MonashHealth

MONASH GENERAL MEDICINE

28 May 2014

Monash Medical Centre
Locked Bag 29
Clayton South
Victoria 3168

Justine Stansen
Hazelwood Mine Fire Inquiry
PO Box 3460
Gippsland MC VIC 3841

Dear Justine,

RE: Hazelwood Coalmine Fire Health Effects Report

Provided by
Donald Campbell

Position

Professor of Medicine	Program Director
Southern Clinical School	General Medicine Program
Monash University	Monash Health

Qualifications
MBBS Honours (Monash 1978)
MD (Monash 1990)
(Thesis: Immunohistological Analysis of Interstitial Lung Disease)

M Med Sci (Clinical Epidemiology) (Newcastle 1998)
(Thesis: South Australian Asthma Mortality Case Control Study)

Expertise
Respiratory, Sleep and General Physician
Clinical Epidemiologist

I have previously undertaken several asthma mortality studies, a confidential review of mortality in hospitals, and a review of the lung health program of former SEC power industry workers in the Latrobe Valley.

Other positions
Board member:
Asthma Foundation Victoria (1997 to present)
Orygen Youth Mental Health Centre Research Advisory Committee.
President-elect Internal Medicine Society of Australia and New Zealand

Copy of CV attached

Kind Regards,

Prof Don Campbell
Professor of Medicine
Southern Clinical School
Monash University
Director General Medicine Program
Monash Health

Hazelwood Coalmine Fire Health Effects Report

Plain Language Summary

The Hazelwood Coal mine fire has the potential for long and short term adverse health effects principally due to the release of known air pollutants including CO, ozone, and particulate matter. Components of Particulate Matter include fine particles <2.5 micron diameter ($PM_{2.5}$) and coarse particles <10 micron diameter (PM_{10}), which exert different effects due to differences in chemical composition and site of deposition in the respiratory tract.

The major health effects associated with exposure to these pollutants are summarized in the Summary Table below.

Unborn children, children, asthmatics and diabetics and those with chronic cardio-respiratory disease are at increased risk of adverse health consequences, as are exposed firefighters and exposed outdoor workers. Smoke from a fire can also have adverse psychological and psychiatric health effects.

Future research should determine the short and long term health consequences (morbidity, mortality and health services use) arising from exposure to the smoke from the fire, and include a focus on improving the physical and mental health of individuals and the community.

Summary; Major Health Effects Associated with Exposure to Key Pollutants

Agent	Exposure (Short or Long)	Group affected	Predominant Health Effects	
			Short term	Long term
CO	Short /high dose	Unborn	Fetal death	Low birth weight, Premature birth, small for dates
	Intensity/ duration	Firefighters	Death, neurological sequelae	Neurological and cardiac sequelae
Ozone	Intensity dependent	Children	Respiratory symptoms, new asthma, worse asthma	Respiratory symptoms, new asthma, worse asthma
		Adults	Respiratory symptoms, new asthma, worse asthma	Respiratory symptoms, new asthma, worse asthma
		Chronic cardio-respiratory illness	Worse asthma, cardio-respiratory morbidity and mortality	Worse asthma, cardio-respiratory morbidity and mortality
PM2.5	Intensity dependent	Unborn	SFD, LBW, Premature birth	Lung growth retardation
		Children	Respiratory symptoms, new asthma, worse asthma	Respiratory symptoms, new asthma, worse asthma
		Adults	Cardio-respiratory morbidity and mortality, Diabetes	Cardio-respiratory morbidity and mortality, lung cancer
		Chronic cardio-respiratory illness		Cardio-respiratory morbidity and mortality, lung cancer
PM10	Intensity dependent	Unborn	Small for dates	?
		Children	?	?
		Adults	Cardio-respiratory morbidity and mortality	?
		Chronic cardio-respiratory illness	Cardio-respiratory morbidity and mortality	?

Health Effects also includes a variety of psychological and mental health effects

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Hazelwood Coalmine Fire Health Effects Report

1 Introduction

1 **Hazelwood Power Station** is a base-load power station built between 1964 and 1971, which relies on deposits from the adjacent open cut brown coal mine. Brown coal from the Latrobe Valley has a high moisture content and contains low levels of ash (< 4%), sulphur (< 1%) and nitrogen (<1%). Discrete minerals and heavy metals are present in minute concentrations. Latrobe Valley brown coal also differs from black coal which has high concentrations of discrete minerals and heavy metals and sulphur.

2 Exposure

2 In a 2007-2008 report the National Pollutant Inventory rated the Hazelwood power stations release of particulate matter ($PM_{2.5}$) as low, oxides of nitrogen as medium, polychlorinated dioxins and furans as high and hydrochloric acid as high, and boron compounds as low (1).

3 A published analysis of brown coal fly ash particles produced from combustion in a Latrobe valley power station revealed five groups of elemental composition, they are Si-rich particles; Ca-rich particles; Fe-rich particles; particles with Mg-Ca Matrix and particles with Si-Ca matrix. Some environmental sensitive elements such as Zn, Pb, Ni, K and Cu are enriched in fine fly ash particles (2).

4 A report on meteorology and air pollution in the Latrobe Valley published in1985 describes the presence of SO_2, NO_2, Ozone, Carbon Monoxide (CO), and particulate pollutants in the atmosphere arising from coal mining and power generation activity. The effect of different types of plume behaviour arising from the smoke stacks of the coal mine are described in relation to meteorological phenomena, including inversion layers, wind speed and direction and the height above ground that pollutants are released into the atmosphere (3). Another early paper describes surface ozone levels in the Latrobe Valley The authors ascribe the formation of ozone to photochemical smog processes, which are affected by the temperature, time of day, wind speed and season (4).

5 Coal mine fires are an increasing problem worldwide. Coal seam fires can be divided into near-surface fires, in which seams extend to the surface and the oxygen required for their ignition comes from the atmosphere, and fires in deep underground mines, where the oxygen comes from the ventilation. Coal combustion in a coal mine fire does not allow for the filtration of particulates or other chemical components. There are no reported studies of the health impact of such fires that are specifically devoted to brown coal fires (5).

6 The nature of the exposure to air-borne pollutants arising from the Hazelwood open-cut coal mine fire was of a high level. This was over and above the normal background exposure in the Latrobe Valley. This period of increased exposure

commenced on 9 February and lasted at least until 10 March, when the fire was officially declared to be under control. Certain parts of the community were affected to a greater degree than other parts of the community during this period of high-level exposure.

7 The constituents of air pollution relevant to the open cut coal mine fire at Hazelwood in February-March 2014 include: CO, Particulates, Ozone, NO_2, and SO_2.

8 Specific pollutants pertaining to the emissions arising from the Hazelwood power station are regularly monitored and reported by the EPA (6), and national ambient air quality standards are established for emissions relevant to the Hazelwood coal mine fire (7). There are also national environment protection measures for air toxics. The five priority air toxics include: Benzene, formaldehyde, toluene, xylene and polycyclic aromatic hydrocarbons (PAH) (8,9). Levels of polycyclic aromatic hydrocarbons are included within the group of chemicals known as Volatile Organic Compounds (VOC's). These are not regularly monitored (10).

3 Susceptibility

9 Those persons susceptible to coal mine fire airborne pollutants include exposed firefighters and outdoor workers, and members of the community, particularly those vulnerable members of the community with chronic cardio-respiratory disease, children and unborn children.

10 The nature of the dose and duration of exposure to individuals arising from the combustion products of a coal mine fire is dependent upon:

- Fuel load (Size of the fire)
- Effects of heat on combustion
- Wind direction and speed
- Time of day
- Ambient temperature
- Nature of the plume from the point source
- Creation of photochemical smog

Health effects that may be caused by exposure to smoke from a brown coal fire, identifying the substance in the smoke that causes each effect

4 Specific Components

4.1 Carbon Monoxide (CO)

11 *CO is an odourless tasteless colourless gas. It is produced as a result of incomplete combustion of coal. It is a known environmental hazard in coal mines and coal-fired power plants, particularly in poorly ventilated closed spaces. It is usually rapidly dispersed in open environments with circulating air. It contributes to the formation of ozone in photochemical smog, however.*

12 The effect of CO exposure is greatest for coal miners and fire fighters, but also potentially for those members of the community who may be exposed to high levels in the acute phase of the fire.

13 Health adverse effects of CO exposure relate to its ability to enter the bloodstream and bind circulating Hemoglobin within red blood cells with a greater affinity than oxygen, thereby displacing bound oxygen and reducing the delivery of oxygen to working tissues, the heart and the brain.

14 At low levels of exposure no health effects may be experienced but rising levels can cause symptoms consistent with oxygen deprivation. These effects are reversible at low levels of exposure, and are preventable by wearing an appropriate respirator. The treatment includes recognition of the effect, removal from continuing exposure and the administration of oxygen, potentially including hyperbaric oxygen.

15 The greatest risk to the exposed firefighter arises when the person does not suspect exposure and therefore enters an area of high concentration without knowing it and with out the ability to detect it.. A similar logic will apply to members of the public exposed to high ambient levels of CO arising from coal mine fire smoke exposure, including particularly those who are vulnerable groups such as those with chronic cardiorespiratory disease, children by virtue of higher metabolic rate and unborn children.

4.1.i Acute poisoning

16 The main manifestations of carbon monoxide poisoning develop in the organ systems most dependent on oxygen use, the CNS and the heart. The initial symptoms of acute carbon monoxide poisoning include headache, nausea, malaise and fatigue. Headache is the most common symptom of acute carbon monoxide poisoning; it is often described as dull, frontal, and continuous.

17 Increasing exposure produces cardiac and neurological abnormalities. There is evidence of long term heart muscle damage and increased long term mortality in association with heart muscle damage in survivors of acute CO poisoning (11). One of the major concerns following acute carbon monoxide poisoning is the severe delayed neurological manifestations that may occur. Problems may include difficulty with higher intellectual functions, short term memory loss, impaired cognition, speech and gait disturbances, Parkinsons' like syndromes, mood disturbance, depression and cortical blindness. These delayed neurological sequelae may occur in up to 50% of poisoned people after 2 to 40 days (12). Carbon monoxide poisoning in pregnant women may also cause severe adverse effects on the fetus as a result of fetal hypoxia (13). While severe CO poisoning poses serious short- and long-term fetal risk, mild accidental exposure is likely to result in normal fetal outcome (14).

The acute effects produced by carbon monoxide in relation to ambient concentration in parts per million are listed below (15):

Concentration	Symptoms
35 ppm (0.0035%)	Headache and dizziness within six to eight hours of constant exposure
100 ppm (0.01%)	Slight headache within two to three hours of exposure
200 ppm (0.02%)	Slight headache within two to three hours of exposure; loss of judgment
400 ppm (0.04%)	Frontal headache within one to two hours of exposure
800 ppm (0.08%)	Dizziness, nausea, and convulsions within 45 minutes of exposure; insensible within 2 hours
1,600 ppm (0.16%)	Headache, tachycardia, dizziness, and nausea within 20 minutes of exposure; death in less than 2 hours of exposure
3,200 ppm (0.32%)	Headache, dizziness and nausea within five to ten minutes of exposure. Death within 30 minutes of exposure.
6,400 ppm (0.64%)	Headache and dizziness in one to two minutes of exposure. Convulsions, respiratory arrest, and death in less than 20 minutes.
12,800 ppm (1.28%)	Unconsciousness after 2–3 breaths. Death in less than three minutes of exposure.

Recommended World Health Organisation (WHO) Air Quality guidelines for Carbon Monoxide (Europe 2000) (16).

18 The following guideline values (ppm values rounded) and periods of time-weighted average exposures have been determined in such a way that the carboxyhaemoglobin (COHb) level of $_{2.5}$% is not exceeded, even when a normal subject engages in light or moderate exercise:

1. 100 mg/m3 (87 ppm) for 15 min
2. 60 mg/m3 (52 ppm) for 30 min
3. 30 mg/m3 (26 ppm) for 1 h
4. 10 mg/m3 (9 ppm) for 8

4.2 Ozone

19 ***Ozone*** *(O_3) is formed from O_2 by the action of UV light and also atmospheric electrical discharges. Ozone's odor is sharp, reminiscent of chlorine, and detectable by many people at concentrations of as little as 10 ppb in air. It is much less stable than O_2, decaying to ordinary diatomic oxygen. It has a varying length half-life (meaning half as concentrated, or half-depleted), depending upon atmospheric conditions (temperature, humidity, and air movement).*

20 *Ozone is a powerful oxidant, far stronger than O_2. This high oxidizing potential, however, causes ozone to damage mucus and respiratory tissues in animals above concentrations of about 100 ppb. This makes ozone a potent respiratory hazard and pollutant near ground level. Exposure of 0.1 to 1 µmol/mol produces headaches, burning eyes and irritation to the respiratory passages*

21 *Low level ozone (or tropospheric ozone) is an atmospheric pollutant formed by the reaction of sunlight on air containing hydrocarbons and nitrogen dioxide that react with O_2 to form ozone directly at the source of the pollution or many kilometers down wind. The ozone at ground level is primarily derived from fossil fuel. Ozone production rises during heat waves.*

22 People with lung disease, children, older adults, and people who are active outdoors may be particularly sensitive to ozone. Children are at greatest risk from exposure to ozone because their lungs are still developing and they are more likely to be active outdoors when ozone levels are high, which increases their exposure. Children are also more likely than adults to have asthma.

23 The current one-hour and four-hour standard levels for ozone according to the EPA Victoria are: 0.10 ppm (parts per million) and 0.08 ppm, respectively. The one-hour and four-hour ozone standards should not be exceeded on more than one day per year.

24 The current WHO guideline for Ozone exposure is 100 µg/m^3 8-hour mean

25 The rationale for this is that as ozone concentrations increase above the guideline value, health effects at the population level become increasingly numerous and severe. Such effects can occur in places where concentrations are currently high due to human activities or are elevated during episodes of very hot weather (17).

4.2.i Short term exposure effects

26 Studies usually report short term effects occurring within days of daily maximum 8 hour mean exposures, but sometimes maximum daily 1 hour exposure levels.

27 The recently published Review of Evidence on Health Aspects of Air Pollution-REVIHAAP Project. (WHO Regional Office for Europe 2013) updates the evidence base since the previously published review in 2005, which had found support only for short-term effects of ozone on mortality and respiratory morbidity. The evidence for a threshold for short term exposure is not consistent, but where a threshold is observed, it is likely to lie below 45 ppb (90 $\mu g/m^3$) (maximum 1- hour) (18,19,20).

28 Previous studies had shown that short-term exposure to ozone impairs pulmonary function:

- People with asthma and allergic rhinitis are somewhat more susceptible to transient alterations in respiratory function due to acute exposure to ozone.
- Changes in pulmonary function are an immediate consequences of ozone exposure.
- Ozone enhances airway responsiveness in both healthy individuals and asthmatics.

29 There is solid evidence that ozone acutely increases morbidity. School absenteeism, hospital admissions or emergency department visits for asthma, respiratory tract infections and exacerbation of chronic airway diseases were the most common health end-points. Studies of mortality showed an increase in mortality due to respiratory disease in the elderly, predominantly from respiratory causes.

30 There is new evidence from short term exposure studies for ozone exposure to be related to increased all cause, cardiorespiratory and respiratory mortality, as well as hospital admissions for respiratory and cardiorespiratory problems. This effect was particularly seen in summer.

31 Comment: monitoring of ozone levels at air quality monitoring stations may well underestimate the exposures of individuals at particular locations in close proximity to intense exposure to smoke arising from the fire source whether in the immediate vicinity or in the plume arising from such a fire.

32 There is strong evidence of a short-term association between ozone exposure and mortality, with larger effects for cardiovascular and respiratory mortality, the elderly, and current-day ozone exposure. In a meta-analysis, a 10-ppb increase in daily ozone at single-day or 2-day average of lags 0, 1, or 2 days was associated with an 0.87% increase in total mortality (95% posterior interval = 0.55% to 1.18%) (21).

33 In Europe, adverse effects of short-term exposure to daily concentrations of ozone (maximum 1-hour or 8-hr mean) on all-cause, cardiovascular and respiratory mortality have been reported. Adverse effects of exposure to daily ozone concentrations on both respiratory and cardiovascular hospital admissions, after adjustment for the effects of particles (PM_{10}), have also been reported.

34 The following table summarizes the findings for European cities from the APHENA study for the relationship between short term ozone exposure and all-cause and cause-specific mortality and cardiovascular and respiratory admissions (19, 22).

35 In Europe, positive associations were observed in the all-year analyses for 1-hour ozone exposure and all-cause mortality, cardiovascular and respiratory mortality – associations that persist after adjustment for PM (PM_{10}). Estimates of all effects had lower CI above zero, with the exception of respiratory mortality. For hospital admissions, positive associations were observed between 1-hour ozone and respiratory and cardiovascular disease after adjustment for PM_{10} only.

Table. Associations between short-term exposure to ozone and Mortality and hospital admissions in European cities in the APHENA study

Outcome	Per cent increase in deaths/admissions (95% CI) per 10 µg/m3 increment in daily maximum 1-hour ozone concentrations	
	Single pollutant	Adjusted for PM_{10}
All-cause mortality a	0.18 (0.07–0.30)	0.21 (0.10–0.31)
Cardiovascular mortality: 75 years and older a	0.22 (0.00–0.45)	0.21 (-0.01–0.43)
Cardiovascular mortality: younger than 75 years a	0.35 (0.12–0.58)	0.36 (0.10–0.62)
Respiratory mortality b	0.19 (-0.06–0.45)	0.21 (-0.08–0.50)
Cardiac admissions: older than 65 years	-0.10 (-0.46–0.27)	0.64 (0.36–0.91)
Respiratory admissions: older than 65 years	0.19 (-0.28–0.67)	0.32 (0.05–0.60)

4.2.ii Long term exposure effects

36 Long term exposure effects are frequently reported in relation to six month summer averages of daily maximum one hour concentrations (23).

37 Previous studies had shown an inverse relationship between chronic ozone exposure and small airway lung function growth rates. There is evidence for an effect of long-term exposure to ozone on respiratory and cardiorespiratory mortality, which for the latter is less conclusive. Also, there is some evidence from other cohorts for an effect on mortality among persons with potentially predisposing conditions (chronic obstructive pulmonary disease, diabetes, congestive heart failure, and myocardial infarction). Additionally, several new follow-up long-term exposure studies have reported adverse effects on asthma incidence, asthma severity, hospital care for asthma and lung function growth.

38 Thus there is evidence that long term exposure to ozone has an effect on: respiratory and cardiorespiratory mortality, asthma incidence and severity, hospital admissions for respiratory and cardiorespiratory illness. In particular, new studies demonstrate a relationship between long term ozone exposure and new onset asthma in children and increased respiratory symptoms in those persons with pre-existing asthma.

39 In summary new studies since 2005 demonstrate:

- effect on impaired lung function growth in non-asthmatic children
- increased numbers of first admissions to hospital in children with high ozone exposure
- increased levels of IgE (ie increasing the propensity to an allergic diathesis)
- worse control of asthma in adults
- increase in pre term birth associated with ozone exposure
- poor lung growth and worsening asthma symptoms in children with high ozone exposures
- evidence of cognitive decline in adults related to high exposure
- impaired prenatal development
- effect on sperm count and motility in humans

4.3 Particulates

40 *"Particulate matter," also known as particle pollution or PM, is a complex mixture of extremely small particles and liquid droplets. Particle pollution is made up of a number of components, including acids (such as nitrates and sulfates), organic chemicals, metals, and soil or dust particles.*

41 *PM can either be directly emitted into the air (primary PM) or be formed secondarily in the atmosphere from gaseous precursors (mainly sulfur dioxide, nitrogen oxides, ammonia and non-methane volatile organic compounds). Primary PM (and also the precursor gases) can have anthropogenic and nonanthropogenic sources (for primary PM, both biogenic and geogenic sources may contribute to PM levels).*

42 *Particle pollution is grouped into two categories:*

1. *PM_{10} Inhalable coarse particles, such as those found near roadways and dusty industries, which are larger than 2.5 microns and smaller than 10 microns in diameter.*
2. *$PM_{2.5}$ Inhalable fine particles, such as those found in smoke and haze, are 2.5 microns in diameter and smaller. These particles can be directly emitted from sources such as coal mine or forest fires, or they can form when gases emitted from power plants, industries and automobiles react in the air.*

43 *The most important chemical constituents of PM are sulfate, nitrate, ammonium, other inorganic ions (such as Na+, K+, Ca2+, Mg2+ and Cl–), organic and elemental carbon, crustal material, particle-bound water and heavy metals. The larger particles (with the diameter between $_{2.5}$ and 10 µg/m3, i.e. the coarse fraction of PM_{10}) usually contain crustal materials and fugitive dust from roads and industry.*

44 The size of particles is directly linked to their potential for causing health problems. Once inhaled, these particles can affect the heart and lungs and cause serious health effects. Exposure to PM in ambient air has been linked to a number of different health outcomes, ranging from modest transient changes in the respiratory tract and impaired pulmonary function, through increased risk of symptoms requiring emergency room or hospital treatment, to increased risk of death from cardiovascular and respiratory diseases or lung cancer. This evidence stems from studies of both acute and chronic exposure. Toxicological evidence supports the observations from epidemiological studies (24).

45 Comment: there is no evidence from reported studies to distinguish the composition of particulate matter produced by complete combustion in a brown coal power station furnace from that produced by incomplete combustion in a coalmine fire or produced from a bushfire.

4.3.i. PM_{10} vs $PM_{2.5}$ What is the difference?

Coarse particles (PM_{10})

46 Inhalable particles less than 10 micrometers (μm) in diameter used as a nominal surrogate for particles between 2.5 and 10μm in diameter; found near roadways and dusty industries.

Fine particles ($PM_{2.5}$)

47 Inhalable particles less than 2.5μm in diameter; generally found in smoke and haze, emitted from natural sources like forest fires and industrial combustion sources, or formed when gases react in the air.

48 Inhalable particles, particularly fine particles, have the greatest demonstrated impact on human health. Their small size allows them to get deep into the lungs and from there they can reach or trigger inflammation in the lung, blood vessels or the heart, and perhaps other organs. Studies have linked PM exposure to health problems (24).

49 The current standards for particulates according to the EPA Victoria are (25):

$PM_{2.5}$: 8 $\mu g/m^3$ annual mean and 25 $\mu g/m^3$ 24-hour mean

PM_{10}: 50 $\mu g/m^3$ 24-hour mean

The standard for PM_{10} should not be exceeded on more than five days.

50 The current WHO exposure guidelines for particulates are (26):

$PM_{2.5}$: 10 $\mu g/m^3$ annual mean 25 $\mu g/m^3$ 24-hour mean

PM_{10}: 20 $\mu g/m^3$ annual mean 50 $\mu g/m^3$ 24-hour mean

51 The rationale for this is: at present, most routine air quality monitoring systems generate data based on the measurement of PM_{10} as opposed to other particulate matter sizes. Consequently, the majority of epidemiological studies use PM_{10} as the exposure indicator. Although PM_{10} is the more widely reported measure, and also the indicator of relevance to the majority of the epidemiological data, for reasons that are discussed below, the WHO Air Quality Guidelines for PM are based on studies that use $PM_{2.5}$ as an indicator. The $PM_{2.5}$ guideline values are converted to the corresponding PM_{10} guideline values by application of a $PM_{2.5}$ / PM_{10} ratio of 0.5. Based on known health effects, both short-term (24-hour) and long-term (annual mean) guidelines are needed for both indicators of PM pollution.

52 Recent WHO evaluations point to the health significance of $PM_{2.5}$ (26,27). In particular, the effects of long term PM exposure on mortality (life expectancy) seem to be attributable to $PM_{2.5}$ rather than to coarser particles. The latter, with a diameter of 2.5–10 μm ($PM_{2.5-10}$), may have more visible impacts on respiratory morbidity. The primary, carbon-centred, combustion- derived particles have been found to have considerable inflammatory potency. Nitrates, sulfates and chlorides belong to components of PM showing lower toxic potency.

53 Health effects are observed at all levels of exposure, indicating that within any large population there is a wide range of susceptibility and that some people are at risk even at the lowest end of the observed concentration range.

54 People with pre-existing heart and lung disease, asthmatics, socially disadvantaged people, children and the unborn child belong to the more vulnerable groups. The other groups who potentially will also be at increased risk including current smokers and otherwise healthy people undertaking vigorous outdoor exercise

55 Coarse particles are an independent entity distinct from fine and ultrafine particles. Epidemiological and toxicological studies have shown PM mass ($PM_{2.5}$ and PM_{10}) comprises fractions with varying types and degrees of health effects, suggesting a role for both the chemical composition (such as transition metals and combustion-derived primary and secondary organic particles) and physical properties (size, particle number and surface area) (19).

56 Short-term exposures to coarse particles (PM_{10}) are associated with adverse respiratory and cardiovascular effects on health, including premature mortality. Data from clinical studies are scarce; toxicological studies report that coarse particles can be as toxic as $PM_{2.5}$ on a mass basis. The difference in risk between coarse and fine PM can, at least partially, be explained by differences in intake and different biological mechanisms.

57 Sufficient evidence exists for proposing a short-term standard for PM_{10}, to protect against the short-term health effects of coarse particles, in addition to fine particles. There is very limited evidence of long-term health effects of coarse particles available to date. No systematic assessment of studies on long-term health effects of PM_{10} in Europe is available. Overall, the ability to separate long-term health effects of PM_{10} from other pollutants, such as NO_2 or $PM_{2.5}$, was limited and the evidence mixed.

58 Epidemiological studies show further evidence that long-term (years) exposure to $PM_{2.5}$ is associated with both mortality and morbidity. The prevalence of diabetes is directly related to $PM_{2.5}$ levels of exposure (28). The evidence base is weaker for PM10, although there is evidence from ecological studies that diabetics are twice as likely as non-diabetics to have a cardiovascular admission to hospital for a given level of PM_{10} exposure (29). Hardly any long-term studies are available for coarse particles.

59 The latest systematic review by Brunekreef & Forsberg (2005) made the scientific community aware again of the potential health risks associated with coarse particles. The review concluded that coarse PM has at least as strong short-term effects on respiratory health as $PM_{2.5}$; also, for cardiovascular effects, some supportive evidence was found (30).

60 The EPA integrated science assessment for PM concluded that, in general, short-term epidemiological studies reported positive associations between mortality and cardiovascular and respiratory hospital admissions (EPA, 2009) (31). For cardiovascular outcomes (admissions and physiological effects), effect estimates of coarse PM were found to be comparable to those of $PM_{2.5}$. On the other hand, it was noted that no associations of coarse PM on lower respiratory symptoms, wheeze, or medication use were reported.

61 The EPA noted in the 2009 integrated science assessment that the composition of coarse PM can vary considerably, but that there is limited evidence on the effects of the various biological and chemical components of coarse PM. Practically no studies compare the effects on health of coarse PM from different sources.

4.3.ii. Short term exposure effects of $PM_{2.5}$

62 Short-term exposure effects are reported as the relationship between short term exposure measured as a 24 hour average over 2 days of monitoring with the outcome measured within days of the exposure.

63 Atkinson et al (32) reported a systematic review and meta analysis of 110 studies examining the relationship between short term exposure to $PM_{2.5}$ and health outcomes including mortality and hospital admissions . They found evidence for adverse health effects of short-term exposure to $PM_{2.5}$ across a range of important health outcomes, diseases and age groups. They found that for all-cause mortality, a 10 $\mu g/m^3$ increase in the level of $PM_{2.5}$ exposure was associated with a 1.04% (95% CI 0.52% to 1.56%) increase in the risk of death. (Ie, as the ambient air level concentration of $PM_{2.5}$ increases by 10 $\mu g/m^3$ there is a 1.04% increase in mortality). Associations for respiratory causes of death were larger than for cardiovascular causes, 1.51% (1.01% to 2.01%) vs 0.84% (0.41% to 1.28%). Associations between $PM_{2.5}$ exposure and death from ischaemic heart disease, stroke and COPD were 3.36% (0.68%, 6.10%), 1.85% (0.74%, 2.97%) and 2.86% (−0.12%, 5.93%) per 10 $\mu g/m^3$ increase, respectively. $PM_{2.5}$ concentrations were positively associated with increased risk of admission for cardiovascular diseases, 0.90% (95% CI 0.26% to 1.53%) and respiratory diseases, 0.96% (95% CI − 0.63% to $_{2.5}$8%) per 10 $\mu g/m^3$, respectively.

64 Kloog et al, found that $PM_{2.5}$ exposure was associated with hospital admissions for all respiratory, cardio vascular disease, stroke, ischemic heart disease and chronic obstructive pulmonary disease admissions. For every 10 $\mu g/m^3$ increase in short-term $PM_{2.5}$ there was a 2.2% increase in respiratory diseases admissions (95% CI = 1.9 to 2.6), and a 0.78% increase in cardiovascular disease (CVD) admission rate (95% CI = 0.5 to 1.0) (33).

65 There is strong evidence that links short term exposure (daily average levels) and mortality and morbidity, with effects not just due to exacerbation but also due to progression of underlying disease. A systematic review of time series analyses has shown that short term exposure to $PM_{2.5}$ associated with low birth weight, pre-term birth and small for gestational age births, and exposure to coarse PM of $\leq$ 10 μM was associated with small for gestational age births (34).

66 There are a lack of studies that address the effects of biomass burning and wildfire smoke on mortality, specifically a lack of studies on cardiovascular health and mortality. Most such studies have lacked statistical power.

67 There is quite a lot of evidence for an effect of wildfire exposure upon respiratory health including total respiratory admissions to hospital, chronic bronchitis and emphysema exacerbations and asthma exacerbations, and acute bronchitis, pneumonia and ED visits. Burning conditions appear to be an important factor in determining the hazard of the combustion derived particles.

68 Clinical studies of traffic generated exposures suggest that 1-2 hour exposures can result in physiological changes, suggesting that this may be sufficient to contribute to exacerbations of chronic disease, with changes in ST segment depression and loss of heart rate variability plus evidence of tachycardia, as effects of short term exposure.

69 Since the WHO Air Quality Guidelines, Global Update 2005 were issued, many new studies from Europe and elsewhere on both short and long-term exposure to $PM_{2.5}$ have been published. These studies provide considerable support for the scientific conclusions in the 2005 Guidelines and suggest additional health outcomes to be associated with $PM_{2.5}$ (18,19).

70 Among the major findings to date **in relation to shorter exposure times (e.g. individual or repeated short episodes of very high exposure, 1h, 24h):** There is further evidence from epidemiological studies that long-term (years) exposure to $PM_{2.5}$ is associated with both mortality and morbidity. The evidence base is weaker for PM_{10}, and hardly any long-term studies are available for coarse particles.

71 There is strong evidence from epidemiological studies that daily (24-hour average) exposures to PM are associated with both mortality and morbidity immediately and in subsequent days. Repeated (multiple day) exposures may result in larger health effects than the effects of single days.

72 While acute and long-term effects are partly interrelated, the long-term effects are not the sum of all short-term effects. Effects of long term exposure are much greater than those observed for short-term exposure suggesting that effects are not just due to exacerbations but may be also due to progression of underlying diseases (18,19).

4.3.iii. Long term exposure effects of $PM_{2.5}$

73 Studies relate outcomes to measured annual average concentration exposure. Measures of effect of long term exposure have focused on mortality as an end point (27).

Table 3.2. Comparison of excess relative risk for mortality from American cohort studies

Study	PM[1] metric[a]	Total mortality		Cardiopulmonary mortality		Lung cancer mortality	
		Excess RR[b]	95% CI (%)	Excess RR	95% CI (%)	Excess RR	95% CI (%)
Six City *(6,11)*	$PM_{2.5}$	13%	4.2–23	18%	6.0–32	18%	–11–57
Six City new *(6)*	$PM_{2.5}$	14%	5.4–23	19%	6.5–33	21%	–8.4–60
ACS *(6)*	$PM_{2.5}$	6.6%	3.5–9.8	12%	6.7–17	1.2%	–8.7–12
ACS new *(6)*	$PM_{2.5}$	7.0%	3.9–10	12%	7.4–17	0.8%	–8.7–11
ACS new *(6)*	$PM_{15-2.5}$	0.4%	–1.4–2.2	0.4%	–2.2–3.1	–1.2%	–7.3–5.1
ACS new *(6)*	$PM_{10/15}$	4.1%	0.9–7.4	7.3%	3.0–12	0.8%	–8.1–11
ACS new *(6)*	$PM_{10/15}$ SSI[c]	1.6%	–0.8–4.1	5.7%	2.5–9.0	–1.6%	–9.1–6.4
ACS extended *(5)*	$PM_{2.5}$ 1979–1983	4.1%	0.8–7.5	5.9%	1.5–10	8.2%	1.1–16
ACS extended *(5)*	$PM_{2.5}$ 1999–2000	5.9%	2.0–9.9	7.9%	2.3–14	12.7%	4.1–22
ACS extended *(5)*	$PM_{2.5}$ average	6.2%	1.6–11	9.3%	3.3–16	13.5%	4.4–23
AHSMOG *(2)*[d]	$PM_{10/15}$	2.1%	–4.5–9.2	0.6%	–7.8–10	81%	14–186
AHSMOG *(12)*[e]	$PM_{2.5}$	8.5%	–2.3–21	23%	–3.0–55	39%	–21–150
Veterans Administration[f]	$PM_{2.5}$	–10.0%	–15–-4.6				

[a] Increments are 10 µg/m³ for $PM_{2.5}$ and 20 µg/m³ for $PM_{10/15}$.
[b] Excess RR (percentage excess relative risk) = 100 × (RR–1), where the RR has been converted from the highest-to-lowest range to the standard increment (10 or 20) by the equation RR = exp(log(RR for range) × /range).
[c] PM measured with size-selective inlet (SSI) technology. The other PM measurements in ACS new *(6)* were based on dichotomous sampler with 15-µm and 2.5-µm cut-off points.
[d] Pooled estimate for males and females.
[e] Using two-pollutant (fine- and coarse-particle) models; males only.
[f] Males only, exposure period 1979–1981, mortality 1982–1988 (from Table 7 in Lipfert et al. *(13)*).
Source: US Environmental Protection Agency *(10)*.

74 The 1995 American Cancer Society sponsored study or "ACS" study, concluded that annual mortality due to cardiopulmonary disease and lung cancer increased in association with an increase in fine particulate matter concentrations. The results of the 2002 follow-up study showed significant associations between $PM_{2.5}$ exposure and elevated risks for cardiopulmonary and lung cancer mortality. The study found that each 10-microgram per-cubic-meter increase in long-term average $PM_{2.5}$ concentrations was associated with approximately a 4% increased risk of death from all natural causes, a 6% increased risk of death from cardiopulmonary disease, and an 8% increased risk of death from lung cancer (27).

75 The lung cancer risk associated with exposure to fine particulate matter is comparable to that faced by nonsmokers who live with smokers, and are exposed long term to secondhand cigarette smoke (35).

76 Further follow up studies indicate that the excess cardiovascular mortality was attributable to ischemic heart disease, dysrhythmias, cardiac arrest and heart failure For every 10mcg increment increase in $PM_{2.5}$ exposure there was an 8-18% increased risk of death (36).

77 Kloog et al observed an association between both short and long term exposure to $PM_{2.5}$ particles and risk of admission to hospital amongst over 65yo's in New England for respiratory, cardiovascular, cerebrovascular and diabetes related illnesses respectively: for every 10 µg/m³ increase in mean exposure over either 2 days or 7 years, mapped to a 10km by 10KM area, the rate of admission went up by 0.7% and 4.22% (37).

78 The same group also examined the relationship between long term exposure to $PM_{2.5}$ and incidence of AMI using the same methods. They found that an IQR increase in area $PM_{2.5}$ exposure (0.59 mcg/m3) was associated with a 16% increase in the odds of AMI. The authors conclude that it is important to consider spatial gradients within an area when examining the relationship between particulate exposure and cardiovascular events (38). Other studies have shown a link between 10 $\mu g/m^3$ increase in exposure and all cause mortality as well as risk of AMI. Exposure to $PM_{2.5}$ exerts an inflammatory effect and also leads to accelerated progression of atherosclerosis.

79 Cesaroni et al conducted a meta-analysis of 11 cohorts participating in the European Study of Cohorts for Air Pollution Effects (ESCAPE). They reported that long term exposure to ambient air pollution (particulate matter) is linked with incidence of coronary events. 100,166 participants were enrolled and were followed for an average of 11.5 years. According to the study result, an increase in estimated annual exposure to $PM_{2.5}$ of just 5 µg/m3 was linked with a 13% increased risk of heart attacks. The authors conclude that long term exposure to particulate matter is associated with incidence of coronary events, and this association persists at levels of exposure below the current European limit values (39).

80 There is further evidence for an effect of $PM_{2.5}$ exposure and development of diabetes in adults, cognitive decline in adults and neurological development in children as well as birth outcomes including pre term birth, LBW and SFD infants (30).

81 Birth cohort studies indicate that there is a relationship between long term $PM_{2.5}$ exposure and respiratory infections and asthma in young children. Work from Southern California has shown that lung function growth in children is reduced in areas with high PM concentrations and that the lung function growth rate changes in step with relocation of children to areas with higher or lower PM concentrations than before. Impacts of pollution on the prevalence of respiratory symptoms in children and adults were also found, though high correlation of various pollutants in those studies precludes attribution of the results of these studies to PM alone (40).

4.4 Nitrogen dioxide

82 *Nitrogen dioxide (NO_2) is one of a group of highly reactive gasses known as "oxides of nitrogen," or "nitrogen oxides (NOx)." NO_2 is the component of greatest interest and the indicator for the larger group of nitrogen oxides (24).*

83 NO_2 concentrations arising from point source exposures such as coal mine fires can be much higher than will be detected at area wide monitors and therefore exposed individuals who spend time at or near such point sources can experience short-term NO_2 exposures considerably higher than measured by the monitoring stations. Tropospheric, or ground level ozone, is not emitted directly into the air, but is created by chemical reactions between oxides of nitrogen (NOx) and volatile organic compounds (VOC) in the presence of heat and sunlight.

84 In addition to contributing to the formation of ground-level ozone, and fine particle pollution, NO_2 is linked with a number of adverse effects on the respiratory system. NOx react with ammonia, moisture, and other compounds to form small particles. These small particles penetrate deeply into sensitive parts of the lungs and can cause or worsen respiratory disease, such as emphysema and bronchitis, and can aggravate existing heart disease, leading to increased hospital admissions and premature death.

85 Current scientific evidence links short-term NO_2 exposures, ranging from 30 minutes to 24 hours, with adverse respiratory effects including airway inflammation in healthy people and increased respiratory symptoms in people with asthma. Also, studies show a connection between breathing elevated short-term NO_2 concentrations, and increased visits to emergency departments and hospital admissions for respiratory issues, especially asthma.

86 NO_2 exposure concentrations are of particular concern for susceptible individuals, including people with lung disease such as asthma and COPD (chronic obstructive pulmonary disease), children, and the elderly.

87 There is a very high level of correlation between the effects of NO_2 exposure and particulate pollution and ozone exposure. Given the weight of evidence in support of $PM_{2.5}$ and ozone exposure as the main constituents of airborne pollution contributing to adverse health outcomes, NO_2 will not be considered further in this report.

4.5 Sulfur dioxide

88 *Sulfur dioxide (SO_2) is one of a group of highly reactive gases known as "oxides of sulfur." SO_2 is linked with a number of adverse effects on the respiratory system (24).*

89 Current scientific evidence links short-term exposures to SO_2, ranging from 5 minutes to 24 hours, with an array of adverse respiratory effects including bronchoconstriction and increased asthma symptoms.

90 This is particularly important for asthmatics at elevated ventilation rates (e.g., while exercising or playing.) Studies also show a connection between short-term exposure and increased emergency departments visits and hospital admissions for respiratory illnesses, particularly for at-risk populations : children, elderly, and asthmatics.

91 SO_2 can react with other compounds in the atmosphere to form small particles. These particles penetrate deeply into sensitive parts of the lungs and can cause or worsen respiratory disease, such as emphysema and bronchitis, and can aggravate existing heart disease, leading to increased hospital admissions and premature death. There is evidence that exposure to SO_2 is associated with pre-term birth (34).

92 There is a very high level of correlation between the effects of SO_2 exposure and particulate pollution and ozone exposure. Given the weight of evidence in support of $PM_{2.5}$ and ozone exposure as the main constituents of airborne pollution contributing to adverse health outcomes, and the low level of SO_2 detected at air pollution monitoring stations in the Latrobe Valley, and low levels of sulphur in coal mined in the Latrobe Valley, SO_2 will not be considered further in this report.

4.6 Volatile organic compounds (VOC's) otherwise referred to as air toxics

93 Very little is known about the health effects of the release of so called air toxics from coal mine fires.

94 Finkelman has described the potential health impacts arising from burning coal beds and waste banks. Volatile elements are commonly enriched in coal deposits. Coal mine fires commonly volatilise these elements. The release of such elements into the atmosphere in the Latrobe Valley arising from the Hazelwood coal mine fire arguably represents an unquantifiable contribution to the additional risk to health associated with the fire. It is a matter of current research to determine whether volatilized elements and Polycyclic aromatic hydrocarbons (PAH's) can contribute to risk to health in association with particulate air pollution in either an additive or synergistic way (41).

95 Engel et al describe the gas emissions, minerals and tars associated with three ongoing coal mine fires in the Powder River Basin in Wyoming in the USA. They describe emission concentrations of CO_2, CO, and volatile gases such as benzene and volatile metal species at levels that may be of potential human health and environmental concern near the fires. They observe that the natural coal fires do not represent complete combustion and the emissions from the fires are not controlled (42). There are interactions amongst air pollutants that change the toxicity of the mixture. These occur at the level of physicochemical interactions in air as well at the biological level. Very few epidemiologic studies have examined the potential for interaction amongst pollutants. This is likely due to their moderate to high correlations. The existence of such pollutant mixtures makes it often difficult, in an uncontrolled setting, to determine either independent or synergistic effects of ambient air pollutants. Synergistic effects at the biological level between ultrafine particles and transition metals, and between particles and VOCs have been shown indicating larger combined impact on human health than would be expected for the separate entities.

96 Airborne particles of any kind can carry aero-allergens or toxic condensed vapours such that their impact can be substantially larger than without particles. There is a trend that the smaller the particles the stronger the adjuvant effects are. Limited evidence has been published suggesting that the nitrogen dioxide can enhance allergic responses. There is some evidence of potential interactions amongst pollutants and high temperature (19). Independent of known effects of ozone and $PM_{2.5}$ it is not possible to ascribe an independent health effect to short term exposure to ambient air pollution of the type associated with the Hazelwood coal mine fire, either in the short or the long term.

Table 1. Short term health effects that may be caused by exposure to smoke from a brown coal fire, identifying the substance in the smoke that causes each effect.

Affected group	Effect	Substance responsible
Unborn	Fetal death, low birth weight, premature labour, small for dates	CO (high dose),
Young children	Irritation to eyes and mucous membranes	$PM_{2.5}$ and larger particulates
	Irritation to Respiratory tract: new onset asthma and bronchitis, exacerbation of existing asthma creating ongoing reactive airways which are sensitive to further insults, Eg, virus. Increased morbidity and hospital admissions	Ozone and $PM_{2.5}$
	Impaired growth in lung function	Ozone and $PM_{2.5}$
	Death, long term cardiac and or neurological damage	CO (high dose),
	Psychosocial effects which increase the risk of family violence, drug and alcohol use, depression and anxiety, post traumatic stress and phobias.	$PM_{2.5}$ and larger particulates
Firefighters*	Irritation to eyes and mucous membranes	$PM_{2.5}$ and larger particulates
	Irritation to Respiratory tract: sinusitis, asthma and acute bronchitis, exacerbation of COPD, hospital admission and death	Ozone and $PM_{2.5}$
	Effects on cardiac conduction: arrhythmias, death	Ozone, $PM_{2.5}$ and CO
	Effect on coagulation state: stroke, heart attack and death	$PM_{2.5}$
	Short term: headache, nausea and lethargy due to hypoxia. Death. Long term: cardiac and or neurological damage, death	CO (high dose)
	Psychosocial effects which increase the risk of family violence, drug and alcohol use, depression and anxiety, post traumatic stress and phobias.	$PM_{2.5}$ and larger particulates

General Population*	Irritation to eyes and mucous membranes	$PM_{2.5}$ and larger particulates
	Respiratory tract: New onset sinusitis, asthma and acute bronchitis, Exacerbation of asthma, COPD, hospital admission and death	Ozone and $PM_{2.5}$
	Effects on cardiac conduction: arrhythmias, death	Ozone, $PM_{2.5}$ and CO
	Effect on coagulation state: stroke, heart attack and death	Ozone and $PM_{2.5}$
	Death, long term cardiac and or neurological damage	CO (high dose)
	Psychosocial effects which increase the risk of family violence, drug and alcohol use, depression and anxiety, post traumatic stress and phobias.	$PM_{2.5}$ and larger particulates
People with pre-existing ailments*: **Asthma, COPD, ischaemic heart disease, congestive cardiac failure.**	Irritation to eyes and mucous membranes	$PM_{2.5}$ and larger particulates
	Respiratory tract: sinusitis, exacerbation of asthma, exacerbation of COPD, Hospital admission and death	Ozone, $PM_{2.5}$
	Effects on cardiac conduction: arrhythmias, death	$PM_{2.5}$ and CO
	Effect on coagulation state: stroke and heart attack, hospital admission and death.	Ozone and $PM_{2.5}$
	Exacerbation of pre-existing congestive cardiac failure, hospital admission and death.	Ozone and $PM_{2.5}$
	Death, long term cardiac and or neurological damage	CO (high dose)
	Psychosocial effects which increase the risk of family violence, drug and alcohol use, depression and anxiety, post traumatic stress and phobias.	$PM_{2.5}$ and larger particulates

* Diabetes and current smoking may increase risk in these categories

Table 2. Medium to Long term health effects that may be caused by exposure to smoke from a brown coal fire, identifying the substance in the smoke that causes each effect

Affected group	Effect	Substance responsible
Unborn	Pre-term birth, Reduced birth weight due to intrauterine growth retardation.	Ozone
	Reduced post natal growth rates ,Reduced lung function growth rates	Ozone $PM_{2.5}$
Young children	Reduced lung function growth rates. Respiratory tract: new onset asthma and bronchitis, exacerbation of existing asthma creating ongoing reactive airways which are sensitive to further insults, Eg, virus. Increased morbidity and hospital admissions.	Ozone, $PM_{2.5}$
	Impaired growth in lung function	Ozone and $PM_{2.5}$
	Impaired neurological development.	Ozone
	Psychosocial effects which increase the risk of family violence, drug and alcohol use, depression and anxiety, post traumatic stress and phobias.	$PM_{2.5}$ and larger particulates
Firefighters*	Respiratory tract: increased risk of sinusitis, new onset asthma, acute bronchitis, exacerbation of pre-existing asthma creating ongoing reactive airways which are sensitive to further insults, Eg, virus. Exacerbation of unrecognized COPD. Increased risk of morbidity and hospital admissions, death.	Ozone and $PM_{2.5}$
	Effects on cardiac conduction: increased risk of arrhythmias, death.	Ozone and $PM_{2.5}$
	Effect on coagulation state: increased risk of stroke, heart attack and death	Ozone and $PM_{2.5}$
	Increased risk of Lung cancer and death	$PM_{2.5}$
	Psychosocial effects which increase the risk of family violence, drug and alcohol use, depression and anxiety, post traumatic stress and phobias.	$PM_{2.5}$ and larger particulates

General Population*	Respiratory tract: increased risk of sinusitis, new onset asthma, acute bronchitis, exacerbation of pre-existing asthma creating ongoing reactive airways which are sensitive to further insults, Eg, virus. Increased risk of morbidity and hospital admissions and death	Ozone and $PM_{2.5}$
	Effects on cardiac conduction: increased risk of arrhythmias, morbidity, hospital admissions and death	Ozone and $PM_{2.5}$
	Effect on coagulation state: increased risk of stroke, heart attack morbidity, hospital admissions and death	Ozone and $PM_{2.5}$
	Increased risk of Lung cancer and death	$PM_{2.5}$
	Long term cognitive decline	Ozone
	Psychosocial effects which increase the risk of family violence, drug and alcohol use, depression and anxiety, post traumatic stress and phobias.	$PM_{2.5}$ and larger particulates
People with pre-existing ailments: COPD, asthma, ischaemic heart disease, congestive cardiac failure	Irritation to Respiratory tract: sinusitis, exacerbation of asthma and of COPD, creation of hyper-reactive airway which are sensitive to further insults eg virus, and increased risk of bacterial infection. Increased risk of hospital admission, pneumonia and death.	Ozone and $PM_{2.5}$
	Effects on cardiac conduction: increased risk of arrhythmias, morbidity, hospital admissions and death	Ozone and $PM_{2.5}$
	Effect on coagulation state: increased risk of stroke, heart attack, morbidity, hospital admissions and death	Ozone and $PM_{2.5}$
	Increased risk of Lung cancer and death	$PM_{2.5}$
	Long term cognitive decline	Ozone
	Exacerbation of pre-existing congestive cardiac failure. Increased risk of hospital admission and death.	Ozone and $PM_{2.5}$
	Psychosocial effects which increase the risk of family violence, drug and alcohol use, depression and anxiety, post traumatic stress and phobias.	$PM_{2.5}$ and larger particulates

* Diabetes and current smoking may increase risk in these categories

5 Was the evidence available in February 2014 about these matters certain?

97 *Is there any gap in the medical understanding of the health effects of exposure to smoke from a brown coal fire?*

98 The medical understanding of the health effects of exposure to smoke from this brown coal mine fire has been hampered by the lack of any studies devoted specifically to the evaluation of the health sequelae of air pollutants emitted from a point source fire, in particular a coal mine fire in an open cut brown coal mine, arising as a consequence of a bushfire or wildfire, of unprecedented duration..

99 There is a gap in the understanding of the community mental health effects of exposure to a coalmine fire or bushfire, and the extent to which social determinants of health either protect against or exacerbate the mental health effects. Ie to what extent is community and individual mental health and resilience affected by exposure to a coalmine or bushfire?

100 The newly available evidence concerns long term effects of ozone and further evidence for short term effects of particulate exposures, particularly $PM_{2.5}$ on health measures in vulnerable communities.

101 "Is there a need for further research?" The answer is an unqualified "Yes". The directly relevant research questions pertain to the health effects of coal mine fires and bushfires, particularly of prolonged duration, although one hopes that the opportunity to study such a fire will not come again.

102 However, as climate change creates an increasing likelihood of extreme climate events and bushfires, plus the ongoing use of brown coal from open cut mines in Victoria's power stations, we can possibly expect the conditions which might give rise to a coalmine fire to arise more frequently in future.

6 Long term health study on the potential impact of the Mine fire.

103 *What are the essential features of the study including scope and duration of the study? Who should conduct the study?*

104 A long term study to examine the impact of the Mine fire should have as its objectives to improve the physical and mental health of the residents of the Latrobe Valley and to contribute to the body of knowledge arising from the fire to contribute to the better management of the health consequences of such fires in future. It is noted that a study of the Health of the residents of the Latrobe Valley was to be undertaken in the 1980's, so it is hoped that the opportunity to conduct such a study some 30 years later is not lost.

105 The study should be conducted under the governance of an independent Steering Committee responsible for the oversight of the Study, which is required to report annually to the Victorian Parliament. The study should be established with the intent that it will run for 20 years to ensure that it asks the right questions and achieves its objectives.

106 The Steering Committee should have an independent chair and include community representatives from the Latrobe Valley with specific reference to representatives of disadvantaged communities such as indigenous communities, as well as international and interstate academic representatives.

107 The research should be conducted by a consortium of academic entities from within Victoria including acknowledged experts in public health and environmental and occupational health and health economics. This structure will ensure that there is accountability for both the research direction and questions, and for maximal collaboration between entities within the state.

108 The research should be funded by the Victorian Government and the Mine Owner, and should examine the health effects of mine and fire generated air pollution and translate this into improved health outcomes for the community, as well as making important contributions to the international research literature.

109 The researchers should include:

1. Meteorologists and environmental health physicians, occupational health specialists and mathematical modelers to developing better predictive tools to describe the conditions under which environmental hazards might arise from such a fire and to enable the services involved to take such action as may be possible to prevent or reduce the chances of adverse health effects arising from such a fire from occurring again.
2. Epidemiologists and mental health experts who would participate in studies of the mental health impact of fire exposure, including effects on children and family violence, and the effect of psychosocial factors upon risk for adverse mental health outcomes and the effect of interventions to manage these outcomes.

110 The specific research could include:

1. An examination of the patterns of health service utilisation and adverse health outcomes (eg deaths) in the Latrobe Valley, following on from the exposure event (the fire) to determine whether these measures demonstrate an adverse health impact in the short term.

   This could include CUSUM analyses of the relevant events, and comparison with relevant prior time periods. CUSUM analysis tracks the cumulative sum of consecutive differences, whether positive or negative, between an individual measurement and a given standard or target. This allows the early detection of small incremental changes. This type of analysis has been shown to be of value in monitoring trends in the form of small variations, particularly when changes in absolute numbers of events are small. This is more useful than time series analysis for the detection of phenomena which are expected in small numbers, arising following a particular event.

2. A cohort study or time series analyses linking exposure data to health outcomes data along traditional epidemiological study lines.

111 The program of research to be undertaken could include linked epidemiological, health services and toxicological research. The research should be based on an inception cohort of the residents of the Latrobe Valley, with ongoing recruitment of young subjects, to create a population based cohort.

112 The psychosocial research should aim to promote resilience and improved health outcomes with a focus on creating community resilience and good health practices at both the individual and community level, for vulnerable communities in the Latrobe Valley in particular.

113 It will be important for measures that are collected to be comparable with those collected in other studies and with widely available population measures to ensure comparability and facilitate research cooperation.

114 The primary focus of the health impact assessment should be $PM_{2.5}$ particulate pollution exposure. There is a case to include broader measures of social determinants of health, measures of psychosocial morbidity and measures fo individual and community resilience as important predictive and outcome factors as well.

115 The final selection of metrics, outcomes and functions will require an interdisciplinary team with relevant expertise. The interdisciplinary research team may attempt to expand upon previous health impact assessments, with an attempt to develop methods and tools to integrate new evidence of effects of $PM_{2.5}$, such as its impact on birth outcomes and the incidence of childhood asthma..

7 Identify any key studies in relation to long term health effects of exposure to smoke from brown coal

116 There are no studies of the medium to long term health effects of exposure to smoke from brown coal, nor any well conducted studies of the health outcomes of exposure to smoke from coal mine fires.

117 The following references constitute the best compendium and most recent updates on health related to air pollution. These reports are highly relevant and offer insights into causal mechanisms and associations that have been quoted extensively in this report. In addition they provide insights into methods and scope of health impact assessments related to such exposures.

1. Air quality guidelines. Global update 2005. WHO Regional Office for Europe. Copenhagen 2006.
2. Review of evidence on health aspects of air pollution- REVIHAAP Project. (WHO Regional Office for Europe 2013
3. Report on Health risks of particulate matter from long-range transboundary air pollution. Joint WHO / Convention Task Force on the Health Aspects of Air Pollution. WHO European Centre for Environment and Health. Bonn Office. 2006.
4. Atkinson RW, et al. Epidemiological time series studies of $PM_{2.5}$ and daily mortality and hospital admissions: a systematic review and meta-analysis. Thorax. 2014 Apr 4.

References quoted in the Report

1. Australian National Pollutant Inventory. http://www.npi.gov.au/
2. Zou, Linda 2001, Microscopic nature and elemental composition of brown coal fly ash particles from a large coal fired power station, in APCSEET 2001 : Proceedings of the 3rd Asia-Pacific Conference on Sustainable Energy and Environmental Technologies, World Scientific, Singapore, pp. 392-397.
3. A Bell, Ecos 43, Autumn 1985 pp 24-28.
4. Galbally IE, et al. Surface Ozone at rural sites in the Latrobe valley and Cape Grim, Australia. Atmospheric Environment 1986;20:2403-2422.
5. Stracher, G. (2012). Natural and anthropogenic coal fires. Retrieved from http://www.eoearth.org/view/article/154790
6. Australian Government National Pollutant Inventory for Hazelwood power station 2010/2011. http://www.sourcewatch.org/index.php?title=Hazelwood_Power_Station
7. National Environment Protection Measure for Ambient Air Quality).http://www.environment.gov.au/topics/environment-protection/air-quality/air-quality-standards
8. National Environment Protection (Air Toxics) Measure http://www.scew.gov.au/nepms/air-toxics
9. Annual Report to the NEPC on the Air Toxics NEPM for Victoria for the year ended 31 December 2010. http://www.scew.gov.au/system/files/resources/771a0614-ec78-bd54-911c-34c20e2836f3/files/2010-report-vic.pdf
10. EPA Victoria Hourly Air Quality Data Table. http://www.epa.vic.gov.au/Our-work/Monitoring-the-environment/Air-quality-bulletins/Hourly-air-quality-data-table
11. Henry CR, et al. Myocardial Injury and Long-term Mortality Following Moderate to Severe Carbon Monoxide Poisoning. JAMA. 2006;295:398-402.
12. Lewis Goldfrank; Neal Flomenbaum; Neal Lewin; Mary Ann Howland; Robert Hoffman; Lewis Nelson (2002). "Carbon Monoxide". Goldfrank's toxicologic emergencies (7th ed.). New York: McGraw-Hill. pp. 1689–1704.
13. Farrow JR, Davis GJ, Roy TM, McCloud LC, Nichols GR (November 1990). "Fetal death due to nonlethal maternal carbon monoxide poisoning". Journal of Forensic Sciences 35 (6): 1448–1452.
14. Koren G, et al. A multicentre prospective study of fetal outcomes following accidental CO poisoning in pregnancy. ReproductiveToxicology1991;5:397-403.
15. Goldstein M. Carbon monoxide poisoning. Journal of Emergency Nursing. 2008;34: 538–542.
16. Air quality guidelines for Europe ; second edition (WHO regional publications. European series ; No. 91) Copenhagen. 2000. http://www.euro.who.int/__data/assets/pdf_file/0005/74732/E71922.pdf
17. WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Global update 2005 Summary of risk assessment. WHO Geneva 2006 http://whqlibdoc.who.int/hq/2006/WHO_SDE_PHE_OEH_06.02_eng.pdf
18. Review of evidence on health aspects of air pollution- REVIHAAP Project. WHO Regional Office for Europe. Copenhagen. 2013. http://www.euro.who.int/__data/assets/pdf_file/0020/182432/e96762-final.pdf
19. Review of evidence on health aspects of air pollution – REVIHAAP project: final technical report. WHO Regional Office for Europe. Copenhagen 2013. http://www.euro.who.int/en/health-topics/environment-and-health/air-quality/publications/2013/review-of-evidence-on-health-aspects-of-air-pollution-revihaap-project-final-technical-report.

20. Air quality guidelines. Global update 2005. WHO Regional Office for Europe. Copenhagen 2006. http://www.euro.who.int/__data/assets/pdf_file/0005/78638/E90038.pdf?ua=1

21. Michelle L. Bell*, Francesca Dominici†, and Jonathan M. Samet. A Meta-Analysis of Time-Series Studies of Ozone and Mortality With Comparison to the National Morbidity, Mortality, and Air Pollution Study. Epidemiology. 2005 July ; 16(4): 436–445.
22. Samoli E, et al. Acute Effects of Ambient Particulate Matter on Mortality in Europe and North America: Results From the APHENA Study. Environ Health Perspect. 2008;116(11):1480-1486.
23. Jarrett M, et al. Long-Term Ozone Exposure and Mortality. 2009;360:1085-95.
24. What Are the Six Common Air Pollutants? US EPA http://www.epa.gov/airquality/particlepollution/
25. Air Pollution Current standards for particles. EPA Victoria. http://www.epa.vic.gov.au/your-environment/air/air-pollution/particles-in-air
26. WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Global update 2005 Summary of risk assessment. WHO Geneva 200. http://whqlibdoc.who.int/hq/2006/WHO_SDE_PHE_OEH_06.02_eng.pdf
27. Report on Health risks of particulate matter from long-range transboundary air pollution. Joint WHO / Convention Task Force on the Health Aspects of Air Pollution WHO European Centre for Environment and Health. Bonn. 2006. http://www.euro.who.int/__data/assets/pdf_file/0006/78657/E88189.pdf
28. Pearson JF, et al. Association between fine particulate matter and diabetes prevalence in the US. Diabetes Care 2010;33:2196-2201.
29. Zanobetti A, et al. Cardiovascular damage by airborne particles: are diabetics more susceptible? Epidemiology 2002;13:588-592.
30. Brunekreef B, Forsberg B (2005). Epidemiological evidence of effects of coarse airborne particles on health. *European Respiratory Journal*, 26(2):309–318.
31. EPA (2009). *Integrated science assessment for particulate matter (final report)*. Washington, DC, United States Environmental Protection Agency http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546#Download
32. Atkinson RW, et al. Epidemiological time series studies of $PM_{2.5}$ and daily mortality and hospital admissions: a systematic review and meta-analysis. Thorax. 2014 Apr 4. doi: 10.1136/thoraxjnl-2013-204492.
33. Kloog I, Nordio F, Zanobetti A, Coull BA, Koutrakis P, et al. (2014) Short Term Effects of Particle Exposure on Hospital Admissions in the Mid-Atlantic States: A Population Estimate. PLoS ONE 9(2): e88578. doi:10.1371/journal.pone.0088578
34. Shah PS, et al. Air pollution and birth outcomes: A systematic review. Environment International 2011;37:498–516.
35. Pope, C., R. Burnett, M. Thun, E. Calle, D. Krewski, K. Ito, and G. Thurston, 2002. Lung Cancer, Cardiopulmonary Mortality, and long-term Exposure to Fine Particulate Air Pollution, Journal of the American Medical Association, 28(9):1132-1141.
36. UNECE Working group on Effects. Modeling and assessment of the health impact of particulate matter and ozone. Geneva,2004. http://www.unece.org/env/documents/2004/eb/wg1/eb.air.wg1.2004.11e.pdf
37. Kloog I, et al Acute and Chronic effects of particles on hospital admissions in New England. PLos One 2012;7 (4):e34664.
38. Madrigano, et al. Long term exposure to $PM_{2.5}$ and incidence of AMI. Environ Health Perspect 2013;121:192-6.
39. Cesaroni G, et al. Long term exposure to ambient air pollution and incidence of acute coronary events: prospective cohort study andmeta-analysis in 11 European cohorts from the ESCAPE project. BMJ 2014; 348:doi: 10.1136/bmj.f7412

40. Committee on Environmental Health American Academy of Pediatrics. Ambient Air Pollution: Health Hazards to Children. Pediatrics 2004;114;1699.
41. Finkelman RB. Potential health impacts of burning coal beds and waste banks. Int J Coal Geology 2004;59:19-24
42. Engle MA, et al. gas emissions, minerals and tars associated with three coal fires, Powder River Basin, USA. Science of the total environment 2012;420:146-159.