



Coal mine fires and human health: What do we know?



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ABSTRACT

Coal mine fires are insidious, persistent, and as widespread as the occurrence of coal itself, yet their potential adverse human health impacts have been poorly characterised. We aimed to summarise the existing literature regarding the health harms associated with coal mine fires and other relevant environmental exposures. We searched the literature for studies of coal mine fires, their emissions, and any aspect of human health. In the absence of health evidence specific to coal mine fires, we included studies of domestic coal combustion and outdoor air pollution from forest fire smoke, for which emission profiles are broadly similar. Coal mine fires cause physical hazards and poor air quality. Proximity to the source of pollution and smouldering combustion typical of coal mine fires increase the risk of community exposure to high concentrations of known toxins such as aerosolised particles, and products of incomplete combustion. Coal mine fire smoke is likely to have short-term adverse respiratory impacts. Adverse cardiovascular outcomes and increased mortality are also plausible depending upon the magnitude of exposure and the number of people affected. There is insufficient evidence to determine the likelihood of other health outcomes. There are major gaps in the available evidence for health outcomes associated with exposure to poor air quality for time periods of weeks to months. The incomplete evidence base hampers actions to mitigate harms in a timely, scientifically-informed manner. The need to further understand the health impacts of coal mine fires is pressing, particularly as they disproportionately affect vulnerable and disadvantaged communities and are likely to become more frequent and severe as a consequence of climate change. Crown Copyright © 2015 Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

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Abbreviations: CO, Carbon monoxide; CO₂, Carbon dioxide; COPD, chronic obstructive pulmonary disease; JCF, Jharia Coal Fields; km, kilometre; PAH, polycyclic aromatic hydrocarbon; PM, Particulate Matter; PM₁₀, Particulate Matter with aerodynamic diameter less than 10 µm; PM_{2.5}, Particulate Matter with aerodynamic diameter less than 2.5 µm; PPB, parts per billion; PPM, parts per million; USA, United States of America.

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1. Introduction

Fires in subterranean coal deposits are a natural phenomenon, and many have been burning for millennia, including Australia's Burning Mountain and Powder River Basin in the United States of America (USA) (Heffern and Coates, 2004; Krajick, 2005; Stracher et al., 2011). Coal mine fires are widespread, and currently thousands are burning

throughout the world, especially in India, China, and the USA (Fig. 1) (Stracher, 2007; Stracher and Taylor, 2004). The number of coal fires has increased dramatically since the Industrial Revolution as a result of human activity such as mining, land clearing, and anthropogenic climate change (Stracher, 2007). Whilst coal fires may originate distant to coal mines, the majority of the available literature, case studies and government reports focus on coal mine fires. The impacts of coal mine fires



Fig. 1. Global distribution of coal mine fires (note: the figure does not indicate the density of coal fires in affected regions and is likely to understate the distribution in developing countries where data and surveillance of coal mine fires is less robust). Adapted from Stracher et al., 2011.



Fig. 2. Densely populated communities reside amongst the JCF fires in Eastern India that have been burning for almost a century.
Photo: Johnny Haglund.

may be considered in health, environmental and economic terms (Finkelman and Stracher, 2010; Stracher, 2007; Stracher and Taylor, 2004). Whilst the environmental and economic harms associated with coal mine fires have been somewhat elucidated, the potential health impacts have received less attention.

The need for evidence to guide the public health response to coal mine fires is clear and has been demonstrated empirically across three global case studies: the Jharia Coal Fields (JCF) in Eastern India, the town of Centralia in Pennsylvania, USA, and the Hazelwood coal mine fire in regional Australia. The JCF fires are an extreme case where fires span 450 kilometer (km)² and have been burning for almost a century in the immediate proximity of densely populated communities (Prakash, 2007; Sarkar et al., 2007) (Fig. 2). Whilst the population level impact of harms resulting from the air pollution liberated from these fires has never been determined, the physical threats are starkly evident in the countless disappearances of individuals and homes through unstable ground over the years (Magnier, 2012; Michalski et al., 1997). To date, efforts to relocate communities nearby the JCF fires have been fragmented and hindered by a lack of clear evidence-based imperative to do so (Magnier, 2012). The Centralia mine fire in Pennsylvania has been burning since 1962. Despite repeated unsuccessful efforts to extinguish the fire, it extended to the coal seam underneath the town, leading to recurrent land subsidence and episodes of CO poisoning (Fig. 3) (DeKok, 1986). Eventually the town was relocated



Fig. 3. Subsidence along the abandoned Route 61 highway due to the underground coal mine fire in Centralia, USA.
Photo: Angela Parriott.



Fig. 4. An aerial view of the smoke plume that covered Morwell and surrounding towns in regional Victoria, Australia during the Hazelwood coal mine fire in 2014.
Photo: Mike Keating.

between 1985 and 1991, with only a few residents remaining today (Krajick, 2005; Stracher and Taylor, 2004). The US government has since been heavily criticised for the lack of decisive, timely action for the residents of Centralia (DeKok, 1986). A more recent case study is that of the 2014 Hazelwood coal mine fire in Australia, which lasted six weeks and caused unprecedented episodes of air pollution in nearby towns (Fig. 4). As outlined by a parliamentary enquiry into the response, the lack of available evidence outlining the short- and long-term harms associated with coal fire smoke exposure considerably hindered the public health response to the Hazelwood mine fire (Parliament of Victoria, 2014).

The public health importance of air pollution is well established and is supported by robust evidence concerning the health harms associated with ambient particulate air pollution and source-specific indoor and outdoor air pollution, such as that resulting from forest and peat fires and domestic coal combustion (Lim et al., 2013; Liu et al., 2015; Pope and Dockery, 2006; Zhang and Smith, 2007). The health harms associated with coal mine fires have received comparatively less attention. In light of the global distribution of coal mine fires, their increasing occurrence and their predominance in impoverished and disadvantaged communities, there is a clear need and opportunity for further research to guide decision makers in mitigating health harms at a population level. A very similar spectrum of toxic components to those described for coal fires has also been described for other forms of indoor and outdoor solid fuel combustion including forest fire, bushfire, and peat fire smoke, and from indoor domestic coal fires (Alves et al., 2011; Betha et al., 2013; De Vos et al., 2009; Melendez-Perez et al., 2014). It is therefore plausible that the health harms associated with coal mine fires may be comparable to those observed with forest fire smoke and domestic coal combustion, if the various exposure dynamics are accounted for, including the substrate consumed, combustion conditions, the varying concentration of potentially toxic trace elements in the substrate, the duration and intensity of exposure, and the characteristics of the population exposed. Below we review and summarise the available evidence concerning the health impacts associated with coal mine fires, highlight gaps in the evidence, and draw upon findings of the health impacts from comparable exposures.

2. Method

We initially searched the literature for the highest level of evidence available about the characteristics of coal fire emissions and impacts of coal mine fires on any aspect of human health and wellbeing, including publicly available university and government reports. Due to the paucity of health evidence specific to coal mine fire smoke, we expanded the search criteria to include indoor and outdoor smoke from the

domestic use of coal and outdoor air pollution from forest and peat fires. Although there is large variation in the intensity and duration of exposure within and between these sources, the composition of smoke demonstrates a similar spectrum of chemical compounds to coal mine fires as the process of incomplete combustion of a solid, hydrocarbon-based fuel source is common to these sources. Emissions include carbon dioxide (CO₂); Particulate Matter (PM); products of incomplete combustion including hydrocarbons, aldehydes and polycyclic aromatic hydrocarbons (PAH); and trace elements such as sulphur, arsenic, lead, and mercury (Alves et al., 2011; Betha et al., 2013; Finley et al., 2009; Sigler et al., 2003; Zhang et al., 2008).

2.1. Search strategy

We searched peer-reviewed articles using PubMed, Cochrane Library, ScienceDirect, Web of Science, GreenFILE, ProQuest, and Scopus electronic databases. Searches were conducted from March to July 2015. The following search terms were used: 'coal mine fire', 'coal fire', 'coal combustion' and any of the following: 'health', 'morbidity', 'hospital', 'respiratory', 'asthma', 'cardiac', 'cardiovascular', or 'mortality.' In the absence of literature pertaining specifically to coal mine fires, the following search terms were applied: 'domestic coal' or 'forest fire'; 'wildfire' or 'bushfire'; and any of the following: 'health', 'morbidity', 'hospital', 'respiratory', 'asthma', 'cardiac', 'cardiovascular', or 'mortality.' The title and abstracts of retrieved papers were reviewed and the exclusion criteria applied. A 'snowballing' technique was employed, whereby relevant citations listed in review papers were followed up. Additionally, a supplementary search of key authors in the field of coal fires was conducted.

Publications were excluded if they were not available in full-text, if they assessed exposure only and not the associated health outcome, or were not available in English. No date limitation of published work was applied. Retrieved publications were stored in an EndNote Library. Duplications were identified by manual search and discarded. Studies were summarised based on study setting; design; exposure characterisation; and outcome assessment, key findings, and strength of association if available. Limitations of studies, including likelihood of bias, were considered.

3. Results

We found 193 studies and 68 were discarded because they did not meet the inclusion criteria. Of the 125 studies included in this review, 26 were descriptive case studies of coal mine fires, two related to exposure to coal-fired boilers, 43 to domestic coal use, and 54 to forest fire smoke exposure. In the absence of peer-reviewed literature, 12 reports from grey literature were included. Grey literature refers to reports from universities, government, and other reputable organisations produced external to academic and commercial channels. The vast majority of coal mine fire research addressed the composition, toxicology and epidemiology of smoke exposure. A small number of papers addressed physical injuries.

3.1. Components of outdoor smoke and their known health impacts

Pollutants generated by coal combustion are similar to that generated from domestic solid fuel combustion and outdoor biomass fires. Pollutants may be broadly categorised as gases, PM, volatile organic compounds, and trace elements and many are known to be deleterious to human health, such as CO, PAHs, benzene, arsenic, fluorine, mercury, selenium, formaldehyde, nitrogen and sulphur-based compounds, and particles with an aerodynamic diameter less than 10 µm (PM₁₀) (Table 1) (Betha et al., 2013; De Vos et al., 2009; Ezzati and Kammen, 2002; Finkelman, 2004; Finkelman and Stracher, 2010; Hower et al., 2009; Melendez-Perez et al., 2014; Stracher, 2007; Stracher and Taylor, 2004; Zhang et al., 2008).

PM is the most important component of outdoor smoke for which there is a large evidence base that outlines the detrimental population health impacts associated with PM exposure. In particular, fine particulate matter with an aerodynamic diameter less than 2.5 µm (PM_{2.5}) are able to penetrate deeply within the alveoli and are thought to have the greatest role in affecting human health, in particular respiratory and cardiovascular morbidity (Pope and Dockery, 2006).

3.2. Coal mine fires and their emissions

Producing a detailed description of the emissions generated by coal mine fires is problematic for many reasons. First, the composition of coal, and the pollutants liberated during combustion, varies geographically (Finkelman, 2004; Finkelman and Gross, 1999). Second, the nature of coal fire emissions varies with the nature of combustion (Zhang et al., 2008). Coal mine fires tend to burn less efficiently and for longer periods than a coal-burning power plant, resulting in the release of a wide range of partially oxidised by-products including benzene, toluene, and xylene. Finally, the emission profile of coal combustion varies spatially and temporally. Modelling the manner in which emissions flux through soil and overburden into the ambient air, along with the meteorological conditions that impact this, is challenging and further sophistication of models is required to determine the potential exposure of communities residing nearby coal mine fires (Engle et al., 2013; O'Keefe et al., 2010).

There have been few studies that have characterised the emissions generated from coal mine fires, the majority of which are abandoned coal mines in Kentucky and Wyoming, USA (Engle et al., 2012; Hower et al., 2009; Hower et al., 2011; Hower et al., 2013; O'Keefe et al., 2010; O'Keefe et al., 2011). These studies have characterised up to 62 different compounds in the coal fire emissions (Hower et al., 2013). The concentration of emissions at vent surfaces varies substantially for all compounds that are potentially harmful to human health. Maximal concentrations recorded at vent surfaces across studies for the following compounds is as follows: CO (>27 000 parts per million (ppm)), mercury (>2100 µg/m³), benzene (>400 parts per billion (ppb)), toluene (>397 ppb) and, greenhouse gas CO₂ (>17% v/v) (Engle et al., 2012; Hower et al., 2009; Hower et al., 2011; Hower et al., 2013; O'Keefe et al., 2011; Pone et al., 2007). Furthermore, study of the soot and sublimates associated with these coal mine fires demonstrates the presence of a range of nano- and ultrafine minerals including harmful trace elements such as selenium, arsenic, and mercury (Pone et al., 2007; Silva et al., 2011; Silva et al., 2012). At the global scale it has been estimated that CO₂ emissions resulting from coal mine fires vary from 12 kg CO₂e yr⁻¹ m⁻² to 8200 kg CO₂e yr⁻¹ m⁻² and that the annual CO₂ emissions from coal fires in the US is approximately 1.4 × 10⁷ to 2.9 × 10⁸ tonne per year (Carras et al., 2009; O'Keefe et al., 2010). However, further sophistication of modelling is required to fully appreciate the contribution of coal fires to greenhouse gas emissions and other atmospheric pollutants (Engle et al., 2013; Engle et al., 2011).

Extrapolating the impact of these documented emissions on human health is difficult, as these emissions vary significantly with the intensity of spontaneous combustion and with meteorological conditions, in particular temperature (Carras et al., 2009; Hower et al., 2009; Hower et al., 2011; Hower et al., 2013). Additionally, many of these emissions are documented at surface vents of fires distant from communities, and as such understanding the probable exposure to communities or residents and the associated health impacts is challenging. The relevance of these emissions on human health will depend upon the nature in which emissions flux from coal fire vents, the proximity of exposed communities, as well as the duration of exposure. Despite these limitations, available studies support that coal fires are a source of locally dangerous levels of CO, benzene, mercury, and other gases, as well as a significant contributor to greenhouse gas emissions, which may indirectly impact health through climate change (Carras et al., 2009; Department of the Environment and Heritage, 2015; Engle et al., 2011; O'Keefe et al., 2010; The WHO European Centre for Environment and Health, 2010).

Table 1

Major constituents of smoke from biomass, peat and coal combustion and associated health impacts.

Adapted from Naeher et al. (2007), Agency for Toxic Substances and Disease Registry (2007), (2012), Bencko et al. (1980), Bolling et al. (2009), Carras et al. (2009), Chen et al. (2014), Finkelman and Gross (1999), Johnston and Bowman (2014), Liu et al. (2015), Melendez-Perez et al. (2014), Mirza (2003), Pope and Dockery (2006), Reid JS et al. (2005), Saarnio et al. (2010), Youssef et al. (2014), and Zhang and Smith (2007).

Constituent	Health impacts
Particulate matter	Particulate matter primarily comprises organic and elemental carbon components along with smaller contributions from inorganic species. Smaller sized particles ($PM_{10, 2.5}$) are able to reach the alveoli and have the greatest potential to cause cardiovascular and respiratory harm. There is substantial evidence that particulate matter is associated with a wide range of adverse health outcomes, in particular all-cause mortality, cardiovascular and respiratory morbidity, inflammation, oxidative stress, and pro-coagulation.
Inorganic acids Carbon monoxide	Carbon monoxide is produced through incomplete combustion produced more abundantly from smouldering than flaming combustion. Carbon monoxide is a neurotoxin that impairs the oxygen carrying capability of haemoglobin. Symptoms and signs of acute carbon monoxide poisoning include headache, nausea, weakness, mental confusion, visual disturbances, chest pain, dyspnea, palpitations, cardiac arrhythmias, myocardial ischemia, pulmonary edema, seizures, loss of consciousness, coma and death. Exposure to carbon monoxide below the concentration at which poisoning occurs is associated with exacerbations of asthma in children, increased risk of congestive cardiac failure, ischemic heart disease, myocardial infarction, stroke, neurological impairment, and cognitive changes
Ozone	Ozone is formed photo-chemically near the top of smoke plumes in sunlight conditions. Ozone is a secondary pollutant formed chemically from the cycling of nitrogen oxide species in the atmosphere. It is more likely to be an important co-pollutant at locations distant from, rather than adjacent to, the source of combustion. Ozone is associated with exacerbations of respiratory diseases.
Nitrogen and sulphur-based compounds	Both nitrogen and sulphur-based compounds are produced in proportion to their content in the burning substrate and the combustion efficiency of the fire. Smouldering combustion tends to produce reduced nitrogen compounds such as ammonia, whereas flaming combustion produces oxides of nitrogen. These compounds are respiratory irritants.
Carbon dioxide, methane	Greenhouse gases including carbon dioxide and methane contribute to climate change. Indirectly, climate change is associated with an increased occurrence of malnutrition and nutritional-related diseases as a result of food insecurity, infectious disease outbreaks and illness, and injury secondary to extreme weather events.
Hydrocarbons Examples Polycyclic aromatic hydrocarbons – such as benzo(a)pyrene; benzene	Produced by incomplete combustion. These may be saturated, unsaturated, monoaromatic, or polycyclic aromatics. Some, such as benzo(a)pyrene, are mutagenic and carcinogenic. Butadiene, an unsaturated hydrocarbon is an irritant and neurotoxic. This group includes semi-volatile and volatile organic compounds such as benzene, naphthalene, and toluene. They are respiratory tract irritants. Benzene and naphthalene are classified as carcinogens. Acute toxic exposure to benzene (concentrations of 10 000–20 000 ppm) may be fatal. Exposure to lower concentrations of benzene (700–3000 ppm) have been documented to cause drowsiness, dizziness, tachycardia, headaches, tremor, arrhythmias, confusion, and unconsciousness
Oxygenated organic molecules Aldehydes	Some aldehydes such as acrolein are extremely irritating to mucous membranes of the human body. Others, such as formaldehyde, are carcinogenic. Some reduce the ability of scavenger cells in the lungs to engulf foreign bacteria.
Organic alcohols and acids Phenols Quinones	These include methanol and acetic acid, which are irritants and are teratogenic. Examples include catechol and cresol. These are known to be irritants, mutagenic, carcinogenic, and teratogenic. Quinones such as hydroxyquinone are irritants, allergenic, cause oxidative stress and inflammation, and are possibly carcinogenic.
Free radicals	Free radicals, such as semiquinones, are abundantly produced but most undergo condensation within a few seconds. Some may persist for up to 20 min and some may remain in organic material. They cause oxidative stress, inflammation, and are possibly carcinogenic.
Trace elements	Trace elements may occur in gaseous form or be attached to particulate matter. Health harms arising from trace elements liberated during coal combustion have been well documented for arsenic, beryllium, fluorine, selenium, and mercury. Other trace elements liberated during coal combustion which may be harmful to human health include boron, chromium, vanadium, manganese, nickel, copper, zinc, aluminium, germanium, lead, molybdenum, cadmium, antimony, tin, and thallium
Arsenic Beryllium Fluorine Mercury Selenium	Skin lesions; gastrointestinal illness: neurotoxicity; nephrotoxicity; bladder, lung, and skin cancer Immune dysfunction, respiratory disease, skin disease, and lymphatic/haematological illness Dental and skeletal fluorosis Neurotoxic, lung and prostate cancer Hair and nail loss, paraesthesia, nausea, and dizziness

Looking at the global case studies, the data outlining emissions associated with the JCF fires in India and Centralia mine fire in the USA are limited. A single study exploring the air quality surrounding the JCF in India, reported that the 24-hour average concentration of PM_{10} was as high as $780 \mu\text{g}/\text{m}^3$ in work areas and $170 \mu\text{g}/\text{m}^3$ in the ambient air (Ghose and Majee, 2007). These values considerably exceed the World Health Organisation standards of $50 \mu\text{g}/\text{m}^3$ per day (World Health Organisation, 2005). Field studies of the Centralia mine fire in the USA have reported varied CO levels between 2200–7500 ppm measured near vents and boreholes (Department of Environmental Protection Pennsylvania, 2015; Stracher et al., 2004). There does not appear to be any publically available data outlining the concentrations of PM_{10} or $PM_{2.5}$ in Centralia. Although 24-hour total suspended particulate concentrations, which are a relatively poor surrogate of PM_{10} concentrations that are not regulated, have been recorded in the range of 7–

$39 \mu\text{g}/\text{m}^3$ (Department of Environmental Protection Pennsylvania, 2015).

The only publically available data concerning community exposure to coal mine fire emissions is that of the 2014 Hazelwood coal mine fire in Victoria, Australia. The fire caused severe episodes of air pollution in Morwell, a town of 14 000 people, where most residential areas were situated within 5 km of the burning coal face, some being as close as 500 m (Fig. 4) (Australian Bureau of Statistics, 2013b). Concentrations in excess of Australian and international air quality guidelines for $PM_{2.5}$, carbon monoxide (CO), PAH (benzo(a)pyrene), and benzene were recorded in residential areas during the fire period. $PM_{2.5}$ exceeded 24-hour average guidelines of $25 \mu\text{g}/\text{m}^3$ on 21 days during the fire, with the highest peak recorded at $800 \mu\text{g}/\text{m}^3$ (Environment Protection Authority Victoria, 2015). CO levels exceeded 8-hour average guidelines of 9 ppm on seven days during the early weeks of the fire

while benzene measurements exceeded the United States Agency for Toxic Substances and Disease Registry 24-hour average standard of 9 ppb on three occasions, with measurements ranging from 9.2–14 ppb (Parliament of Victoria, 2014). Peaks in the concentration of benzo[a]pyrene during the coal mine fire period resulted in the annual guideline for PAHs (as benzo[a]pyrene equivalents) of 0.3 ng/m³ to be breached by 0.1 ng/m³. Ozone, nitric dioxide, sulphur dioxide, and trace metal concentrations remained within regulatory guidelines (Environment Protection Authority Victoria, 2015). Moderate elevations of PM_{2.5} were also documented in towns up to 15 km away in the surrounding valley, an area inhabited by an additional 58 000 people (Australian Bureau of Statistics, 2013a, 2013b; Environment Protection Authority Victoria, 2015).

3.3. Evaluating the health impacts associated with coal mine fires – the challenges

The existing evidence exploring coal mine fires and their associated impacts, in particular health impacts, is extremely limited. For this reason, we draw upon evidence outlining the health impacts associated with comparable exposures for which the spectrum of emissions is broadly similar – indoor air pollution from domestic coal use and outdoor air pollution from forest and peat fires. A very similar spectrum of toxic components to those described above for coal fires, has also been described for forest fire, bushfire, and peat fire smoke, and from indoor domestic coal fires. For example, coal, peat, and forest fires can all liberate metals derived from the soil and this will vary by location (Betha et al., 2013; Melendez-Perez et al., 2014). Further, as inefficient combustion occurs in all these settings, a similar suite of products of incomplete combustion is generated (Table 1). Domestic coal combustion is useful as a comparable exposure as the substrate is the most similar to that burned in outdoor coal fires. However, indoor domestic exposure is likely to be of greater magnitude and is usually chronic in nature. Outdoor exposures from forest fires are more comparable to coal mine fires in terms of duration and intensity of the smoke event. However, there is huge variation between individual fires. Forest fire emissions vary depending on the substrate burned (wood, leaves, peat), the conditions of combustion, such as oxygen availability and fuel moisture, and the distance from the location of the fire as many components continue to undergo further chemical reactions after generation (Betha et al., 2013; De Vos et al., 2009; Melendez-Perez et al., 2014).

Despite the merits in drawing upon these analogous exposures, we recognise that characterising the population health impacts following an air pollution event will vary based on a number of variables, including the size and characteristics of the population exposed, local health infrastructure, the geochemistry of the substrate consumed, the combustion conditions of the fire, the duration and intensity of exposure. The air pollution pyramid provides a conceptual framework of the relative order and severity of health impacts following population level exposure to air pollution (Fig. 5). In the absence of coal mine fire-specific literature, we feel that it is appropriate to draw upon these comparable exposures to highlight the existing gaps in data, as well to extrapolate the possible health impacts associated with coal mine fires to inform decision makers in the intervening period until coal mine fire-specific epidemiological findings are available. Individual studies exploring the health impacts associated with domestic coal combustion and forest fire smoke exposure are presented in Tables 2 and 3 respectively. Below, we summarise the range of health impacts reported in the literature, from symptoms of illness to morbidity and mortality associated with exposure to fire smoke.

3.4. Symptoms of illness

3.4.1. Evidence from coal mine fires

There are no peer-reviewed publications specifically assessing the individual or public health impacts associated with air pollution liberated from coal mine fires. However, some non-peer-reviewed government and university reports evaluating health impacts of the 2014 Australian Hazelwood coal mine fire are available (Barnett, 2014; Brook, 2014; Flander and English, 2014; Parliament of Victoria, 2014). As a part of a parliamentary inquiry into the circumstances of the fire, the state health department submitted an evaluation of impacts on health services during the period of the fire (Brook, 2014). This report described a substantial increase in utilisation of a nurse-led telephone health advisory service and in visits to general practices and other primary care services in the region during the fire period. Conditions for which there was an increased consultation demand included irritation of the eyes and throat; respiratory symptoms such as coughing; anxiety related to the smoke and ash; and a range of other concerns including headaches, nausea or vomiting, blurred vision, and requests for CO testing (Brook, 2014).

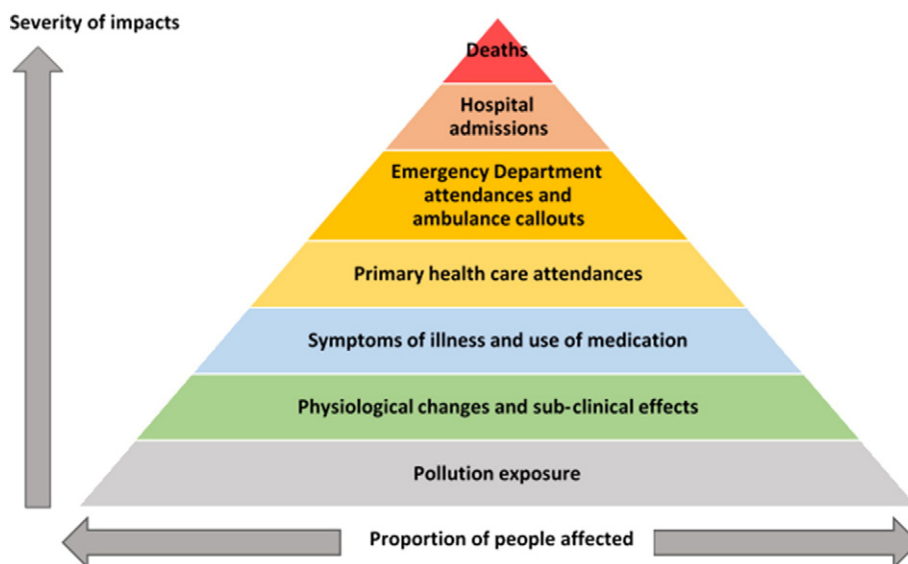


Fig. 5. The air pollution pyramid is a framework commonly used to describe the spectrum of health impacts from exposure to air pollution. It illustrates the inverse relationship between the severity of outcomes and the proportion of people affected by them.

Table 2

Summary of studies exploring the association between health outcomes and domestic coal combustion in China.
Format adapted from Liu et al. (2015).

Health outcome	Total number of studies	Number of studies that observed a positive association	Number of studies that did not observe a positive association	Studies that observed a positive association	Studies that did not observe a positive association
Fluorosis	7	7	0	Ando et al. (1998), Chen et al. (1993), Dai et al. (2007), Luo et al. (2011), Watanabe et al. (2000), Wu et al. (2004), and Zhang and Cao (1996)	–
Arsenosis	2	2	0	Shraim et al. (2003), and Zheng et al. (2005)	–
Respiratory symptoms	4	4	0	Liu et al. (2013), Pope and Xu (1993), Qian et al. (2004), and Salo et al. (2004)	–
Asthma and chronic obstructive pulmonary disease	2	2	0	Tao et al. (1992), and Zheng et al. (2002)	–
Reduced lung function	2	2	0	Tao et al. (1992), and Xu et al. (1991)	–
Lung cancer	5	4	1	Dai et al. (1996), Kleinerman et al. (2002), Liu et al. (1991), and Luo et al. (1996)	Ko et al. (1997)
Esophageal cancer	1	1	0	Pan et al. (1999)	–
Delayed childhood development	1	1	0	Tang et al. (2008)	–

3.4.2. Evidence from analogous exposures

Similar symptoms have been reported following exposure to indoor air pollution from domestic coal use (Liu et al., 2013; Qian et al., 2004; Salo et al., 2004) and forest fire smoke (Kolbe and Gilchrist, 2009; Kunii et al., 2002; Shusterman et al., 1993).

3.4.3. Conclusion

The constituents of smoke are known to be irritants to mucosal surfaces and, as such, it is biologically plausible that similar symptoms are

evident following coal mine fire smoke exposure compared to other sources of air pollution (Naeher et al., 2007).

3.5. Carbon monoxide poisoning

3.5.1. Evidence from coal mine fires

There are no peer-reviewed studies exploring the association between exposure to coal mine fire smoke and CO poisoning. However, coal fires have been demonstrated to produce significant CO emissions.

Table 3

Summary of studies exploring the association between health outcomes and forest fire smoke exposure.
Format adapted from Liu et al. (2015).

Health outcome	Total number of studies	Number of studies that observed a positive association	Number of studies that did not observe a positive association	Studies that observed a positive association	Studies that did not observe a positive association
Reduced birth weight	2	1	1	Holstius et al. (2012)	Prass et al. (2012)
Asthma	12	11	1	Delfino et al. (2008), Dohrenwend et al. (2013), Duclos et al. (1990), Henderson et al. (2011), Johnston et al. (2002), Johnston et al. (2007), Johnston et al. (2006), Martin et al. (2013), Morgan et al. (2010), Rappold et al. (2011), and Viswanathan et al. (2006)	Smith et al. (1996)
Other respiratory conditions	26	25	1	Chen et al. (2006), Dohrenwend et al. (2013), Duclos et al. (1990), Emmanuel (2000), Hanigan et al. (2008), Henderson et al. (2011), Ignotti et al. (2010), Johnston et al. (2007), Kolbe and Gilchrist (2009), Kunii et al. (2002), Kunzli et al. (2006), Lee et al. (2009), de Mendonca et al. (2006), Mirabelli et al. (2009), Moore et al. (2006), Morgan et al. (2010), Mott et al. (2002), Mott et al. (2005), Rappold et al. (2011), Rappold et al. (2012), Schranz et al. (2010), Shusterman et al. (1993), Tham et al. (2009), Thelen et al. (2013), and Viswanathan et al. (2006)	Azevedo et al. (2010)
Cardiovascular conditions	11	6	5	Azevedo et al. (2010), Haikerwal et al. (2015), Johnston et al. (2007), Lee et al. (2009), ^a Rappold et al. (2011), and Rappold et al. (2012)	Duclos et al. (1990), Hanigan et al. (2008), Henderson et al. (2011), Moore et al. (2006), and Morgan et al. (2010)
Systemic inflammation	2	2	0	Huttunen et al. (2012), and Tan et al. (2000)	–
Medication use (respiratory and anxiolytic-hypnotic)	4	4	0	Caamano-Isorna et al. (2011), Elliott et al. (2013), Johnston et al. (2006), and Vora et al. (2011)	–
Mortality	10	8	2	Analitis et al. (2011), Faustini et al. (2015), Johnston et al. (2011), Morgan et al. (2010), Nunes et al. (2013), Sahani et al. (2014), Sastry (2002), and Shaposhnikov et al. (2014)	Hanninen et al. (2009), and Vedal and Dutton (2006)

^a Inverse association.

Annual CO emissions generated by the Ruth Mullins fire in the USA have been documented at 21 ± 1.8 tonne per annum (O'Keefe et al., 2010). Government reports have demonstrated elevated ambient CO concentrations in association with coal mine fires including the Australian Hazelwood coal mine fire in 2014, during which several firefighters required treatment for CO poisoning (Parliament of Victoria, 2014). A community health assessment centre established during the fire tested 1879 residents for elevated blood carboxyhaemoglobin, a marker of exposure to CO. Of these, 3% ($n = 53$) met the screening criteria for referral to hospital for further assessment for CO exposure. However, information was not provided about their final diagnosis (Brook, 2014). Centralia residents were also subject to empirical reports of repeated episodes of exposure to CO within their homes, with many relying on CO monitors or canaries to monitor this risk (DeKok, 1986). However, the nature of this exposure has not been quantified in any grey or peer-reviewed literature.

3.5.2. Evidence from analogous exposures

While CO exposure is a well-recognised occupational hazard for fire fighters (Reinhardt and Ottmar, 2004), and elevations of CO above accepted ambient guidelines have only occasionally been described in forest and peat fires in community settings (Reisen and Brown, 2006), no studies have reported CO toxicity in residents of communities affected by forest fire smoke. In contrast, CO exposure is a well-established risk of any combustion occurring in conditions of poor ventilation. Indoor coal combustion for example, has been associated with numerous cases of CO poisoning, including fatalities (Zhang and Smith, 2007).

3.5.3. Conclusion

CO usually dissipates quickly in outdoor settings. However, coal mine fires are often subterranean, where burning conditions are relatively anaerobic and likely to favour CO production. Thus, elevated ambient CO exposure in surrounding areas could be a greater risk for non-occupational exposure to coal mine smoke, than for other kinds of landscape fires. This is supported by observations of harmful elevations in atmospheric CO during the Centralia and Hazelwood coal mine fires (DeKok, 1986; Environment Protection Authority Victoria, 2015).

3.6. Toxicity from exposure to trace elements

3.6.1. Evidence from coal mine fires

The concentration of trace elements, such as arsenic and selenium, in coal varies geographically (Finkelman and Gross, 1999). For example, concentrations of up to 2000 mg/kg of fluorine, 35 000 mg/kg of arsenic and 8000 mg/kg of selenium have been documented in certain seams of Chinese coal, which is problematic due to their widespread domestic use (Zheng et al., 1999). To date, there are no studies that explore the health impacts associated with liberation of trace elements resulting from coal mine fires.

3.6.2. Evidence from analogous exposures

Liberation of trace elements such as mercury has been documented in association with forest and peat fires, but no studies have explored potential health impacts associated with this (Betha et al., 2013). However, commercial combustion of coal enriched in beryllium and arsenic has been demonstrated to cause immune dysfunction in exposed individuals (Bencko et al., 1980; Bencko et al., 1988). Domestic coal combustion in China has been associated with endemics of arsenosis, fluorosis, and selenosis, owing, in part, to the enrichment of much of China's surface coal in these trace elements (Dai et al., 2012; Finkelman et al., 1999). In epidemiological studies fluorosis, which causes skeletal and dental dysplasia, has been linked with domestic coal combustion with the association being influenced by the fluorine-content of the clay used as a coal-burning additive and in briquette-making, method and duration of burning, and the ventilation of the home (Ando et al., 1998; Chen et al., 2014; Chen et al., 1993; Dai et al., 2007; Dai et al.,

2012; Dai et al., 2004; Luo et al., 2011; Watanabe et al., 2000; Wu et al., 2004; Zhang and Cao, 1996; Zheng et al., 1999; Zheng et al., 2007). Indoor combustion of coal with arsenic concentrations in excess of 100 mg/kg has been associated with symptoms of arsenosis, affecting the central nervous system, digestive system, and skin (Chen et al., 2014; Dai et al., 2012; Liu et al., 2002; Shraim et al., 2003; Zheng et al., 1999; Zheng et al., 2005). Deforestation and exposure of selenium-rich coal in the 1960s, which was subsequently exploited by local residents for domestic use, has been implicated in selenosis endemics. Characteristic selenosis symptoms, include hair and nail loss, paraesthesia, nausea, and dizziness (Chen et al., 2014; Zheng et al., 1999).

Ingestion of food that has been dried over open fire pits containing coal enriched in toxic trace elements is an important pathway of exposure and could be responsible for greater individual intake than directly inhaling polluted air (Chaoke et al., 1997; Dai et al., 2007; Dai et al., 2012; Dai et al., 2004; Finkelman et al., 1999; Luo et al., 2011; Zhang and Smith, 2007; Zheng et al., 2007). Selenosis is also linked to the use of coal ash as a fertiliser and subsequent consumption of selenium-rich crops (Chen et al., 2014; Dai et al., 2012).

3.6.3. Conclusion

Toxicity from trace elements released by the domestic combustion of coal have been measured in settings where coal is enriched in the elements of concern, individual exposure is chronic, and occurs through multiple pathways in addition to inhalation of polluted air. While these conditions will not necessarily apply to many coal mine fires, there are examples, such as the JCF fires in India, where large residential communities continue to live in close proximity to indefinitely burning mine fires and experience chronic exposure to coal smoke (Fig. 2). Risk of toxicity from trace elements should be considered in situations where the coal deposit is enriched in elements of concern, and exposure to the fire emissions is ongoing.

3.7. Respiratory morbidity

3.7.1. Evidence from coal mine fires

Peer-reviewed literature exploring this association following coal mine fire exposure is lacking.

3.7.2. Evidence from analogous exposures

The vast literature on indoor and outdoor particulate air pollution from all sources clearly demonstrates adverse impacts on the development and functioning of the respiratory system in children and adults. This includes the development of new cases of lung disease as well as exacerbation of existing conditions (Dockery and Ware, 2015; Liu et al., 2015; Upadhyay et al., 2014). Additionally, the evidence concerning indoor pollution from domestic coal combustion and respiratory morbidity is strong (Table 2). In adults, exposure has been associated with a range of adverse respiratory outcomes, including increased likelihood of respiratory symptoms, impaired lung function, and respiratory conditions such as chronic obstructive pulmonary disease (COPD) (Pope and Xu, 1993; Tao et al., 1992; Xu et al., 1991). Children are particularly vulnerable to the health effects associated with exposure to domestic coal combustion. These include an increased risk of developing a range of respiratory symptoms, infections, and asthma (Liu et al., 2013; Qian et al., 2004; Salo et al., 2004; Smith et al., 2000; Zheng et al., 2002).

Similarly, the evidence surrounding the impact of forest fire smoke on adverse respiratory health is robust, particularly for asthma which has been the subject of considerable research (Table 3). Studies have consistently reported associations between exposure to forest fire smoke and exacerbations of asthma, as measured through onset of symptoms, medication use, Emergency Department presentations, and hospital admissions (Delfino et al., 2008; Dohrenwend et al., 2013; Duclos et al., 1990; Henderson et al., 2011; Johnston et al., 2002; Johnston et al., 2006; Martin et al., 2013; Morgan et al., 2010; Vora et al., 2011).

The association between forest fire smoke exposure and respiratory outcomes apart from asthma is also well established, including increased respiratory symptoms, increased use of respiratory medication, increased likelihood of physician and Emergency Department visits, and hospital admissions for respiratory disease. Diseases associated with short-term increases in fire smoke exposure, include pneumonia, acute bronchitis, and COPD (Caamano-Isorna et al., 2011; Chen et al., 2006; De Mendonça et al., 2006; Elliott et al., 2013; Emmanuel, 2000; Henderson et al., 2011; Lee et al., 2009; Martin et al., 2013; Moore et al., 2006; Rappold et al., 2011; Schranz et al., 2010; Tham et al., 2009; Thelen et al., 2013; Visawanathan et al., 2006). Those with a history of asthma, or pre-existing cardiopulmonary disease, are more likely to report symptoms (Kolbe and Gilchrist, 2009; Kunii et al., 2002; Kunzli et al., 2006; Mirabelli et al., 2009; Mott et al., 2005; Mott et al., 2002).

3.7.3. Conclusion

The evidence base supporting an association between exposure to both indoor air pollution from domestic coal combustion and outdoor air pollution from forest fire smoke is strong. It is therefore very likely that exposure to coal mine fire smoke would be associated with respiratory morbidity at the population level, particularly in susceptible individuals.

3.8. Cardiovascular morbidity

3.8.1. Evidence from coal mine fires

There are no studies exploring the impact of air pollution resulting from coal mine fires on cardiovascular morbidity.

3.8.2. Evidence from analogous exposures

The wider literature concerning exposure to outdoor particulate air pollution from any source conclusively demonstrates both short and long-term associations with heart disease including sub-clinical changes in autonomic function, admissions to hospital for dysrhythmias, heart failure, ischaemic heart disease, and cardiovascular mortality (Pope and Dockery, 2006). Whilst there are no studies exploring the association between domestic coal combustion and cardiovascular morbidity, the literature exploring this association following episodic forest fire smoke exposure reports mixed results. The finding of an association appears to vary with the exposure metric used (Azevedo et al., 2010), the subpopulation examined (Azevedo et al., 2010; Johnston et al., 2007), and the cardiovascular endpoint measured. Cardiac arrest, ischaemic heart disease, and heart failure appear to be the outcomes most commonly reported to be associated with episodic fire smoke pollution events (Dennekamp et al., 2015; Haikerwal et al., 2015; Rappold et al., 2012; Rappold et al., 2011). However, publication bias is likely to be a factor in all fire smoke studies and contribute to an under-estimation of the true association between exposure and outcome, but we cannot evaluate the size of this effect.

3.8.3. Conclusion

While studies of the specific setting of coal mine fires are lacking, there is strong evidence from the wider literature that short and long-term exposure to elevated concentrations of PM is associated with worsening of heart disease. It is plausible that coal mine fire emission could contribute to worsening cardiovascular outcomes. However, the size of the impacts can be small and not always measurable in small populations.

3.9. Immune system impairment

3.9.1. Evidence from coal mine fires

There are no published studies that have explored the association between coal mine fire smoke exposure and immune system impairment.

3.9.2. Evidence from analogous exposures

Whilst the wider PM literature demonstrates an association between exposure to fine PM and systemic inflammation, few studies have explored the association between domestic coal combustion and immune function and tend to be either relatively dated or written in languages other than English. Zhang et al. (2007) summarises that six studies have demonstrated that domestic coal smoke exposure is associated with reductions in serum immunoglobulin G content, peripheral T-lymphocyte activity, interleukin-2 induction activity, and natural killer cell activity. This suggests that domestic coal smoke exposure is associated with an increased risk of developing infections and illnesses as a result of immunocompromise (Zhang and Smith, 2007). Indeed, increased respiratory infections have been clearly associated with indoor smoke exposure from the use of solid fuels, including coal (Po et al., 2011; Shen et al., 2009). Additionally, brief exposure to biomass smoke has also been associated with short-term changes in inflammatory markers (Huttunen et al., 2012; Tan et al., 2000).

3.9.3. Conclusion

There is evidence of systemic inflammation following brief biomass and domestic coal exposure and this is a plausible outcome from coal fire smoke exposure. However, caution is needed as the relative exposure from indoor coal smoke exposure could be far greater than outdoor smoke exposure, for which the evidence based is much more limited.

3.10. Perinatal outcomes

3.10.1. Evidence from coal mine fires

To date, there are no studies exploring the impact of coal mine fire smoke exposure in utero on perinatal and early childhood outcomes.

3.10.2. Evidence from analogous exposures

Exposure to ambient air pollution from commercial coal combustion has been associated with an increase in umbilical PAH-DNA adduct levels (a marker of ambient exposure to PAHs) and decrements in one or more developmental quotients (Tang et al., 2008). Perinatal exposure to forest and peat fire smoke has been demonstrated to be significantly associated with an increased likelihood of perinatal and childhood mortality, along with reductions in birth weight following second and third trimester exposure (Holstius et al., 2012; Jayachandran, 2009). A single study found no significant association between in utero exposure to forest fires (as determined by number of hotspots per region) and birth weight (Prass et al., 2012). The relationship between the number of satellite hotspots and smoke exposure, as measured by Prass et al. (2012), is not known. Additionally, this study did not address seasonal variation of birth weight and is likely to be subject to bias.

3.10.3. Conclusion

While studies of outdoor air pollution suggested that adverse perinatal outcomes might plausibly be associated with maternal exposure to coal mine fire smoke, the evidence based is inadequate to comment.

3.11. Cancer

3.11.1. Evidence from coal mine fires

There are no peer-reviewed studies exploring the association between exposure to coal mine fire smoke and malignancy.

3.11.2. Evidence from analogous exposures

Both ambient and indoor air pollution are classified as group 1 carcinogens (International Agency for Research on Cancer, 2006). The association between indoor coal combustion and lung cancer has been demonstrated in a number of studies (Dai et al., 1996; Kleinerman et al., 2002; Liu et al., 1991; Luo et al., 1996). A meta-analysis of the literature exploring the association between indoor air pollution and lung cancer in China by Zhao et al. (2006) confirmed that domestic coal use

for heating and cooking was associated with a two-fold increased risk of lung cancer (Zhao et al., 2006). Individual studies have demonstrated women to be at higher risk of lung cancer secondary to domestic coal use, as a function of increased time spent indoors (Liu et al., 1991). The geographical variation of trace elements in coal utilised for domestic purposes also appears important in the causal pathway. In Xuan Wei County, China, the enrichment of coal in fine silica ($<10\ \mu\text{m}$) has been implicated in the elevated lung cancer mortality observed in non-smoking women in this region. In particular, the combined interaction of silica and volatile organic matter (termed the silica-volatile interaction) has been implicated in the aetiology of lung cancer in this region (Dai et al., 2008; Large et al., 2009).

Although less extensively studied than lung cancer, a nested case-control in China found that any exposure to coal for domestic heating and cooking was associated with a doubling of the risk of esophageal cancer (Pan et al., 1999). Roth et al. (1998) suggested that the aetiology of esophageal cancer may result from contamination of foodstuffs with carcinogen-causing products of incomplete coal combustion in addition to direct respiration of airborne pollutants (Roth et al., 1998).

3.11.3. Conclusion

As cancer is a long-term outcome resulting from the interaction of many genetic and environmental risk factors and long-term exposure to carcinogens, the relevance of the above findings for coal mine fire smoke exposure is not clear.

3.12. Mortality

3.12.1. Evidence from coal mine fires

The association between coal mine fire smoke exposure and mortality has not been examined in any peer-reviewed publications. However, analyses of mortality trends at the time of the Australian Hazelwood coal mine fire (2014) are outlined in two publicly available university reports (Barnett, 2014; Flander and English, 2014). The first of which was a rapid analysis of mortality conducted by Barnett (2014), a university based researcher at the request of a community group. This Bayesian analysis adjusted for long-term trends and seasonal cycles in deaths, and monthly maximum temperature. The main finding was an 82% chance that deaths in the 70 000 people living in the surrounding region, increased by 10% during the fire period (Relative Risk 1.10, 95% Credible Interval 0.89, 1.34) (Barnett, 2014). A second university report by Flander and English (2014) found that during the year of the Hazelwood coal mine fire (2014) there were 9.2 additional monthly deaths than expected for February–March in the region surrounding the fire, based on predictive modelling of mortality data from the previous five years. However, this analysis did not adjust for important covariates such as ambient temperature. Wide confidence intervals around these estimates including the null meant the findings were inconclusive (Flander and English, 2014). Many factors may have contributed to the observed increase in deaths in the region, mine fire smoke being one of these. However, for both analyses an increase in deaths was observed in more distant communities in the region rather than in the town most severely exposed to the smoke. Other contributing factors could have included usual background fluctuations or coincidental hot weather.

3.12.2. Evidence from analogous exposures

The wider literature exploring the effects of PM and domestic coal smoke, and to a lesser extent forest fires, supports an association of increased all-cause and cardiopulmonary mortality, for which no safe threshold of exposure has been determined (Pope and Dockery, 2006). Empirical studies of exposure to domestic coal smoke have demonstrated associations with deaths from pneumonia (Shen et al., 2009) and lung cancer (Mumford et al., 1987). We did not find empirical studies that have examined cardiovascular mortality despite this being the main driver of global modelled estimates of mortality attributable to

domestic solid fuels (Lim et al., 2013). Mortality from outdoor coal smoke resulting from domestic combustion has been evaluated in Europe. In the UK and Ireland, coal was a common source of home heating up until the 1990s and this led to episodes of severe outdoor pollution from coal smoke during the winter months. One of the more famous examples was the London Fog of 1952 in which 12 000 excess deaths due to both acute and persisting effects of the fog occurred (Bell and Davis, 2001). However air quality before, during and after the London episode was considerably worse than that typically documented for coal mine fires. More recently, reductions in mortality have been observed in association with progressive bans of the use of coal for domestic heating in Ireland during the 1990s. Respiratory mortality declined by 17%, 9% and 3% following the 1990, 1995, and 1998 bans respectively (Dockery et al., 2013).

Although there have been fewer studies of the association between forest fire smoke and mortality the evidence is emerging for associations with all-cause (non-trauma) mortality with generally similar effect sizes reported to urban background PM. The evidence for associations with cause-specific (e.g. respiratory or cardiovascular mortality) is emerging but studies are fewer and the results have been less consistent (Analitis et al., 2011; Faustini et al., 2015; Jayachandran, 2009; Johnston et al., 2011; Nunes et al., 2013; Sahani et al., 2014; Sastry, 2002; Shaposhnikov et al., 2014).

3.12.3. Conclusion

The evidence base concerning analogous exposures suggest that increased all-cause mortality following coal mine fire smoke exposure is possible, and will depend upon the duration and magnitude of exposure and the population characteristics of those exposed.

4. Discussion

The existing evidence exploring coal mine fires and their associated impacts, in particular health impacts, is extremely limited. Analogous exposures for which there is a more robust evidence base, including indoor and outdoor pollution from domestic coal combustion and forest fires, suggest that exposure to coal fire smoke exposure is likely to be associated with increased population risks of mortality and respiratory and cardiovascular morbidity, depending upon the size of the population exposed and the magnitude of the exposure. Coal fire smoke has the potential to adversely affect perinatal and childhood development and contribute to an increased risk of lung and esophageal malignancy. Referring to these analogous exposures in the absence of literature specifically exploring the health effects associated with coal mine fires provides some insight into the probable health impacts, but the relative merits and limitations of these exposures must be considered. Public health impacts of coal smoke exposure will vary according to the duration and magnitude of exposure, combustion conditions, the toxic profile of the smoke, and the underlying health status of the people affected.

Domestic coal combustion is the most comparable exposure to coal mine fire smoke. However, anticipating potential health impacts that may result from coal mine fire from domestic coal combustion is problematic as the profile of emissions, duration, and intensity of exposure are likely to vary significantly. The indoor concentration of PM generated from domestic coal combustion is much greater than ambient concentrations and indoor exposure is likely to persist for many years, or even a lifetime (Zhang et al., 2008). Long-term exposure to air pollution has greater health impacts than short-term exposures and as such, health outcomes associated with coal mine fire emissions may be less likely than that for domestic coal combustion, especially for longer term outcomes such as cancer (Dai et al., 1996; Kleinerman et al., 2002; Pope and Dockery, 2006).

The evidence from forest and peat fires is likely to be more comparable in terms of exposure intensity and duration. However, it is difficult to generalise as the duration of human exposure to mine fire emissions

is highly variable. Emissions from some coal mine fires, such as Australia's Hazelwood Mine (2014), are controlled within weeks, while others, such as India's JCF complex, continue to cause chronic human exposure more akin to that of domestic coal use. Although, in forest fires, the substrate is biomass rather than fossilised fuel in the form of coal, the general spectrum of species emitted from forest and peat fires is also likely to be broadly similar as all include products of incomplete combustion such as CO, aerosolised PM, and trace elements.

Additional limitations apply to the included publications, including the heterogeneous methodology of assigning exposure to forest fire smoke and domestic coal combustion. The limited number of studies exploring the health impacts associated with coal mine fires is likely to be for many reasons. The presence of associated gross environmental impacts may deflect attention from the health impacts; coal fires predominately occur in low income countries with more limited health and research infrastructure; the associated health problems may be subtle, masked by other health issues in these populations; or the fires are rare (Finkelman and Stracher, 2010). The absence of studies that demonstrate no association between coal fire smoke exposure and adverse health outcomes may simply reflect publication bias or lack of epidemiological research, rather than a lack of an association. Some of the largest coal fires affect poor and marginalised communities in India and China, where there could be less available research capacity or political will to investigate.

Coal mine fires are costly. The ability of a country to mount an adequate response to mitigate the hazards of coal mine fires is constrained by its economic resources and often relocation of communities is economically favourable to extinguishing a coal fire (Whitehouse and Mulyana, 2004). For example, after an initial \$US 4 million was spent on unsuccessful efforts to extinguish the Centralia mine fire, a further \$US 42 million was allocated to relocate the 1100 residents and businesses (Stracher and Taylor, 2004). The Hazelwood mine fire in Australia, which lasted just six weeks, was estimated to have cost approximately \$AU 100 million, of which \$AU 32.5 million was absorbed in firefighting efforts and \$AU 3 million in temporary relocation assistance for residents (Parliament of Victoria, 2014). In India, where densely populated communities reside amongst the JCF fires that have been burning for almost a century, the government proposed \$US 1.4 billion to relocate 90 000 residents to new settlements in 1996. However, as of 2012, only 1150 families have relocated, reportedly due to construction delays and local resistance to moving (Magnier, 2012). Indirect costs associated with coal fires are even more difficult to quantify. They include environmental degradation and the potential contribution to climate change, along with the health and social impacts of the fires (Parliament of Victoria, 2014; Whitehouse and Mulyana, 2004). Many of the worst coal fires in the world occur in impoverished and disadvantaged areas where the public health response capacity is limited. Further, as low income countries are most vulnerable to the effects of climate change, and climate change is likely to precipitate an increase in the occurrence and severity of coal fires, it is these countries that will continue to be the most vulnerable to the indirect health and environmental effects of coal fires (Fried et al., 2008; Mirza, 2003).

It is clear that the lack of evidence surrounding the health impacts associated with coal mine fires hinders the ability of governments and public health officials to mitigate the impacts of coal mine fire events in a timely, scientifically-informed manner. This was highlighted in the review of the public health response to the Hazelwood mine fire in Australia, which was criticised for the delayed relocation advice for vulnerable subpopulations and the provision of mixed health messages (Parliament of Victoria, 2014). Research is particularly needed in the development of exposure assessment tools and biomarkers including study of coal mine fire emissions where communities are actually exposed rather than at surface vents, and refinement of exposure modelling; studies of the effects of coal smoke on a range of perinatal, childhood, respiratory, cardiovascular and immune system outcomes; defining vulnerable subpopulation groups; assessing the impacts of at

different exposure time periods including periods of days to months; and assessing the effectiveness interventions in mitigating adverse health outcomes (Fullerton et al., 2008; Parliament of Victoria, 2014).

5. Conclusion

Coal mine fires represent a significant threat to human health. However, the extent of this threat is largely unquantified. Potential outcomes can only be extrapolated from studies exploring other, theoretically similar, air pollution events. Analogous exposures suggest that any exposure to coal mine fire smoke is likely to be associated with increased risks of adverse respiratory health outcomes. Adverse cardiovascular outcomes, increased mortality and other population impacts described for forest fire smoke exposure are also potentially important outcomes depending upon the magnitude of exposure and the number of people affected. While evidence for the domestic use of coal suggests that chronic exposure could contribute to increased risks of malignancy and toxicity from trace elements, there are major gaps in the available evidence for health outcomes associated with exposure to poor air quality for time periods of weeks to months. Such paucity of data severely limits the ability of governments and public health officials to mitigate the impacts of coal mine fire events in a timely, scientifically-based manner. The need to further understand the health impacts of coal mine fires is pressing, as they disproportionately affect vulnerable and disadvantaged communities.

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