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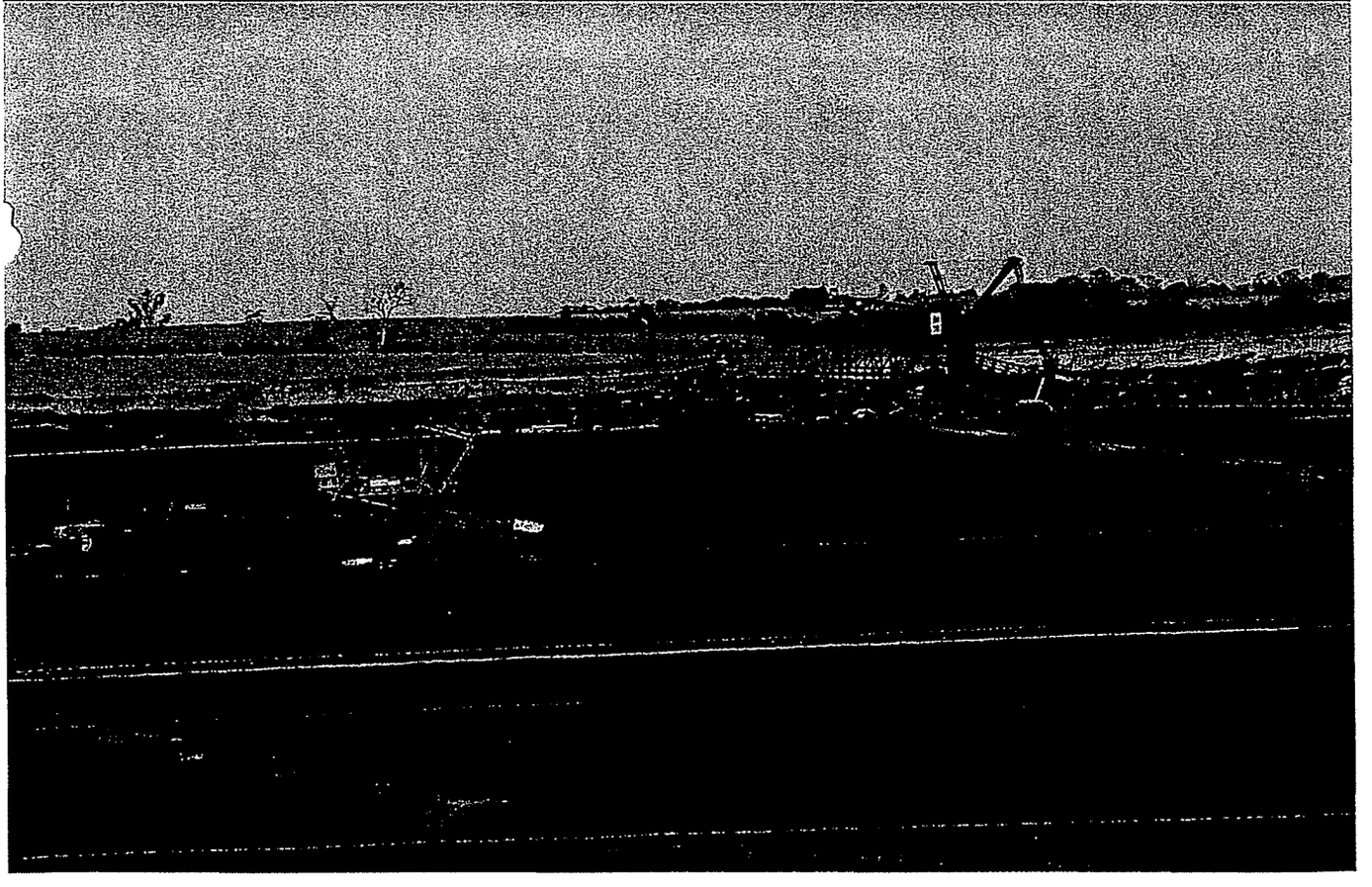
Final Report
Rapid Health Risk Assessment (RHRA)

Prepared for the Department of Health

12 March, 2014

Authors: Professor Michael Abramson, Dr Martine Dennekamp, Professor Malcolm Sim, Associate Professor Manoj Gambhir, Professor Brian Priestly, Dr Fay Johnston (Menzies Research Institute, University of Tasmania), Dr Lisa Demos, Professor John McNeil.



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Glossary of Terms

Ambient air	Outdoor air
Acute Lower Respiratory Infection (ALRI)	Acute lower respiratory infections usually includes acute bronchitis and bronchiolitis, influenza and pneumonia
Carbon monoxide (CO)	A poisonous gas produced by incomplete oxidation of fossil fuels. Carbon monoxide is poisonous by virtue of its capacity to bind to haemoglobin much more easily than oxygen
Carboxy-haemoglobin (COHb)	A compound produced by the exposure of haemoglobin to carbon monoxide. Carbon monoxide is inhaled into the lungs, absorbed through the alveoli, and bound to haemoglobin in the blood, blocking the sites for oxygen transport
Case-crossover study	An epidemiological study comparing ambient conditions during a period when an individual suffered an acute health effect with a period when no such effect
Chronic obstructive pulmonary disease (COPD)	Long-standing disease of the airways of the lung associated with increased production of phlegm and shortness of breath and often caused by cigarette smoking
Cohort study	An epidemiological study involving subjects exposed to pollutant(s) suspected of being related to the development of the disease under investigation. The entire study population is followed over time
Electrocardiogram (ECG)	A recording, from electrodes placed on the chest and limbs, of electrical changes originating in the muscle of the heart
ESCAPE	European Study of Cohorts for Air Pollution Effects
Ischaemic heart disease (IHD)	Disease of the heart caused by a reduction in the blood flow to the myocardium (the muscle of the heart wall) due to narrowing of the coronary arteries

Meta-analysis	Statistical analysis that allow the results of epidemiological studies to be combined
National Environment Protection Measure (NEPM)	A special set of national objectives designed to assist in protecting or managing particular aspects of the environment. The concept of NEPMs is unique and is provided for under the National Environment Protection Acts (NEPC Acts). NEPMs can be made about a variety of environmental matters as prescribed by the NEPC Acts, including ambient air quality
Polycyclic Aromatic Hydrocarbons (PAHs)	A group of organic contaminants that form from the incomplete combustion of hydrocarbons, such as coal. Many of these can cause cancer
$PM_{2.5}$	The concentrations (expressed in $\mu g/m^3$) of particles of less than 2.5 µm in the air
PM_{10}	The concentrations (expressed in $\mu g/m^3$) of particles of less than 10 µm in the air
Sulphur dioxide (SO_2)	An acidic gas formed by oxidation of sulphur found in fossil fuel
Time-series study	A study using an epidemiological method involving the relationship between outcome (e.g. number of deaths or hospital admissions in a population) and explanatory variables (e.g. pollutant concentrations) using measures of these variables at regular (usually daily) time intervals
Volatile Organic Compounds (VOCs)	Organic chemical compounds whose composition makes it possible for them to evaporate under normal indoor atmospheric conditions of temperature and pressure
µm	Abbreviation for micrometre or micron (a unit of length). 1µm = one thousandth of a millimetre
95% Confidence interval (95%CI)	The degree of uncertainty associated with a sample statistic, i.e. 95% Cl means that there is a 95% chance that the true value lies between the two values

FINAL REPORT OF RAPID HEALTH RISK ASSESSMENT

Executive Summary

This Rapid Health Risk Assessment was commissioned by the Department of Health to help understand the potential long term health effects on the Morwell community of short to medium-term exposure to coal mine fire smoke, including particulates and gaseous pollutants.

The specific aspects of the brown coal fire smoke which are addressed in this review are:

1. identifying the hazards of most concern in the coal mine smoke;
2. characterisation of the level of risk to the Morwell community, including vulnerable subgroups;
3. indicating how the risks would change with persisting exposure up to a year
4. identifying reports of long-term health outcomes from comparable incidents;
5. Providing recommendations for other relevant air toxics which should be measured; and
6. Identifying factors increasing risk.

The main health outcomes requested to be included in the review were heart and lung conditions, cancer and birth outcomes. Health risks to mine workers, firefighters and other emergency workers were not specifically asked to be included in this review.

The fire in the Hazelwood brown coal mine fire involved exposure of a nearby township to plumes of smoke which persisted over a period. To date there are no published health studies done specifically in relation to exposure to smoke from fires in open cut brown coal mines, similar to that at Morwell. As there was little published literature on the health effects of medium term high exposures from coal fire smoke on long term health outcomes, this review extrapolates and predicts effects on health by using what is known about health effects from urban air pollution and from biomass smoke.

The studies of urban air pollution used in the assessment of PM2.5 risks typically involved exposure to other air pollutants as well as particles. Despite statistical approaches that attempt to isolate the impact of PM2.5 alone, these adjustments are often limited and this potential confounding must be considered in assessing the health effects of fine particle exposure.

The principal risks to the health of the Morwell community from brown coal fire smoke arise from exposure to fine particulates ($PM_{2.5}$) and carbon monoxide (CO). There does not appear to be any significant risk from sulphur dioxide (SO_2). The potential risks from other air toxic hazards are currently unknown but are unlikely to substantially alter the conclusions in this Report. Available air quality data have been derived from two stationary monitoring sites in Morwell East and Morwell South. It has not been possible to allow for variability in personal exposures in assessing risks to health in this review.

Regarding possible causes of death from the inhalation of coal fire smoke, a meta-analysis of landmark cohort studies of urban air pollution has estimated an excess of risk of 6.2% (95%CI 4.1 – 8.4%) per 10 $\mu g/m^3$ of $PM_{2.5}$ for all-cause mortality and 10.6% (95%CI 5.4, 16.0%) per 10 $\mu g/m^3$ for cardiovascular mortality. In addition, four studies have found associations between forest fire smoke and all-cause mortality. Most large cohort studies have not found a significant increase in the risk of all cause mortality associated with CO alone.

Regarding health outcomes not resulting in death, the European Study of Cohorts for Air Pollution Effects (ESCAPE) recently found that a 5 $\mu g/m^3$ increase in estimated annual mean $PM_{2.5}$ was associated with a 13% increased risk of coronary events (95%CI 0.98 to 1.30). Positive associations were detected below the current annual European limit value for $PM_{2.5}$ of 25 $\mu g/m^3$. The most convincing evidence for adverse cardiovascular effects is from short term CO exposures that result in blood carboxy-haemoglobin (COHb) levels ≥ 2.4%, with effects occurring at the lowest levels in subjects with heart disease.

Regarding cancer, the International Agency for Research on Cancer recently concluded that particulate air pollution was a Class 1 human carcinogen. The ESCAPE lung cancer meta-analysis found a pooled hazard ratio for all lung cancers of 1·18 (0·96–1·46) per 5 $\mu g/m^3$ $PM_{2.5}$. The review also found that indoor smoke from black coal fires is a risk factor for lung cancer, particularly among non-smoking women in China.

Regarding birth outcomes, the ESCAPE meta-analysis of mother-child cohort studies found that a 5 $\mu g/m^3$ increase in concentration of $PM_{2.5}$ during pregnancy was associated with an increased risk of low birthweight at term (OR 1·18, 95%CI 1·06-1·33). An increased risk was also found for concentrations lower than the present European annual $PM_{2.5}$ limit. Two studies have found small but significant declines in birth weight for babies antenatally exposed during wildfires. There is also some evidence that antenatal CO exposure can be associated with low birthweight and preterm birth.

Based on these findings about the types of health outcomes related to air pollutants, epidemiological modelling undertaken as part of this review found that for combined $PM_{2.5}$ exposures around 250 $\mu g/m^3$ in Morwell South and for exposures around the National Environment Protection Measure (NEPM) in the rest of Morwell, no additional deaths would be expected even if the exposure continues for 6 weeks. However, if this level of exposure had persisted for 3 months this level of $PM_{2.5}$ might be expected to result in some additional deaths from IHD (0.5 additional deaths), Stroke (0.2), COPD (0.1), Lung Cancer (0.1) and Acute Lower Respiratory Infection (ALRI) (0.2).

The most vulnerable subpopulations in relation to health risks from exposure to $PM_{2.5}$ include children (< 5 years old), the elderly (> 65 years old), those with chronic heart and lung diseases such as Ischaemic Heart Disease (IHD), asthma and Chronic Obstructive Pulmonary Disease (COPD), and pregnant women. In addition to these well documented vulnerable groups, other factors may also increase the risk of health effects. These would include lower socioeconomic status. In addition the confounding effects of other known risk factors for these chronic diseases need to be considered. These would include cigarette smoking, obesity, hypertension, high cholesterol, diabetes, physical inactivity, occupational exposures etc.

The review has found that other relevant air toxics, apart from $PM_{2.5}$ and CO, should be measured. Measurements are currently being made of Polycyclic Aromatic Hydrocarbons (PAHs), dioxins and furans, formaldehyde and other carbonyls, chemical composition of particles, Volatile Organic Compounds (VOCs) and gaseous mercury. The results of these measurements were not available at the time of this report. It is not considered that any other air toxics need to be measured.

1. Background

This Rapid Health Risk Assessment (RHRA) was commissioned to assist the Department of Health to understand the potential long term health effects on the community of short to medium-term exposure (intermittent over weeks to months) to coal mine fire smoke, including particulates and gaseous pollutants.

The specific aspects of the risks to human health relating to the Morwell coal fire which the Department of Health has specifically asked to be addressed in this review are:

- *Characterisation of the level of risk to Morwell community posed by brown coal fire smoke:*
  - identifying the hazards and incorporating variable exposures experienced by differing community members
  - explicit reference to subpopulations with differing vulnerabilities
- *How does the risk change with persisting exposure; i.e. for periods of 3 weeks, 6 weeks, 3 months, 6 months, 9 months, or 1 year.*
- *Reports of long-term health outcomes from comparable incidents or circumstances*
- *Health outcomes of relevance:*
  - *Mortality*
  - *Morbidity*
  - *Cancer*
  - *Birth outcomes*
- *Recommendations for other relevant air toxics which should be measured*
- *Factors increasing risk*

The fire in the Hazelwood brown coal mine fire is unprecedented in that a township only a few hundred metres from the mine that has caught fire, exposing inhabitants to sometimes intense plumes of smoke over a period of several weeks. Unfortunately, to date there appear to be no published health studies done specifically in relation to exposure to smoke from fires in open cut brown coal mines, similar to that at Morwell.

The other unique aspect is that the exposure to the coal mine smoke has resulted in intermittent and on occasion high concentrations of pollutants, lasting for several weeks and which may persist for some months. As there is little published literature on the health effects of medium term high exposures on long term health outcomes, this review extrapolates and predicts effects on health by using what is known about health effects from urban air pollution and from biomass smoke.

In summary, without the availability of established scientific evidence related to health outcomes for this precise situation, this review relies on estimates of the scope of potential health effects based on knowledge and judgements related to what is known from similar air pollution research.

Of the criteria pollutants measured by the EPA, particles < 2.5 μm diameter ($PM_{2.5}$) and carbon monoxide (CO) are the ones that have most significantly increased. These have exceeded current National Environment Protection Measures (NEPM) for ambient air quality as a result of the smoke from the brown coal fire. Although other pollutants that may be elevated will be discussed, this report will mainly focus on the health risks from $PM_{2.5}$ and CO.

The focus of this review was requested to be the health risks on the Morwell community, rather than health risks related to the mine workers, firefighters and other emergency personnel.

2. Particulate Matter

2.1 Long term health effects of $PM_{2.5}$ urban air pollution

There is a well established association between short term $PM_{2.5}$ exposure and acute effects on cardiovascular and respiratory health. However there is now also evidence of long term $PM_{2.5}$ exposure and effects on the respiratory and cardiovascular systems. These studies show an increase in mortality and exacerbations of existing conditions. To date, the only association that has been found with the development of a disease has been with respiratory illness in children.

There are quite a few landmark studies on long term effects of urban air pollution on health. These include the:

- Harvard Six Cities Study(1-3) - a cohort study following 8111 adults in six US cities (1976 - 2009)
- American Cancer Society Study(4-6) - a cohort study of 552,800 adults from 51 US cities (1982 - 1998)
- Women's Health Initiative Observational Study(7) - cohort of 65,000 postmenopausal women from 36 US cities (1994-1998)
- Netherlands Cohort Study(8) - cohort of 120,000 men and women from 204 municipalities (1997 - 1996)
- Nurses' Health study(9) - cohort of 66,000 women from the US (1992-2002)
- Canadian National Cohort(10) - 2.1 million Canadians (1991-2001)
- European Study of Cohorts for Air Pollution Effects (ESCAPE) - several European Cohort Studies - results published recently(11)

Table 1 summarises the populations, follow-up periods, mean $PM_{2.5}$ concentrations proportional changes in risk of all-cause mortality and cardiovascular mortality associated with a 10$\mu g/m^3$ increase in $PM_{2.5}$.

The long term health effects in the above studies focus almost exclusively on mortality. Although the effect estimates in the above studies varies considerably, there is a clear association between $PM_{2.5}$ and all-cause mortality and mortality of respiratory and cardiovascular causes. The estimated cardiovascular effects tend to be stronger than the respiratory effect estimates. A review looking at several landmark studies on mortality found a pooled effect estimate of 6% for all-cause mortality expressed as excess risk per 10 $\mu g/m^3$ increase in $PM_{2.5}$ exposure(12). The landmark studies relate to long-term exposures to ambient air concentrations, that are generally much longer in duration that the elevated exposures for Morwell residents. There is likely to be a real difficulty in extrapolating the results of the above studies to the shorter-term exposures associated with this RHRA.

It is important to note, that very recently the International Agency for Research on Cancer (IARC) has classified ambient particulate air pollution as a known human carcinogen (Class 1)(13). Thus long term exposures to $PM_{2.5}$ are also likely to result in additional cases of lung cancer.

Table 1. Cohort studies on particulate matter <2.5μm($PM_{2.5}$) and mortality from all causes and cardiovascular diseases, adapted from Hoek et al (12)

Study	Population	Follow-up period	Mean Concentration $PM_{2.5}$ ($\mu g/m^3$)	% change in risk (95%CI) in all-cause mortality associated with a 10$\mu g/m^3$ increase PM	% change in risk (95%CI) in CV mortality associated with a 10$\mu g/m^3$ increase PM
Harvard six cities (1)	8111 adults (6 US cities)	1976 - 1989	18 (range 11–30)	13 (4, 23)	18 (6, 32)
Harvard six cities (2)	8096 adults (6 US cities)	1979 - 1998	15 (range 10–22)	16 (7, 26)	28 (13,44)
Harvard six cities (3)	8096 adults (6 US cities)	1974 - 2009	16 (range 11–24)	14 (7, 22)	26 (14, 40)
American Cancer Society (ACS) study (4)	552,800 adults (51 US cities)	1982 - 1989	18 (range 9–34)	26 (8, 47)	
ACS study (5)	500,000 adults (51 US cities)	1982 - 1998	18 (SD: 4)	6 (2, 11)	9 (3, 16)
ACS sub-cohort study (6)	22,905 subjects (LA area)	1982 - 2000	Range ~9 – 27	17 (5, 30)	26 (1, 60)
Women's Health Initiative Observational Study (7)	65,893 postmenopausal women (36 US metropolitan areas)	1994 - 1998	14 (range 3–28)		76 (25,147)
Netherlands Cohort Study (8)	120, 852 subjects (204 municipalities)	1987 - 1996	28 (range 23–37)	6 (–3, 16)	4 (–10, 21)
Nurses' Health Study (9)	66,250 women (US north eastern metropolitan areas)	1992 - 2002	14 (range 6–28)	26 (2, 54)	-
Medicare national cohort (14)	13.2 million US elderly Medicare recipients	2000 - 2005	13 (SD 4)	4 (3, 6)	-
California teachers study (15)	45,000 females	2002 - 2007	18 (7–39)	6 (–4, 16)	19 (5, 36)
Health professionals follow-up study (16)	17,545 highly educated US men	1989 - 2003	18 (SD 3)	–14 (–28,2)	3 (–17, 26)
Vancouver cohort (17)	452,735 residents 45–85 yr	1999 - 2002	4 (0 – 10)		7 (-14, 32)
US trucking industry cohort (18)	53,814 men (4 trucking companies)	1985 - 2000	14.1 (SD 4.0)	10 (2, 18)	5 (–7, 19)
Canadian national cohort (10)	2.1 million Canadians > 25 yr	1991 - 2001	9 (2 – 19)	10 (5, 15)	15 (7, 24)

Mortality

For the studies in table 1 above, pooled effect estimates expressed as percent excess risk per 10 $\mu g/m^3$ $PM_{2.5}$ was 6.2% (95%CI: 4.1 – 8.4%) for all-cause mortality and 10.6% (95%CI 5.4, 16.0%) per 10 $\mu g/m^3$ for cardiovascular mortality.(12) A meta-analysis of 33 time-series and case-crossover studies conducted in China showed that each 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 0.38% (95%CI: 0.31, 0.45) increase in total mortality, 0.51% (95%CI: 0.30, 0.73) in respiratory mortality and 0.44% (95%CI: 0.33, 0.54) in cardiovascular mortality.(19)

A prospective cohort of 2.1 million adults from the 1991 Canadian census mortality follow-up study was exposed to relatively low mean (±SD) $PM_{2.5}$ exposure levels of 8.7 ± 3.9 $\mu g/m^3$.(20) In fully adjusted models a 10-$\mu g/m^3$ elevation in $PM_{2.5}$ exposure was associated with an increase in risk of diabetes-related mortality (HR 1.49; 95%CI 1.37-1.62). The change in risk to the population persisted at $PM_{2.5}$ concentrations <5 $\mu g/m^3$.

Even within concentration ranges well below the present European annual mean limit value of 25 $\mu g/m^3$, long-term exposure to fine particulate air pollution was associated with natural-cause mortality in the 367,251 subjects from the ESCAPE study.(11) A significantly increased hazard ratio (HR) was found for $PM_{2.5}$ of 1.07 (95%CI 1.02-1.13) per 5 $\mu g/m^3$. This remained significantly raised even when only participants exposed to pollutant concentrations lower than the European annual mean limit (HR 1.06, 95%CI 1.00-1.12) or below 20 $\mu g/m^3$ (1.07, 95%CI 1.01-1.13) were included.

Cardiovascular and Cerebrovascular Events

The cardiovascular effects of short- and long-term exposure to $PM_{2.5}$ have been comprehensively reviewed for the American Heart Association.(21) The scientific statement concluded that exposure to $PM_{2.5}$ over a few hours to weeks could trigger cardiovascular disease–related mortality and nonfatal events. Longer-term exposure (eg. a few years) increased the risk of cardiovascular mortality to an even greater extent than exposures over a few days. It reduced life expectancy within more highly exposed segments of the population by several months to a few years. Reductions in $PM_{2.5}$ levels were associated with decreases in cardiovascular mortality within a time frame as short as a few years.

The ESCAPE Study recruited 100,166 people (Finland, Sweden, Denmark, Germany, Italy) free from cardiovascular events and followed them for an average of 11.5 years from 1997 – 2007.(22) A 5 $\mu g/m^3$ increase in estimated annual mean $PM_{2.5}$ was associated with a 13% increased risk of coronary events (HR 1.13, 95%CI 0.98 to 1.30). Positive associations were detected below the current annual European limit value for $PM_{2.5}$ of 25 $\mu g/m^3$ (1.18, 1.01 to 1.39 for a 5 $\mu g/m^3$ increase in $PM_{2.5}$).

The Multi-Ethnic Study of Atherosclerosis (MESA) reported that 2.5 $\mu g/m^3$ higher levels of residential $PM_{2.5}$ during the follow-up period were associated with 5.0 µm/year (95%CI 2.6 to 7.4 µm/year) greater intima-medial thickness (IMT) progression among persons in the same metropolitan area as well as slowed IMT progression with reductions in $PM_{2.5}$.(23)

In the Women's Health Initiative an increase of 10$\mu g/m^3$ in $PM_{2.5}$ concentration was associated with a 24% increase in the risk of a cardiovascular event (HR 1.24; 95%CI 1.09 to 1.41), 76% increase in the risk of death from cardiovascular disease (HR 1.76; 95%CI, 1.25 to 2.47) and an increased risk of

a cerebrovascular event (HR 1.35; 95%CI 1.08 to 1.68).(7) However not all studies have shown an increase in cerebrovascular events.

Lung Disease

The review by Hoek and co-workers concluded that the long-term exposure to $PM_{2.5}$ was more clearly associated with mortality from cardiovascular disease (particularly ischaemic heart disease) than from non-malignant respiratory diseases (pooled estimate 3%, 95% CI –6, 13%).(12)

The ESCAPE meta-analysis for Chronic Obstructive Pulmonary Disease (COPD) combined data from the ECRHS, NSHD, SALIA and SAPALDIA cohorts(24). There were 6550 and 3692 subjects with NO_2 and $PM_{2.5}$ data respectively. Exposure assessment was conducted with land use regression models and back-extrapolation. Surrogate measures were traffic intensity on the nearest road and load on roads within 100m of residence. COPD prevalence and incidence were defined both by the Global initiative for Obstructive Lung Disease (GOLD) criteria and the lower limit of normal. Logistic regression models adjusted for age, sex, height, BMI, education and smoking. Meta-analysis was performed with random effects models. There were weak but nonsignificant positive associations between NO_2, $PM_{2.5}$ and the prevalence / incidence of COPD. However significant overall associations were found with COPD and traffic intensity in females and never-smokers.

The ESCAPE lung cancer meta-analysis included 312,944 subjects from 17 cohort studies in 9 European countries and had a mean follow-up of 12.8 years. The hazard ratio (HR) for $PM_{2.5}$ for all lung cancers was 1·18 (0·96–1·46) per 5 $\mu g/m^3$ and 1·55 (1·05–2·29) for adenocarcinomas of the lung. (25) An analysis of birth cohort studies from ESCAPE which included Germany, Sweden, the Netherlands, and the United Kingdom measured lung function at 6–8 years of age (n = 5,921).(26) Annual average exposure to air pollution at the birth address and current address was estimated by land-use regression models. Associations of lung function with estimated air pollution levels and traffic indicators were estimated for each cohort. Changes in forced expiratory volume in 1 sec (FEV_1) were –1.77% (95% CI: –3.34, –0.18%) for a 5-$\mu g/m^3$ increase in $PM_{2.5}$.

Birthweight

Pooled data from 14 population-based mother-child cohort studies in 12 European countries (ESCAPE) included 74,178 women who had singleton deliveries between 1994 and 2011. The endpoint was low birth-weight at term defined as weight <2500 g at birth after 37 weeks of gestation.(27) A 5 $\mu g/m^3$ increase in concentration of $PM_{2.5}$ during pregnancy was associated with an increased risk of low birthweight at term (adjusted odds ratio [OR] 1·18, 95%CI 1·06-1·33). An increased risk was also found for concentrations lower than the present European annual $PM_{2.5}$ limit of 25 $\mu g/m^3$ (OR for 5 $\mu g/m^3$ increase in participants exposed to concentrations of less than 20 $\mu g/m^3$ 1·41, 95%CI 1·20-1·65). The population attributable risk estimated for a reduction in $PM_{2.5}$ concentration to 10 $\mu g/m^3$ during pregnancy corresponded to a decrease of 22% (95% CI 8-33%) in cases of low birthweight at term.

Australian Studies

The findings of these international cohort and time series studies have been replicated in Australia. Studies investigating the effects of $PM_{2.5}$ on hospital admissions found strong associations in

Melbourne, Sydney and Brisbane for admissions for respiratory and cardiovascular disease, asthma (especially in children <14 years) and those with COPD.(28) The strongest associations found in these studies were in the elderly and children. A study conducted in four Australian cities (Brisbane, Melbourne, Perth and Sydney), found significant associations between particles and all-cause mortality. Meta-analyses carried out for three cities yielded estimates for the increase in the daily total number of deaths of 0.9% (-0.7% to 2.5%) for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration(29).

2.2 Long term health effects of biomass smoke

The studies reporting on the health effects of biomass smoke may be more relevant to the current situation than those which examined urban particulate pollution. Unfortunately there are very few studies to date that have investigated the health effects of bushfire, peat or sugar cane smoke on long term health. This is in part because it is very difficult to measure long term health effects from short to medium term exposures.

There is an established association between short term exposure to bushfire smoke and effects on respiratory health – mainly measured by Emergency Department (ED) presentations and hospital admissions. Only a limited number of studies have looked at health outcomes other than emergency presentations and admissions. There is a suggestion of effects on cardiovascular presentations and admissions. However we have recently found that during bushfire smoke in Melbourne, there was a significant increase in the number of out-of-hospital cardiac arrests attended by ambulance personnel(30). These do not show up in hospital data as most arrests are fatal and are not recorded in the hospital system. Although the studies have only looked at short term health effects, this does not mean that the effects on the individual are short term as well. For example if someone has a cardiac arrest and survives with brain damage, the effects will be long lasting for that individual.

Susceptible populations

Studies specific to severe episodes of pollution due to forest and peat fires are relatively few in number and only a small proportion have studies individuals by risk factors such as age, and underlying health status(31-38). The findings from these studies are consistent with those of the wider air pollution literature i.e. that those most susceptible to forest fire smoke are the elderly and those with pre-existing heart or lung disease.

Biomass smoke and mortality

Only fairly recently have studies been adequately powered and able to investigate associations between forest fire smoke and mortality. In particular four studies, two from Sydney(31, 39), one each from Athens(40) and Malaysia(32) have found associations between forest fire smoke and mortality. Larger studies are needed to determine which causes of mortality are most affected(41). However in a regional Australian town seasonally affected by biomass smoke from woodheaters, an intervention that reduced winter $PM_{2.5}$ by 40% was associated with a reduction in winter mortality especially for cardiorespiratory causes of death(42).

Biomass smoke and birth outcomes

There is emerging evidence concerning perinatal exposure to episodes of smoke pollution and adverse outcomes in the neonatal period and infancy. Holstius et al(43) and Breton et al(44) both found small but significant declines in birth weight for babies antenatally exposed during the 2003 southern California wildfires. Jayachandran(45) studied the impact of a severe episode of forest fire smoke pollution that persisted for several months in Indonesia. She found that exposure to wildfire smoke, especially in the three months prior to birth, was associated with reduced foetal and infant survival. A very recent, as yet unpublished, study of Rhesus monkeys(46) demonstrated reduced cytokine production at the age of three in monkeys who had been exposed to smoke from Californian wildfires soon after they were born, compared with monkeys of a similar age who were not exposed. This could suggest that early life exposures might have immunological effects that persist into adolescence. However this requires confirmation in human studies.

Cancer and biomass smoke

There are no studies that have investigated the relationship between bushfire smoke and cancer. However there is one study(47) that found that sugar cane cutters exhibited increased frequency of cytological abnormalities in blood lymphocytes (micronuclei/1000 cells) compared to a control group, possibly due to exposure to emissions derived from sugar cane burning. On the other hand indoor smoke from black coal fires is a well recognised risk factor for lung cancer, particularly among non-smoking women in China. A recent meta-analysis of 25 case-control studies found that household coal use was associated with an overall 2.15 (95%CI 1.61, 2.89) fold increased risk of lung cancer(48). There was variation in effects across regions, with the highest risk found in southern China and Taiwan. This variation is thought to be due to other constituents of black coal. The elevated risk persisted when the studies were stratified by gender, smoking, sample size, design and language of publication.

3. Carbon Monoxide

There are two relevant international guidelines for carbon monoxide (CO): one for outdoor(49) and one for indoor air(50). There are many studies on the health effects of carbon monoxide poisoning and short term low dose exposures.(51) Continuous low level CO exposure for up to 8 days has been reported to result in ECG changes in p waves in healthy subjects.(52) Table 2 summarises longer term cohort studies, the populations, durations of followup and mortality per 1 ppm increase in CO concentrations.

Table 2. Cohort studies of Carbon Monoxide Exposure and Mortality

Population	Follow-up period	Mortality per 1 ppm increase CO concentration (95% CI)	Reference
552,138 US adults (151 metropolitan areas)	1982-1998	All cause RR= 0.97 (0.93, 1.0) Cardiopulmonary RR= 0.95 (0.88, 0.99) Lung cancer RR= 0.90 (0.83, 0.96)	(5)
28,402 US postmenopausal women 50 -79 years (36 metropolitan areas)	1994–2003	Cardiovascular HR =0.92 (0.71, 1.21) using single-pollutant model HR= 0.93 (0.67, 1.30) using multi-pollutant model	(7)
70,000 US male military veterans (mean 51 years) with hypertension	1976-2001	All cause RR= 1.032 (0.954, 1.117) using single-pollutant model RR=1.023 (95% CI: 0.939, 1.115) after adjustment for NO2 & O3	(53-55)

RR= relative risk, HR= Hazards ratio

Mortality

In a reanalysis of data from Pope and co-workers of 552,138 US adults (5) with more extensive treatment of co-variates the relative risk for death from all causes was estimated to be approximately 0.98 (95%CI: 0.92, 1.03) for an increase in ambient carbon monoxide concentration from 0.19 to 3.95 ppm.(56)

Several multi-city studies have found significant associations between increasing short-term average ambient air CO concentration and increasing mortality risk when CO is considered in single-pollutant models, with the associations attenuated when models were adjusted for other co-pollutants.(57) In a time-series study of mortality in 82 US cities during the period 1987– 1994, a 1 ppm increase in ambient air carbon monoxide concentration (lag 1 day) was associated with increased all-cause mortality of 0.46% (95%CI: 0.18, 0.73), but this was not significant after adjustment for air concentration of PM_{10} alone or PM_{10} and NO_2.(58) A time-series analysis of mortality in 19 European cities participating in the APHEA-2 (Air Pollution and Health: A European Approach) project during 1990–1997 found a significant association between CO and total non-accidental and cardiovascular mortality in single-pollutant models.(59) The estimated effect size for total mortality based on a single-pollutant model ranged from 0.59% increase (95%CI: 0.41, 1.79) to 1.20% (95%CI: 0.63, 1.77) per 1 mg/m^3 increase in CO concentration (0–1-day lag). The effect on cardiovascular mortality ranged from 0.8% increase (95%CI: 0.53, 1.07) to 1.25% (95%CI: 0.30, 2.21).

Overall, the epidemiologic evidence is consistent with a causal relationship between short-term exposure to environmentally relevant CO concentrations and all cause mortality.(60) The associations were strongest for people with existing cardiovascular disease and the elderly (>65 years).

Cardiovascular effects

Cardiovascular effects of inhalation exposures to CO have been evaluated in controlled human clinical trials, epidemiological studies, and various animal studies.(57) In general, these studies provide convincing evidence for adverse cardiovascular effects in association with short term CO exposures that result in blood carboxyhaemoglobin (COHb) levels ≥ 2.4%, with effects occurring at the lowest levels in subjects with coronary artery disease. Mean ambient air carbon monoxide concentrations reported in studies that have found CO-associated adverse cardiovascular outcomes ranged from 0.5 to 10 ppm, with maximum values ranging from 2 to 50 ppm.(57) These values correspond to approximate steady-state blood COHb levels of <2% for the mean and <10% for the maximum.

Ambient daily CO levels have been associated with increases in daily mortality and hospital admissions for cardiovascular diseases.(61) Groups within the population considered to be most susceptible to the effects of CO are people with cardiovascular disease, in particular the elderly with ischaemic heart disease.(28, 61) Studies of hospital admissions and emergency department visits for ischemic heart disease (IHD) and congestive heart failure (CHF) provide the strongest evidence of ambient CO being associated with adverse CVD outcomes.(28)

Single Australian city studies have found consistent associations with CO for hospital admissions and emergency department attendances for cardiovascular outcomes(62, 63). The strongest effects were found for admissions for cardiovascular disease in the elderly (>65 years) and all age groups, admissions for ischaemic heart disease and admissions for asthma in the 0–14 year age group.

Lung Disease

Relatively few controlled clinical trials have evaluated adverse respiratory effects of CO exposure. (57) Clinical trials have been conducted in very small numbers of healthy subjects under acute exposure conditions. There is a lack of strong evidence for associations between ambient air carbon monoxide concentrations at <30 ppm and pulmonary function from epidemiological studies.

Studies of children have also yielded mixed results. However one study of 263 Western Australian children demonstrated significant associations between CO (8 hour) and symptoms such as wheeze/rattle and runny/blocked nose (lag 5 and additive exposure over 5 days).(64) These associations were observed even though air pollutant concentrations were below national standards throughout the study period.

There is some evidence for associations between increasing air carbon monoxide concentrations and increasing severity of asthma.(57) In a study of 990 children aged 5–12 years from eight North American cities a 1 ppm increase in air carbon monoxide concentration (lag=0 days) was associated with an odds ratio of 1.08 (95%CI: 1.01, 1.14) for asthma symptoms.(65) However it is quite likely

that CO is serving as a surrogate for other combustion products, rather than having direct effects on the respiratory system.

Birth and Developmental Outcomes

Carbon monoxide has been associated with birth and developmental outcomes in international studies.(60) The most compelling evidence for a CO-induced effect on birth and developmental outcomes is for preterm birth (PTB) and cardiac birth defects. There is limited epidemiologic evidence that during early pregnancy (e.g., first month and first trimester) CO is associated with an increased risk of PTB. There is evidence of ambient CO exposure during pregnancy having a negative effect on foetal growth in epidemiologic studies.(66) In the Children's Health Study, a 1.4-ppm difference in first-trimester CO exposure was associated with 21.7 g lower birth weight (95%CI 1.1-42.3 g) and 20% increased risk of intrauterine growth retardation (95%CI, 1.0-1.4).(67)

A study that estimated the average exposure of five common air pollutants during pregnancy for births in metropolitan Sydney between 1998 and 2000 reported a reduction of 7 (95%CI -5.0 to 19.0) to 29 (95%CI 7.0 to 51.0) grams in birth weight for a 1ppm increase in mean carbon monoxide levels.(68) However another study which investigated the effect of prenatal exposure to six common urban air pollutants in the Sydney metropolitan area on pre-term birth between 1998 and 2000 found no clear impact of air pollutants on gestational age.(69)

Sensitive Groups

The results of epidemiological studies have found no evidence for a threshold below which adverse health effects in sensitive groups have not been observed after exposure to CO. The most sensitive groups to the effects of CO are people with existing cardiovascular disease, including ischaemic heart disease, pregnant women and the elderly.(28)

4. Morwell Air Quality Data

The fire at the coal mine started on the 9th February 2014. The data from the EPA started in Morwell East on the 17th Feb and in Morwell South on the 21st Feb. CO monitoring started on the 19th of Feb in Morwell East and 21st Feb in Morwell South. Both CO and $PM_{2.5}$ data were provided up until the morning of the 3rd of March.

Using the hourly concentrations, the average concentration to date Morwell South for $PM_{2.5}$ was 180µg/m^3 and the median 66 µg/m^3. As can be seen from Figure 1, the data are highly skewed. For Morwell East the average and median concentrations were 32 µg/m^3 and 13 µg/m^3 respectively. For CO in Morwell South the average and median concentrations were 3.3 ppm and 1.7 ppm respectively. For Morwell East the average and median concentration was 0.5 ppm and 0.1 ppm respectively. We use these observations to extrapolate effects on health (see below).

The figures below show the hourly air concentrations of the raw data as provided by the EPA.

Figure 1: Hourly average $PM_{2.5}$ concentrations in Morwell East and Morwell South

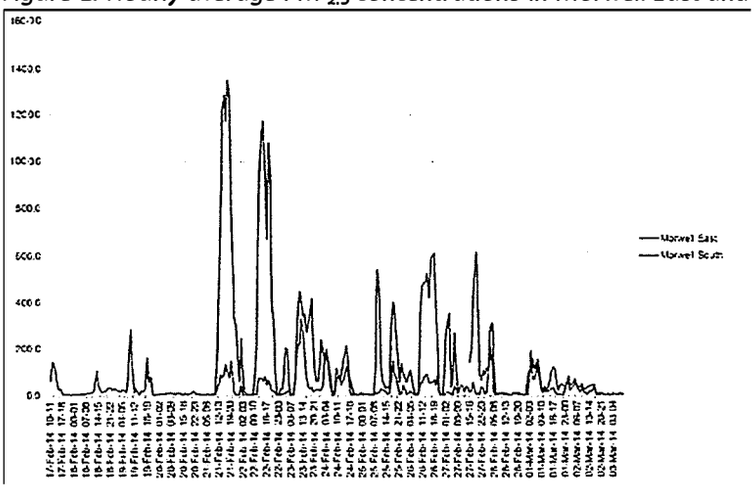
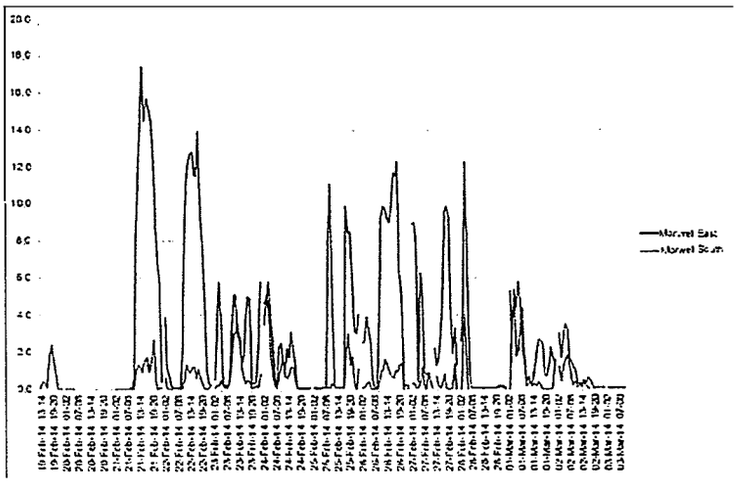


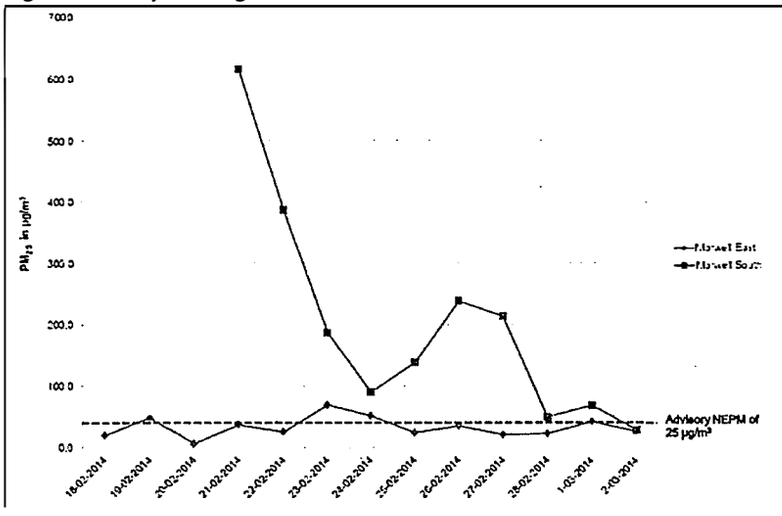
Figure 2: Hourly Average CO Concentrations (ppm) in Morwell East and Morwell South



How do the Morwell Air Quality Data compare with current standards?

The 24 hour National Environmental Protection Measure (NEPM) advisory standard for $PM_{2.5}$ is 25µg/m^3 with a maximum of 5 exceedances per year. The EPA $PM_{2.5}$ data provided for Morwell South started on the 17th of Feb, so it is quite likely exposures prior to that may have been higher. The average of the 14 days for which we have data is 33 µg/m^3 for Morwell East and for the 10 days in Morwell South the average is 202 µg/m^3. From figure 3 it is clear that exposures were very high when monitoring started. It is unfortunate that we do not know the concentrations prior to that. It is important to note, that even though there is a NEPM for $PM_{2.5}$, there is no 'safe level' below which there are no health effects expected. Numerous studies have been published where effects have been found well below the current air quality guidelines. The association between PM and effects on health has been shown to be linear.

Figure 3: Daily average PM2.5 concentrations in Morwell East and South



The 8 hour NEPM for CO is 9ppm with a maximum of 1 exceedance per year. This NEPM for CO was exceeded on 2 occasions in Morwell South for when measurements were available, i.e. the first 2 days the measurements were available, i.e. the 21st and the 22nd Feb. Therefore it is possible that the CO NEPM may have been exceeded on several days since the start of the fire on the 9th of February. Despite this the concentrations of CO are likely to have been mainly an occupational hazard rather than an environmental one. The long-term effects of exposure (at the measured levels) on health of the population of Morwell are likely to be minimal.

Sulphur dioxide (SO_2) is often a concern as a pollutant as a result from combustion. However in Victoria this is much less of an issue, mainly because brown coal has a very low sulphur content. The 1 hour NEPM for Sulphur dioxide is 200 ppb and the 24 hour NEPM 80 ppb. The sulphur dioxide measurements we have available for Morwell South (21st Feb - 7th March) confirm the low sulphur content of brown coal smoke. The hourly maximum concentration measured was 35ppb, so this even below the 24 hour NEPM. Health effects of SO_2 are unlikely to be observed at this level.

How does Morwell Air Quality compare with other cities / smoking etc?

Smoking: To put these concentrations into context, Burnett et al(70) estimated that smoking a single cigarette was equivalent to breathing a daily ambient concentration of $PM_{2.5}$ of 667 µg/m^3. The vast majority of a smoker's increase in mortality occurred at doses received before he/she was half way through the first cigarette of each day. It is also well known that cigarette smokers tend to have carboxyhaemoglobin concentrations substantially higher than non-smokers, and generally above a level considered to be the threshold for a range of adverse health effects associated with short-term exposures(51). The confounding effects of cigarette smoking would need to be factored into any analysis of carboxyhaemoglobin concentrations measured in Morwell residents during this incident.

Other Cities: *The London Smog event*: In December 1952, for 5 days London was covered by a very thick fog, due to unfavourable weather conditions trapping air pollution from homes, power plants and factories. The pollution was thought to have mainly been caused by smoke from the burning of coal. Bell and Davis(71) estimated that about 12,000 excess deaths occurred from December 1952 through February 1953 because of acute and persisting effects of the London smog. However there is an important difference between the London Smog episode and the coal mine smoke in Morwell.

Sulphur dioxide and acid aerosols are thought to have played a very important role in the death rate. And as shown above, the SO_2 in the Morwell smoke was well below the air quality standards.

As a comparison with other polluted cities in our region, the average (SD) $PM_{2.5}$ concentration in Beijing from 2007-2008 was 82 (52) $\mu g/m^3$. In addition the average concentration in Shanghai from 2007-2008 was 54 (31) $\mu g/m^3$. In Guangzuo the average concentration from 2007-2008 was 70 $\mu g/m^3$.(72) So therefore on several days in Morwell the concentration was at least as bad, or worse than the yearly average in some of the most polluted cities in China.

5. What Is The Risk And How Does Risk Change With Persisting Exposure?

Epidemiological modelling was undertaken to address this question. The population at risk has been assumed to be 14,005 which was the total population of Morwell in the 2011 census. Background mortality risks for Victoria were calculated using 2011 ABS data. Cause specific mortality rates of Ischaemic Heart Disease (IHD), Stroke, Chronic Obstructive Pulmonary Disease (COPD) and Acute Lower Respiratory Infection (ALRI) for Victoria were obtained from: http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/3303.0Chapter42011 Lung cancer rates for Victoria in 2010 were obtained from: http://www.cancervic.org.au/research/registry-statistics/cancer-in-victoria

Increases in absolute risk due to $PM_{2.5}$ exposure were calculated using the integrated risk functions recently published by Burnett et al(70). These functions provided an estimate and 95%CI for the additional number of cause-specific deaths attributable to PM_{25}. It should be noted that this study primarily related to ambient $PM_{2.5}$ exposure levels (e.g. from urban air pollution) and these tended to be considerably longer-term exposures than in the scenario we are analysing here. Therefore the additional risks attributable to $PM_{2.5}$ exposure published by Burnett et al(70) may only fully accrue when overall exposure times are longer than those we consider for Morwell. The exposure scenario is a combination of the daily average NEPM, which was the average exposure in Morwell East and the average exposure in Morwell South, where 1500 people were assumed to have been exposed. As requested, alternative durations of 3 weeks, 6 weeks, 6 months, 9 months and 1 year were modelled. The expected number of deaths for each duration of exposure and cause are summarised in Table 3. It can be seen that for this combined exposure scenario, no additional deaths would be expected even if the exposure continues for 6 weeks. However after 3 months, this level of exposure would be expected to result in some additional deaths from IHD, Stroke, COPD, Lung Cancer and ALRI.

Table 3. Modelled deaths from Ischaemic Heart Disease (IHD), Stroke, Chronic Obstructive Pulmonary Disease (COPD), Lung Cancer and Acute Lower Respiratory Infection (ALRI) in Morwell for a combined $PM_{2.5}$ exposure scenario and alternative durations of exposure

Cause of Mortality	Expected number of deaths in Morwell at Victorian rate	Duration of exposure	Additional no. deaths [95% CI]
		3 weeks	0.3 [0.2, 0.4]
		6 weeks	0.4 [0.3, 0.6]
		3 months	0.5 [0.4, 1.6]

IHD	12.9	6 months	2.4 [1.4, 4]
		9 months	2.9 [2.1, 5]
		1 year	3.7 [2.4, 6]
		3 weeks	0 [0, 0.3]
		6 weeks	0.1 [0, 0.2]
		3 months	0.2 [0, 0.4]
Stroke	3.1	6 months	0.5 [0.1, 0.8]
		9 months	0.8 [0.2, 1.2]
		1 year	1.1 [0.3, 2]
		3 weeks	0 [0, 0]
		6 weeks	0 [0, 0.1]
		3 months	0.1 [0, 0.2]
COPD	4.1	6 months	0.4 [0.1, 0.7]
		9 months	0.5 [0.3, 1.1]
		1 year	0.7 [0.3, 1.4]
		3 weeks	0 [0, 0]
		6 weeks	0.1 [0, 0.1]
		3 months	0.1 [0, 0.2]
Lung Cancer	4.8	6 months	0.6 [0.1, 1]
		9 months	0.8 [0.1, 1.5]
		1 year	1.3 [0.3, 2]
		3 weeks	0 [0, 0.1]
		6 weeks	0.1 [0.1, 0.1]
		3 months	0.2 [0.1, 0.4]
ALRI	3.6	6 months	0.6 [0.3, 1.1]
		9 months	1 [0.5, 1.5]
		1 year	1.2 [0.7, 1.9]

Other potential pollutants currently being measured:

It is difficult to estimate possible health effects of other pollutants being emitted by the brown coal fire smoke as we do not have results from the measurements currently being conducted. EPA and CSIRO have set up monitoring in Morwell to investigate these other potentially relevant pollutants, but unfortunately to date these samples have not been analysed. EPA and CSIRO are currently measuring:

- Polycyclic Aromatic Hydrocarbons (PAH), both particle and gas phase
- Particle and gas phase dioxins and furans, dioxin-like PCBs, BFRs
- Formaldehyde and other carbonyls
- Chemical composition of particles: soluble ions, elemental analysis (including metals), organic and elemental carbon
- Volatile Organic Compounds (VOC), including benzene, toluene, ethylbenzene, xylenes and 10 other common VOC
- Gaseous Elemental Mercury

Comment: Some of the above airborne pollutants (e.g. VOCs, formaldehyde and other carbonyls) are more likely to be associated with acute health impacts, such as mucous membrane irritation (eye and respiratory tract), while others (PAHs, dioxins, mercury) are more likely to be associated with longer-term or delayed health impacts, such as cancer, adverse reproductive outcomes and neurotoxicity. It is difficult to speculate on the potential for these health outcomes for the residents of Morwell in the absence of the monitoring data, and it may also be difficult to differentiate exposures to some of these pollutants from sources other than the coal fires (vehicle exhaust and other industrial or background sources).

In the case of dioxins and furans, it is well established that combustion sources rich in chlorinated aromatic compounds (e.g. bushfires, incinerators) are a significant source of airborne emissions of highly toxic chlorinated dibenzodioxins such as 2,3,7,8-tetrachlorodibenzodioxin (TCDD) and other congeners(73). However the toxicity profile of these congeners varies over a wide range, and it is usual to express the aggregate toxicity of a mixture of 'dioxin' congeners in terms of equivalent TCDD toxicity (TEQ), derived using Toxic Equivalency Factors (TEFs) that vary from 1 (for TCDD) to 0.0003 for the less toxic congeners. The profile of dioxin emissions from a brown coal fire relative to other combustion sources is unknown.

Direct inhalation of dioxins from airborne sources generally makes a very small contribution to overall intake in the general population of Australia(74), with more than 95% of exposure occurring through dietary intake of food, mainly fatty foods of animal origin (e.g. meat, dairy). This does not mean that inhalation exposure of Morwell residents to dioxins may not have been significant during the major phase of the fires, but it will be more difficult to assess in the absence of contemporaneous monitoring data. It may be necessary to extend dioxin monitoring to fatty foods sourced in the region, or to contamination of domestic surfaces, where hand-to-mouth contact by children may be a significant mode of systemic exposure.

Final remark:

The effects of biomass and peat smoke studied to date are broadly similar to urban air pollution in terms of mortality, and impacts on the heart, lungs and pregnancy, although the evidence base is much smaller. The main difference is that short term respiratory impacts could be greater with biomass smoke exposure, presumably due to the numerous respiratory irritants in the smoke.

6. Summary of major findings and conclusions

Based on the findings in this review, the following summarises the specific questions posed by the Department of Health:

- *Characterisation of the level of risk to the Morwell community posed by brown coal fire smoke*
    - identifying the hazards and incorporating variable exposures experienced by differing community members

The principal risks to the health of the Morwell community from brown coal fire smoke arise from exposure to fine particulates ($PM_{2.5}$) and carbon monoxide (CO). There does not appear to

be any significant risk from sulphur dioxide (SO_2). The potential risks from other air toxic hazards are currently unknown. Available air quality data have been derived from two stationary monitoring sites in Morwell East and Morwell South and it has not been possible to allow for variability in personal exposures in assessing risks to health in this review.

- explicit reference to subpopulations with differing vulnerabilities

The most vulnerable subpopulations include children (< 5 years old), the elderly (> 65 years old), those with chronic cardio-respiratory diseases such as Ischaemic Heart Disease (IHD), asthma and Chronic Obstructive Pulmonary Disease (COPD), and pregnant women. The likely effects on these groups are detailed in the main body of this report.

- *How does the risk change with persisting exposure; i.e. for periods of 3 weeks, 6 weeks, 3 months, 6 months, 9 months, or 1 year.*

Epidemiological modelling has been undertaken to address this question (see Table 3 on page 15). For combined $PM_{2.5}$ exposures around 250 $\mu g/m^3$ in Morwell South and around the National Environment Protection Measure (NEPM) in the rest of Morwell, no additional deaths would be expected even if the exposure continues for 6 weeks. However, after 3 months this level of $PM_{2.5}$ exposure would be expected to result in some additional deaths from IHD (0.5 additional deaths), Stroke (0.2), COPD (0.1), Lung Cancer (0.1)and Acute Lower Respiratory Infection (ALRI) (0.2).

- *Reports of long-term health outcomes from comparable incidents or circumstances*

No reports relating to any directly comparable previous incidents were able to be identified. Thus, this review has extrapolated from previous studies of the health effects of urban particulate air pollution and biomass smoke.

- *Health outcomes of relevance:*

  - *Mortality*

    A meta-analysis of landmark cohort studies of urban air pollution estimated that excess risk per 10 $\mu g/m^3$ $PM_{2.5}$ was 6.2% (95%CI 4.1 – 8.4%) for all-cause mortality and 10.6% (95%CI 5.4, 16.0%) per 10 $\mu g/m^3$ for cardiovascular mortality. Four studies have found associations between forest fire smoke and all-cause mortality. Most large cohort studies have not found a significant increase in the risk of all cause mortality associated with CO alone.

  - *Morbidity*

    The European Study of Cohorts for Air Pollution Effects (ESCAPE) recently found that a 5 $\mu g/m^3$ increase in estimated annual mean $PM_{2.5}$ was associated with a 13% increased risk of coronary events (95%CI 0.98 to 1.30). Positive associations were detected below the current annual European limit value for $PM_{2.5}$ of 25 $\mu g/m^3$. The most convincing evidence for adverse cardiovascular effects is from short term CO exposures that result in blood carboxy-haemoglobin (COHb) levels ≥ 2.4%, with effects occurring at the lowest levels in subjects with IHD.

- *Cancer*

  The International Agency for Research on Cancer recently concluded that particulate air pollution was a Class 1 human carcinogen. The ESCAPE lung cancer meta-analysis found a pooled hazard ratio (HR) for all lung cancers of 1·18 (0·96–1·46) per 5 μg/m^3 $PM_{2.5}$ and 1·55 (1·05–2·29) for adenocarcinomas of the lung. Indoor smoke from black coal fires is a well recognised risk factor for lung cancer, particularly among non-smoking women in China.

- *Birth outcomes*

  The ESCAPE meta-analysis of mother-child cohort studies found that a 5 μg/m^3 increase in concentration of $PM_{2.5}$ during pregnancy was associated with an increased risk of low birthweight at term (OR 1·18, 95%CI 1·06-1·33). An increased risk was also found for concentrations lower than the present European annual $PM_{2.5}$ limit. Two studies have found small but significant declines in birth weight for babies antenatally exposed during wildfires. There is also some evidence that antenatal CO exposure can be associated with low birthweight and preterm birth.

- *Recommendations for other relevant air toxics which should be measured*

  Measurements are currently being made of Polycyclic Aromatic Hydrocarbons (PAHs), dioxins and furans, formaldehyde and other carbonyls, chemical composition of particles, Volatile Organic Compounds (VOCs) and gaseous mercury. The results of these measurements were not available at the time of this report. However it is not considered that any other air toxics need to be measured.

- *Factors increasing risk*

  See above discussion of vulnerable subpopulations. Others would include lower socioeconomic status and indigenous people. In addition the confounding effects of other known risk factors for these chronic diseases need to be considered. These would include cigarette smoking, obesity, hypertension, hypercholesterolaemia, diabetes mellitus, physical activity, occupational exposures etc.

ACKNOWLEDGMENTS

We are very grateful to the British Columbia Centre for Disease Control for making available a confidential draft of the document: a review of 'health impacts of forest fire smoke exposure' (Reid et al, 2014) specifically for the purpose of this health risk assessment. The air quality data supplied by the EPA were raw data and therefor "EPA does not warrant that the data is error free "

AUTHOR BIOGRAPHIES

Professor Michael Abramson is Deputy Head of the Department of Epidemiology & Preventive Medicine and visiting medical officer in Allergy, Immunology & Respiratory medicine at the Alfred Hospital in Melbourne. He graduated in Medicine from Monash University in 1979 and received his PhD from the University of Newcastle in 1990 for research into occupational asthma. His current research program covers the epidemiology of asthma and chronic obstructive pulmonary disease, including genetic and environmental risk factors such as smoking, air pollution and occupation, and the role of spirometry in managing these conditions. This work has been supported by the National Health & Medical Research Council, Australian Research Council, Department of Human Services, Victorian Health Promotion Foundation, Australian Lung Foundation and Asthma Victoria.

Dr Martine Dennekamp is an occupational and environmental epidemiologist working in the field of air pollution, health and the environment. She was awarded a postdoctoral fellowship from the Centre for Air quality & health Research and evaluation (CAR), an NHMRC Centre of Research Excellence. Martine has an MSc in Environmental Sciences from the Netherlands and a PhD in Environmental Medicine from Scotland which included investigating the health effects of particulate air pollution on patients with chronic lung disease. Her environmental health research program and major interests are in the area of air quality and health, and in particular the association between health effects and smoke exposure from planned burns and bushfires, and the association between ambient air pollution and respiratory and cardiovascular health effects.

Professor Malcolm Sim is an Occupational Physician and epidemiologist who leads a team of 25 research and teaching staff in the Monash Centre for Occupational and Environmental Health (MonCOEH). His main research interests include the risks of chronic diseases from occupational and environmental exposures, with a special emphasis on occupational and environmental risk factors for chronic diseases, such as cancer and respiratory disease, exposure assessment, occupational disease surveillance, veteran health research and the health effects of environmental exposures to mobile phones, small particles, arsenic, pesticides and other environmental contaminants. He is a co-investigator on several NHMRC and ARC grants and other national and international collaborative studies. Malcolm is the Editor-in-Chief of Occupational and Environmental Medicine, a specialty journal of the BMJ.

A/Prof Manoj Gambhir is Head of the Epidemiological Modelling Unit, Department of Epidemiology & Preventive Medicine at Monash University. He has been involved in modelling and forecasting for policy in such diverse areas as neglected tropical diseases, HIV Aids and infectious diseases including influenza, pertussis, mumps, measles, rabies and cholera. He has worked at the US Federal Government Centers for Disease Control and Prevention (CDC) based in Atlanta since 2010, where he helped create the National Center for Immunization and Respiratory Diseases (NCIRD) Modelling Unit. His work there involved him in a range of dramatic health events, from targeting outbreaks of respiratory disease in the Middle East to tracing swine influenza illnesses in pig barns at US state fairs. His work at NCIRD included public health analysis and modelling for research and policy and collaboration with US government agencies, academics and the wider community on policy advice.

Professor Brian Priestly is a Professorial Fellow (now part-time) in the Department of Epidemiology & Preventive Medicine (DEPM) at Monash University and Director of the Australian Centre for Human Health Risk Assessment (ACHHRA). ACHHRA's core objective is to provide a national focus for human health risk assessment in the area of food and environment pollutants, and to contribute to workforce development by mounting training programs in health risk assessment. Brian's primary area of expertise is in toxicology. He was recently recognised as a Fellow (FACTRA) in the peer-reviewed register of the Australasian College of Toxicology & Risk Assessment. He has been active on many government technical committees and scientific advisory panels over the past thirty years. He is currently a Science Fellow advising the food (FSANZ), AgVet chemical (APVMA) regulators, as well as being an advisor to the industrial chemical (NICNAS) regulator on toxicological issues, including those relating to the regulation of nanoscale materials.

Dr Fay Johnston is an environmental epidemiologist at the Menzies Research Institute Tasmania. Her research focuses on the public health impacts of bushfires, heat events and outdoor air pollution from biomass combustion. She has studied the health impacts of landscape fire smoke for 15 years and has led several national and international research programs in this field including the global burden of disease modelling of deaths attributable to landscape fire smoke. She is lead investigator of the current Bushfire CRC/DEPI funded program of research 'Smoke impacts on community health and social perceptions' and the ARC funded 'Bushfires, smoke, and people: the risks and benefits from planned burning on the urban–rural interface'. She contributes to an international expert group developing current evidence-based guidelines for the management of severe smoke events form forest fires and was a founding member of the US NSF and NCEAS funded international pyrogeography network. She is also a public health physician and Specialist Medical Advisor, Public and Environmental Health, Department of Health and Human Services, Tasmania.

Dr Lisa Demos is a Clinical pharmacologist and her current positions include Senior Research Fellow, Department of Epidemiology & Preventive Medicine, Monash University and Senior Research Scientist with the Australian Disease Management Association. Lisa qualified as a pharmacist in 1977 and then went on to undertake post graduate studies completing her PhD in1990 in the department of Clinical Pharmacology. Over the years she has work on a very broad range of studies and projects from clinical drug trials, drug use evaluations to government reviews on water fluoridation and health effects of alcohol.

Professor John McNeil has been the head of the Monash University School of Public Health and Preventive Medicine based at the Alfred Hospital in Prahran since 1986. His research background is in epidemiology and clinical pharmacology. He has served on the senior medical staff of the Austin and Alfred Hospitals and the Monash Medical Centre. He is currently CIA of the NIH funded ASPREE study, a large scale randomized clinical trial of low-dose aspirin in persons over 70 years of age. He has a long history of involvement in clinical trials addressing preventive options in heart disease, renal disease, anesthesia, stroke and eye disease. He has published over 350 refereed publications. He has also been instrumental in the development of large-scale clinical registries to improve the measurement and benchmarking of clinical outcomes.

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School of Public Health and Preventive Medicine

The Alfred Centre
99 Commercial Road
Melbourne VIC 3004
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