.99, (95% CI 0.93, 1.05) using a chemical model, and OR = 1.09, (95% CI 1.03,1.15) using a hybrid model. It should be mentioned that exposure methods used did not consider individual-specific differences such as, time spent outdoors, distance from the emission source and/or air quality monitors, precautions taken during BB events (e.g. during wildfires) that may drastically affect personal exposure to BB. Despite of the different methods and criteria used to define and determine BB exposure there is a large body of evidence indicating that exposure to ambient BB-PM is associated with increased morbidity and mortality.

4.2. Length, population and confounders

The length of reviewed studies varied from a few days where a certain fire smoke episode was studied up to several years. For example, Schranz et al. (2010) studied the impact of wildfires during 21–24 October 2007 on respiratory emergency visits in San Diego, US while Martin et al. (2013) calculated risk estimates of respiratory and cardiovascular morbidity for the period 1994–2007 in three cities in Australia. Long-time series are common in regions with distinct fire seasons, such as Australia (e.g., Hanigan et al., 2008; Johnston et al., 2011; Morgan et al., 2010; Smith et al., 1996). Also source apportionment studies conducted in U.S. (Krall et al., 2017; Gass et al., 2015) have studied long-time data. In general, we did not notice any difference in terms of risk estimates and associated errors when compared studies based on short-time series with longer data sets.

Most of these studies focused on wildfires that occurred close to the studied area. Given that wildfires can dramatically increase PM levels hundreds of kilometres away from the point source a few studies investigated whether wildfire emissions can impact the health of populations living in long distances. The Quebec forest fires in Canada caused a regional air pollution episode in US in early July 2002. Hospitalizations rates were increased for both respiratory and cardiovascular diseases during the smoke affected days among the elderly (Le et al., 2014). However, daily mortality rates were unaffected by marked increases in PM2.5 concentrations (Zu et al., 2016). Contradicting results were found in a Finnish study which reported that a 10 μg m^{-3} increase in PM2.5, attributed to long-range transport of vegetation fires, was associated with cardiovascular mortality (Kollanus et al., 2016).

A critical issue in the reviewed studies is related to control for all of the time-varying factors other than PM concentrations, that could confound the temporal association between BB-PM and health outcomes. Important confounders in studies investigating health impacts of BB emissions include weather conditions, with temperature being one of the most important confounding variable. Shaposhnikov et al. (2014), by examining the interaction between heat wave and wildfire smoke, found that the combination of prolonged high temperatures and high PM10 concentrations (>300 μg m^{-3} largely due to wildfires) contributed to over 2000 deaths. Other important confounders include seasonality, weekly trends, and other air pollutants. Most of the studies discussed in this review have adjusted for principal confounding factors such as temperature, humidity, seasonal and weekly trends. Regarding mortality studies only two studies, Nunes and Ignotti (2013) and Vedal and Dutton (2006) did not adjust for weather conditions. In fact, Vedal and Dutton (2006) reported that peak temperatures coincided with peaks in mortality but did not control for temperature effect. As for cardiorespiratory morbidity, six studies (Mirabelli et al., 2009; Resnick et al., 2015; Künzli et al., 2006; Golshan et al., 2002; Johnston et al., 2002; Kunii et al., 2002) did not adjust for weather variables. Studies investigating the impact of BB exposure on respiratory morbidity additionally adjusted for influenza (e.g. Morgan et al., 2010; Chen et al., 2006) and fungal spore counts (Delfino et al., 2009). Some studies also adjusted for other air pollutants such as NO, and NO_2, (e.g. Smith et al., 1996; Duclos et al., 1990; Gent et al., 2009). Reid et al. (2019) using ozone as interaction term reported that risk estimates were not significantly affected when controlling for O_3 concentrations. One study (Faustini et al., 2015) additionally considered the effect of Sahara dust intrusions that occur simultaneously with wildfires.

The vast majority of the reviewed studies focussed on the general population without age classification while a relevant number of studies have assessed whether specific populations are more susceptible to wildfire smoke exposure than the general population. Susceptibility factors investigated included those related to age groups (e.g. Wettstein et al., 2018), pre-existing disease (e.g. Gent et al., 2009), gender (e.g. Stowell et al., 2019), ethnicity (Johnston et al., 2007) and socioeconomic status (Jones et al., 2020). The findings for differential effects by age were inconclusive. Some studies report that health effects were larger in older individuals (Wettstein et al., 2018) while other researchers found higher impact in infants and children (Hutchinson et al., 2018). Pre-existing cardiorespiratory problems may plausibly increase vulnerability to wildfire smoke exposure but again clear conclusions could not be drawn.

5. Conclusions

The present review and meta-analysis results demonstrated that all-cause and cardiovascular mortality is positively associated with exposure to BB emissions. High levels of BB-PM is a risk factor for respiratory diseases. In spite of the inconsistency of results on cardiovascular morbidity, the number of articles finding associations with wildfire smoke and BB-PM exposure with specific cardiovascular outcomes, is clearly higher than the one of those not finding them. We also discussed the significance of using common, harmonized methodologies for BB monitoring and exposure assessment in future epidemiological studies.

6. Recommendations and implications of key findings

The first challenge in studies investigating health impact of BB emissions is monitoring and apportioning BB derived PM. Given the potentially different compositional patterns of PM arising from different biofuels (wood, grass, agro-fuels) it is recommended using more than one tracer for determining source contributions. Thus, the measurement of K, and BB sugars like levoglucosan is highly recommended for receptor modelling studies. Recently, novel instrumentation such as the aerosol chemical speciation monitor (ACSM) combined with a seven-wavelength aethalometer and receptor modelling is used to identify and quantify BB emissions (Petit et al., 2015; Ealo et al., 2016). Misclassification of personal exposure to BB derived PM is a significant factor of bias. Assessment of the exposure is conducted using different methods: surface air pollutant monitors, satellite sensors, chemical-weather models and combination of them. Smoky days are often defined by the occurrence of fire events and additional criteria such as smoke predictions models, PM concentrations and threshold values. A common methodology should be developed for both to identify and determine BB derived PM and to calculate personal exposure to BB emissions.

A lot of studies were available for forest fires, bush fires and prescribed fires (especially from Australia), and less on agricultural and residential BB (studies mostly from US). For example, we found only one study that investigated the health outcomes due to outdoor exposure to BB for domestic heating in Europe (Sarigiannis et al., 2015). We also observed the lack of epidemiological studies in Africa were BB emissions due to Savanna fires have high contribution in regional and also global scale (van der Werf et al., 2010).

PM10 and PM2.5 were indistinctively used in epidemiological studies for BB-PM. The use of PM2.5 is probably more adequate for source apportionment analysis because most of the PM10 mass from BB emissions falls in the PM2.5 fraction (Reid et al., 2005), and the interference of crustal/mineral sources on levels of K (a major tracer of BB emissions) is diminished in PM2.5.

We greatly recommend producing a guideline document to propose tools to harmonise BB source apportionment of ambient PM levels, but also to design the evaluation of health outcomes. Multi-cities studies using harmonized strategies for monitoring, exposure and epidemiology, including the evaluation of the different BB source types should be promoted.

Declaration of competing interest

The authors declare no conflict of interests.

Acknowledgements

The systematic review was funded by WHO as part of a Grant Agreement with Ministry of Foreign Affairs, Norway. Angeliki Karanasiou

acknowledges support received through the Ramón y Cajal program (grant RYC-2014-16885) of the Spanish Ministry of Science, Innovation and Universities. This work was supported by the Spanish Ministry of Science and Innovation (Excelencia Severo Ochoa, Project CEX2018-000794-S).

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.scitotenv.2021.146739.

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