Woolcock Institute of Medical Research,
Centre for Air Quality and Health Research and Evaluation (CAR)

Review of the health impacts of emission sources, types and levels of particulate matter air pollution in ambient air in NSW

Produced for
NSW Environment Protection Authority
and
NSW Ministry of Health, Environmental Health Branch

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Abbreviations

ABS	Australian Bureau of Statistics
ACS	American Cancer Society
BC	Black carbon
BTRE	Bureau of Transport and Regional Economics
CSIRO	Commonwealth Scientific & Industrial Research Organisation
CVD	Cardiovascular disease
EC	Elemental carbon
EU	European Union
HEI	Health Effects Institute (US)
HNEAHS	Hunter New England Area Health Service
IARC	International Agency for Research on Cancer
MI	Myocardial infarction
NEPC	National Environment Protection Council (Australia)
NSW EPA	NSW Environment Protection Authority
OC	Organic carbon
OEH	NSW Office of Environment & Heritage
PAHs	Polycyclic aromatic hydrocarbons
PM	Particulate matter
$PM_{2.5}$	Particulate matter with an aerodynamic diameter less than 2.5 µm
PM_{10}	Particulate matter with an aerodynamic diameter less than 10 µm
$PM_{10-2.5}$	Coarse particulate matter with an aerodynamic diameter between 10 and 2.5 µm
$PM_{0.1}$	Particulate matter with an aerodynamic diameter less than 0.1 µm (also known as ultrafine particles)
PMF	Positive matrix factorisation (A method for estimating the different sources of PM measured in a location)
ROFA	Residual oil fly ash
UFP	Ultrafine particles
μg	Micrograms (a unit of measure for particle weight)
$\mu g/m^3$	Micrograms per cubic metre (unit of measure for particle concentration in air)
USEPA	US Environmental Protection Agency
VOCs	Volatile organic compounds
WHO	World Health Organisation

1. Executive summary

This report provides a comprehensive review of international and Australian evidence related to the health effects of exposure to outdoor (ambient) particulate matter (PM) air pollution. Background information is presented on the characteristics of PM and emission sources, and the various health effects that have been attributed to inhalation of ambient PM. These health effects include: mortality, respiratory, cardiovascular, cancer, central nervous system, developmental and reproductive effects. Many epidemiological studies have demonstrated that increased levels of ambient air PM pollution are associated with increases in mortality and, respiratory and cardiovascular morbidity. These studies form the majority of evidence presented in this report.

Although the relationship between exposure to ambient PM and adverse respiratory and cardiovascular health effects appears solid, the potential for publication bias in air pollution health studies may have influenced the findings of this review. Studies with evidence of significant positive associations are likely to have more chance of being published than studies which show that ambient PM exposure has no health effect. Nevertheless, consistent evidence from a variety of different types of studies, conducted in different locations, with different populations, and by different investigators, is less likely to be undermined by publication bias than evidence with a narrow focus from a few sources. The evidence that ambient PM has impacts on mortality and, respiratory and cardiovascular health is consistent across many sources and hence can be considered to be strong.

A focus of this report is the evidence of health effects associated with exposure to source-specific PM considered relevant to the NSW population. The PM emission sources considered in this report are:

- coal dust;
- coal-fired power stations;
- on-road vehicles (exhaust and non-exhaust emissions);
- on-road diesel vehicle exhaust;
- non-road diesel exhaust (including vehicles, machinery and shipping);
- solid fuel (wood) domestic heating;
- bushfires (including hazard reduction burning);
- crustal dust;
- sea salt; and
- biogenic sources (volatile organic compounds from vegetation).

This compilation of major PM emission sources in NSW was based on the 2008 NSW Environment Protection Authority (EPA) Air Emissions Inventory for the Greater Metropolitan Region (GMR). The Air Emissions Inventory estimates the amounts of source-specific PM emitted annually. It does not estimate population exposure to these emissions (although such data may be used in models to predict exposure). The population of NSW is unevenly distributed, with a heavy concentration in a narrow coastal belt. While this means that the majority of the population is exposed to some of these emission sources (such as vehicle emissions), less of the population is exposed to other

sources (such as bushfires, crustal dust and mining). Hence, it is likely that the presence of certain emissions does not correlate well with widespread population exposure in all areas.

Also considered in this report are the health impacts of PM of different sizes and composition. Namely the:

- health effects specifically attributed to exposure to coarse PM ($PM_{10-2.5}$);
- health effects associated with exposure to ambient concentrations of fine PM ($PM_{2.5}$) that are below the current NSW advisory reporting standard (annual average 8 $\mu g/m^3$);
- health effects specifically attributed to exposure to ultrafine PM ($PM_{0.1}$);
- health impacts of trace metals, organic molecules, sulphates and nitrates in PM.

Based on the evidence in this report, we have made suggestions for future actions to help minimise the health impacts of ambient PM in NSW and, to address evidence gaps. The recommendations that stem from this project are based solely on scientific conclusions on the health effects of PM air pollution and do not consider other issues relevant to policy formulation, such as economic considerations, environmental justice, and other political and social factors.

By world standards, the ambient levels of PM in urban NSW are low. However, the evidence presented in this report suggests that exposure to levels of PM that currently exist in NSW will have measureable adverse impacts on health, particularly in vulnerable people such as individuals with chronic respiratory and cardiovascular diseases, the elderly, and children. Reductions in PM air pollution in NSW are likely to result in health benefits, particularly for these most vulnerable groups.

PM emitted from different sources vary in physical and chemical characteristics. Evidence from controlled exposure studies and what is known about the biological responses to specific chemical components of PM suggest that not all emitted particles should be considered as equal with regards to their adverse health effects. However, to date, many epidemiological studies have not provided conclusive evidence to support this hypothesis. It is likely that the limitations of current methods used to estimate source-specific PM in air containing a mixture of particles derived from many sources, is a major reason for the inconclusive evidence of health effects associated with source-specific particles. Atmospheric transformation of emitted particles adds to the complexity of the problem. This difficulty in source attribution also applies to estimating the relative health impacts of PM from natural and anthropogenic sources. However, in urban environments, particles from anthropogenic sources add substantially to the total burden of ambient PM and to the overall health impact attributable to ambient PM. Therefore efforts to reduce these anthropogenic PM emissions are expected to result in improved health outcomes.

This review of evidence of the health effects of source-specific PM concludes that:

- all PM, regardless of source, should be considered detrimental to health;
- the evidence that coal dust in ambient air is associated with specific health effects in coal mining communities is inconclusive. There is presently limited evidence that in a community setting (that is, non-occupational exposure), that coal dust is more hazardous to health than PM from other sources;
- there is considerably more evidence of health effects linked to exposure to PM from combustion-related emissions (coal-fired power stations, on-road vehicles, diesel exhaust)

compared to other sources of PM. It should be noted that in general, combustion related sources of PM have been more intensively studied than other sources of PM;

- during times of severe dust storms and bushfires, the incidence of adverse health effects related to air pollution exposure is elevated, particularly in people with chronic respiratory disease. There is little evidence that individual particles emitted during these events are more toxic than PM from other sources. The increase in adverse health effects is likely to be a response to the increase in total ambient PM mass concentrations during dust storms and bushfires;
- methods are still evolving for determining the health impact of source-specific PM in the mix of particles in the atmosphere derived from a multitude of sources. At present it is not possible to provide a definitive ranking of the health impact of particles from different sources.

Analytical methods to determine the source of ambient PM (source apportionment) are becoming increasingly more sophisticated, and methods are being refined to provide more accurate estimates of exposure. These advances are likely to result in more accurate determinations, and differentiations, of the health impact of source-specific PM in the future.

There is evidence that fine particles ($PM_{2.5}$) are more detrimental to health and have a wider range of health effects than larger particles. However, larger inhalable particles are not benign, and it has been demonstrated that coarse particles ($PM_{10-2.5}$) have detrimental health impacts and that these health impacts differ from those associated with smaller particles.

There is no evidence of a threshold level of ambient $PM_{2.5}$, below which further reductions in concentration will not provide additional population health benefits.

The impact of PM composition on health is not well understood but it appears that the composition of particles affects the toxicity of PM. Some components of PM that come from combustion sources increase particle toxicity.

An important aspect of this review was to assess the consistency and quality of scientific evidence, and thus specify the confidence with which evidence could be used to base future actions to minimise health impacts. The assessment of quality was primarily applied to epidemiological studies as this evidence is most applicable to environmental and public health action. In an uncontrolled environment there are many confounding factors, known and unknown, that can influence the exposure-response relationship. In assessing the quality of evidence from an epidemiological study, methodological aspects were considered.

If the following occurred, then the quality of evidence was considered high:

- results were adjusted for confounding factors;
- methods minimised bias;
- measured ambient PM (or estimated source-specific PM) reflected true exposure; and
- exposure, confounders and health outcomes were measured in the same population.

High confidence that exposure to PM is associated with a health effect was provided when:

- the quality of evidence was high;
- the magnitude of the effect was high;
- the strength of the effect was high (uncertainty in the estimate of the health effect was low);
- the same or similar health effect was observed in a variety of different studies; and
- there was evidence of a biological mechanism to support observed health effects.

In order to have high confidence that exposure is likely to *cause* detrimental effects on health, requires an integration of evidence from several different types of scientific investigations including epidemiological, controlled human exposure (chamber) and, research animal toxicological studies.

For each section, we provide concluding statements based on the quality of the evidence. These statements are described below for clarity:

- "**The evidence is strong/there is strong evidence** ……" - there is sufficient evidence to have high confidence that the effects reported represent real effects;
- "**The weight of evidence is less than for**……." - the combined evidence for effects is not as strong as for other PM/sources/etc.;
- "**There is insufficient or limited evidence that**……" - the evidence is currently insufficient to conclude that a substance or component has an effect, either because the number of studies is too small, and/or there are substantial limitations in the study methods used. Where this is the case, the reason is presented in the preceding text;
- **"Inconsistent evidence**" - the evidence indicates that study results were inconsistent or varied, with some studies reporting positive (adverse) impacts and some studies reporting negative (or no) impacts;
- **"Lack of evidence" –** very little to no evidence exists.

A summary table of the findings is presented at the end of this Executive Summary (*Table ES*).

From this review of the evidence, we are highly confident that:

- increases in ambient $PM_{2.5}$ and PM_{10} mass are associated with increases in mortality and, increases in cardiovascular and respiratory morbidity;
- exposure to PM from combustion-related sources (coal-fired power stations, on-road vehicles, diesel exhaust) is associated with impacts on cardiovascular and respiratory health.

From these conclusions it is recommended that exposure of the NSW population to *all* PM (regardless of source) be minimised by reducing ambient PM levels to as low as possible. It would also be prudent to minimise exposure to PM from combustion sources and conduct further investigations of the health impacts of exposure to these particles. Given the proximity of much of the NSW population to on-road vehicle emissions, the health effects of this exposure should be a focus of further investigations. In order to minimise exposure to total ambient PM, those sources that emit the greatest quantities of PM in close proximity to people should be the focus of emission reduction actions.

Table ES. Summary of overall evidence of the health effects of different sized PM fractions and specific sources of PM

Particle type or source	Evidence of health effects	Limitations & considerations	Evidence of specific/greater harm from source compared with PM mass in general	Research indicated*	Summary located on page
Coarse PM ($PM_{10-2.5}$) mass	Some evidence of association with increased risk of mortality, and both respiratory and CV hospitalisations. Impacts on respiratory health appear to be at least equivalent to the impacts from $PM_{2.5}$.	A smaller number of health effects studies have considered coarse PM alone, (as studies of PM_{10} include both coarse and fine particles).	-	Australian research on health effects of coarse particles (where study design is feasible and there is statistical power to detect an effect).	162
Fine PM ($PM_{2.5}$) mass	**Sufficient & strong** evidence of causal association between long-term & short-term exposures and CV effects including mortality; likely causal association between long-term and short-term exposures and respiratory effects. No evidence of threshold for effects.		-		162
Ultrafine PM ($PM_{0.1}$) (particle number concentration (PNC))	Some, but limited evidence of short-term exposures associated with respiratory morbidity, CV mortality and CV function effects. The biological effects of controlled exposure to $PM_{0.1}$ are consistent with observed CV outcomes.	Number of studies on UFP reasonably small.	-	Australian research on health effects of UFP (where study design is feasible and there is statistical power to detect an effect).	162
PM composition	Epidemiological and toxicological studies that demonstrate compositional	Number of studies still reasonably small but	There is presently **insufficient evidence** to	Australian research on health effects of PM	172

Particle type or source	Evidence of health effects	Limitations & considerations	Evidence of specific/greater harm from source compared with PM mass in general	Research indicated*	Summary located on page
	variability in PM toxicity, are **strongly suggestive** that PM composition (and not solely mass) influences the health effects of PM, although the evidence is **inconsistent**. Health effects have been associated with PM containing metals such as vanadium and nickel. Organic compounds found in PM, especially those from fossil fuel combustion, can cause health effects. Health outcomes have been associated with both sulphates and nitrates in PM.	growing. It is likely that an array of elements/compounds may be associated with specific outcomes, rather than a specific PM component alone. While there are constituent candidates for adverse health effects (*e.g.* the transition metals vanadium and nickel, organic compounds, sulphates & nitrates), it is presently unclear whether the effects are specific to these components alone.	infer that a particular particle has a greater impact on health than other particles on the basis of composition alone. Because of the correlation between PM sulphates, nitrates, organic compounds and PM mass, it has been difficult to differentiate between the health effects due to these constituents compared with total $PM_{2.5}$ mass.	composition (where study design is feasible and there is statistical power to detect an effect).	
Coal dust	**Insufficient** evidence from community epidemiological studies to be confident in the health effects associated with environmental exposures. Occupational epidemiological studies and animal toxicity studies suggestive of the potential to impact respiratory health, and possibly CV health effects and cancer risk in surrounding communities (ie potentially	Small number of low quality community epidemiological studies conducted. Study methods limited by poor data on confounders eg smoking & SES; and poor exposure measures.	Insufficient evidence	Australian research on health effects of coal dust (where study design is feasible and there is statistical power to detect an effect).	71

Particle type or source	Evidence of health effects	Limitations & considerations	Evidence of specific/greater harm from source compared with PM mass in general	Research indicated*	Summary located on page
	harmful).				
Coal fired power station (CFPS) emissions	**Sufficient & strong** evidence of harm from direct emissions & formation of secondary aerosols, $PM_{2.5}$-sulphate. Metals found in CFPS emissions associated with health effects. **Insufficient** evidence of increased health risks for people living close to coal fired power stations.	Emissions are dispersed widely to affect broad regions and populations.	Some evidence related to secondary sulphates, although PM mass is highly correlated with $PM_{2.5}$-sulphate making it difficult to differentiate the health impacts from this source from those of PM in general.	Australian research on health effects of coal fired power station emission particles (where study design is feasible and there is statistical power to detect an effect) Many studies are from the eastern US where power stations contribute significantly to ambient PM.	85
On-road vehicles	**Sufficient & strong** evidence of increased risk of mortality, CV & respiratory morbidity, from epidemiological studies. Toxicological studies provide mechanistic evidence.	Traffic noise and SES may be confounding factors	Some evidence of increased risk of harm (particularly for CV health).	Australian research on health effects of on-road vehicles (where study design is feasible and there is statistical power to detect an effect) as they have not been extensively studied within Australia.	97

Particle type or source	Evidence of health effects	Limitations & considerations	Evidence of specific/greater harm from source compared with PM mass in general	Research indicated*	Summary located on page
On-road diesel vehicle exhaust emissions	**Strong evidence** of harm for CV and respiratory effects in human chamber studies and CV, respiratory, reproductive, developmental, cancer and allergy augmentation in animal studies. Potentially carcinogenic in ambient air (although carcinogenesis based on occupational studies).	Characterised by black carbon/ elemental carbon and $PM_{2.5}$ absorbance in studies, although these are not specific to only diesel exhaust. Therefore, it is difficult to distinguish diesel exhaust PM from PM from other combustion sources.	Uncertain, but harm is likely to be greater than for PM from non-combustion sources.	Health effects studies relevant to latest diesel engines and diesel fuel are necessary, as technological changes have meant lower emissions in recent years. Appropriately powered and designed Australian based studies may contribute to knowledge about diesel exposure and health outcomes.	110
Non-road diesel exhaust emissions	As above. Occupational exposure associated with increased lung cancer risk.	Very difficult to differentiate non-road from on-road diesel exhaust emissions in environmental settings. Few health studies conducted to specifically investigate non-road diesel exhaust emissions in such settings.	Uncertain, however likely to be greater than for on-road diesel vehicles due to lack of emission standards in Australia for non-road diesel sources.	Difficult to conduct environmental epidemiological studies on non-road diesel exhaust emissions. Chamber studies are possible.	118
Solid fuel (wood)	**Sufficient evidence** of association with increased adverse respiratory health	Health effects of outdoor exposure to PM from	Uncertain. The large number of potentially	Australian research on health effects of solid	127

Particle type or source	Evidence of health effects	Limitations & considerations	Evidence of specific/greater harm from source compared with PM mass in general	Research indicated*	Summary located on page
domestic heating	effects, particularly in children.	domestic wood-fired heating have not been extensively studied. Toxicity of wood smoke is dependent on type of wood burned and degree of combustion.	hazardous compounds in wood smoke suggests that there are health effects of exposure that remain to be determined.	fuel heating particles (where study design is feasible and there is statistical power to detect an effect).	
Bushfires and hazard reduction burning	**Sufficient and strong** evidence (including locally) of association with increased risk of respiratory health effects such as hospital admissions and presentations due to asthma and COPD. Inconsistent evidence of association with increased risk of mortality or CV morbidity.		Uncertain	Australian research on health effects of bushfire particles (where study design is feasible and there is statistical power to detect an effect).	135
Crustal dust	Some evidence of increased risk of daily mortality and respiratory morbidity during dust storm events in Australia. Evidence of these effects as well as CV effects due to crustal dust (other than during dust storm events), from overseas studies. Extreme levels during events particularly impact the elderly, the very young, and people with chronic disease.		Insufficient evidence. The few studies to have investigated the toxicity of crustal dust particles have shown that these particles exhibit similar toxic properties to PM from other sources.	Australian research on health effects of crustal dust particles from non-dust storm periods (where study design is feasible and there is statistical power to detect an effect).	143
Sea salt	**Insufficient evidence** of health effects.	Small number of health	Insufficient evidence.	Australian research on	149

Particle type or source	Evidence of health effects	Limitations & considerations	Evidence of specific/greater harm from source compared with PM mass in general	Research indicated*	Summary located on page
		effects studies on sea salt PM. A few studies show associations with mortality or respiratory morbidity outcomes, however results are inconsistent.		health effects of sea salt particles (where study design is feasible and there is statistical power to detect an effect).	
Biogenic sources	**None (lack of evidence)**. The health effects of PM from biogenic VOCs have not been investigated in Australia or elsewhere and so effects are unknown.	The contribution of biogenic VOCs to ambient PM concentrations in NSW is unknown.	No evidence available		151

*Consideration of feasibility of any health study needs to include the following factors: population available; population potentially exposed; estimation of exposure; prevalence of health effect in general population (these will help to determine whether the study will be adequately powered (statistically) to detect an effect if one truly exists); feasibility and accuracy of measuring /collecting health outcome/symptom/disease data; cost to conduct the study; and what data exits in other geographic areas.

2. Purpose and scope of the report, literature search strategy and, assessment of the quality of scientific studies

2.1 Purpose of the report

The purpose of this report is to provide a review of the evidence in the published literature of the health impacts of outdoor (ambient) particulate matter (PM) air pollution, with a particular focus on PM emissions that the population of NSW is likely to be exposed to. This report not only presents the evidence, but provides comment on the quality of evidence based on methodological considerations, and thus appraises the confidence with which the evidence may be used to inform policy and regulatory actions. Based on the evidence presented in this report, proposals for future actions to address knowledge gaps and minimise the health impacts of PM air pollution in NSW are proffered.

2.2 Scope of the report

This report reviews the published evidence of the health effects of exposure to PM air pollution with regards to:

- major PM emission sources (relevant to NSW);
- particle size; and
- particle composition.

In focussing on the health impacts of source-specific PM in NSW, the report includes data on PM emissions in NSW. However, the following aspects of PM pollution are considered outside the scope of this report:

- characterisation of population exposure to PM;
- PM exposure minimisation strategies;
- the physical and chemical properties of particles and their atmospheric transformation; and
- methods for monitoring ambient PM.

Epidemiology is particularly valuable when assessing population health impacts because it generally deals with the full spectrum of susceptibility in human populations. Therefore, the evidence provided in this report is heavily weighted towards epidemiological studies. This report is concerned with the health impacts of environmental exposures to the general population. However, where environmental epidemiological studies are lacking, studies of occupational exposures are alluded to in order to assess the potential for harm. Findings from controlled human exposure (chamber) studies and toxicological studies in animals are included for the purposes of evaluating the evidence that a plausible biological mechanism supports observed health effects. Studies using isolated cell

cultures are not directly applicable to the human condition. However where such studies are informative they are referred to.

The health effects of particulate air pollution have been studied ever since severe episodes of air pollution were linked to spikes in deaths and hospitalisations in the middle of the last century. Investigations have intensified since the 1990's, when novel methods allowed a more accurate determination of environmental exposure. This is a very large field of investigation. It was beyond the scope of this evidence review, and not the intent of this report, to include every reported epidemiological and toxicological study about the health effects of PM. Therefore, published reports of some investigations were not included, without intentional bias with regard to results (see *literature search strategy*).

2.3 Literature search strategy

Peer-reviewed scientific literature databases and websites (grey literature) were systematically searched. The **peer-reviewed literature databases** that were searched were:

- Web of Science™ from Thomson Reuters™; and
- PubMed of the US National Library of Medicine.

Peer-reviewed scientific literature searches combined search terms for particulate pollution ("particulate matter", "particulate air pollution", "particle air pollution", "particle pollution"), **with search terms specific to the topic being reviewed** (*e.g.* "wildfire", "bushfire", "forest fire", "hazard reduction burn", "prescribed burn" were combined in the search for *Chapter 5.7 Bushfires and hazard reduction burning*).

In Web of Science™, search terms were applied to the *Topic* search field, which means that the search term is searched in the Title, Abstract, Author-supplied Keywords and, Thomson Reuters™ Editor-supplied Keywords (KeyWords Plus®) of each article in the database. In PubMed, search terms were applied to the *Text Word* [tw] search field, which means that the search term is searched in the Title, Abstract, Medical Subject Headings (MeSH) and, Author-supplied Keywords of each article in the database.

For topics that yielded more than 7,000 peer-reviewed articles in the initial search, such as occurred for *on-road vehicles* and *coal-fired power station emissions*, database searches were limited to reviews and Australian studies. Other articles were retrieved from an examination of the reference lists of review articles. In all searches of the peer-reviewed literature, retrieved articles were first examined by title, then by abstract and lastly (where relevant) by review of the whole text. Further articles were obtained by a review of the reference lists of articles that met the inclusion criteria (see below).

For identification of information from grey literature on websites (*Table 2.3.1*), where the website had a search function, the search terms for particulate pollution applied to searches of the peer-reviewed literature were used to search the website.

Whether evidence was included in this review was based on methodological considerations. Study results were *not* the basis for including or excluding evidence. Health and outdoor particulate air

pollution is a large field of scientific investigation. For some topics it was beyond the capacity of this project to include all of the evidence that met our inclusion criteria. In such instances, individual studies that demonstrate the consensus view are presented. Where the sum of evidence was without clear direction this inconsistency is stated.

Table 2.3.1 Websites searched

International	
• World Health Organisation (WHO)	http://who.int/en/
• WHO, Regional Office for Europe	http://www.euro.who.int/en/home
• US Environmental Protection Agency (US EPA)	http://www.epa.gov.au/
• European Union	http://europa.eu/index_en.htm
• European Study of Cohorts for Air Pollution Effects (ESCAPE)	http://escapeproject.eu/
• Committee on the Medical Effects of Air Pollutants (COMEAP)	http://comeap.org.uk/
• The Health Effects Institute	http://www.healtheffects.org/
• International Agency for Research on Cancer (IARC)	http://www.iarc.fr/
• American Heart Association	http://www.heart.org/
• European Respiratory Society	http://ersnet.org/
National	
• The Commonwealth Scientific and Industrial Research Organisation (CSIRO)	http://www.csiro.au/
• Australian Institute of Health and Welfare	http://www.aihw.gov.au/
• Department of the Environment	http://www.environment.gov.au/
• Department of Health	http://www.health.gov.au/
• Department of Infrastructure and Regional Development	http://www.infrastructure.gov.au/
• Department of Industry and Science	http://www.innovation.gov.au/
• National Environment Protection Council	http://www.scew.gov.au/
• National Health and Medical Research Council (NHMRC)	http://www.nhmrc.gov.au/
• Australian Nuclear Science & Technology Organisation (ANSTO)	http://www.ansto.gov.au/
• Minerals Council of Australia	http://www.minerals.org.au/
NSW	
• NSW Environment Protection Authority (EPA)	http://www.epa.nsw.gov.au/
• NSW Health	http://www.health.nsw.gov.au/
• NSW Office of Environment and Heritage	http://www.environment.nsw.gov.au/
• NSW Mining	http://www.nswmining.com.au/

While study results did not form the basis of the selection of evidence provided in this report, it was also not feasible to present every study that showed negative results (*i.e.*, lack of effect). There are many reasons why a study may observe no effect, including aspects of study design or inaccuracies in measurement methods. If many studies did not show an association between exposure and a health effect this is indicated.

The following inclusion/exclusion criteria were applied to literature search results:

Inclusion criteria

- Large (100,000's of subjects), multi-centre epidemiological studies
- Australian epidemiological studies

- Epidemiological studies that gave due consideration to measured and unmeasured confounders of the exposure-health response relationship
- Controlled exposure studies in humans or animals, where exposure was by way of inhalation of concentrated or ambient levels of air pollution particulates
- Reviews of scientific evidence.

Exclusion criteria

- Pollution exposure studies and source speciation data without health outcome data (this is a review of the health effects of exposure, not a review of exposure assessments)
- Epidemiological studies not considered relevant to NSW, either because of the nature of the emission source or, the characteristics of the population (*e.g.*, conducted in a region of the world with very high morbidity and mortality unrelated to air pollution)
- Epidemiological studies in which the study cohort and/or pollution exposure data was deemed too small to have validity
- Quantitative studies without characterisation of the uncertainty of effect estimates (*i.e.* lacking confidence intervals or standard errors)
- Studies (epidemiological and toxicological) of the effects of air pollution gas/particle mixtures without consideration of the effects of PM were generally not included or, if they were included, the point was made that the health effects may be due to gases that co-exist with PM
- Cell culture or molecular studies that provided no mechanistic insight into health effects observed in epidemiological studies.

2.4 Assessment of the quality of scientific studies

Assessment of the quality of individual scientific studies included in this review was based on methodological considerations. Due to the high variability in study methods and sometimes poor descriptions of these methods, it was not possible to quantify study quality. The methods in *Table 2.4.1* indicate attributes that were considered when assessing the quality of scientific studies.

The assessment of the quality of scientific studies has not been reported for each individual study throughout this review. However, deficiencies in study methods are discussed where these deficiencies are considered to result in severe limitations. Furthermore, where the majority of scientific studies for a particular type of PM exposure are of poor quality, the conclusions relating to the health effects of that exposure were that there is **limited or insufficient evidence** of associations between exposure and health effects.

It is possible that many studies may suggest health effects associated with exposure but because the studies are of poor quality, our overall conclusion is that there is limited evidence that exposure results in adverse health outcomes. Conversely, fewer studies of high quality may provide strong evidence that exposure is associated with adverse health outcomes.

Table 2.4.1 Methodological considerations when assessing the quality of scientific studies

Method	Study quality	
	Higher	**Lower**
Results adjusted for possible confounding variables	✓	
Methods minimised bias (*e.g*. objective rather than subjective measures of health)	✓	
Regional health outcome data (*e.g*. state- or county-level) applied to a community-based study		✓
Exposure, confounders and health outcomes measured in the same population	✓	
Proxy measures such as industrial output used to assign exposure		✓
Exposure estimated from the measurement of ambient PM	✓	
Biological effects in chamber and toxicological studies compared with effects in appropriate controls	✓	

3. Particulate matter in ambient air

Particulate matter (PM) in ambient (outdoor) air is a mixture of particles from natural and anthropogenic sources. These particles have solid and liquid components. Natural sources of PM include sea spray, bushfires, crustal dust, vegetation (pollen, fungal spores), volatile organic compounds (VOCs) and animals (fragments of organisms). Anthropogenic sources of PM include combustion engines (vehicular or stationary), power stations, mining, other industrial processes, agriculture and, domestic heating appliances. The composition, size and concentration (by mass or particle number) of PM in ambient air are temporally and spatially variable (Seinfeld and Pandis 2006). These characteristics are influenced by the type and proximity of PM emissions, atmospheric conditions and, the landscape topography and built environment (Vardoulakis et al. 2003, Querol et al. 2004, Putaud et al. 2010, Tong et al. 2012). Emitted particles undergo substantial physical and chemical change in the atmosphere as a consequence of particle to particle interaction, reactions with gases, clouds and rain, and sunlight-driven (photochemical) reactions (Poschl 2005, Seinfeld and Pandis 2006). These atmospheric processes undoubtedly affect both the real and measured health impacts of source-specific PM (Poschl 2002). Unfortunately, there is limited understanding of the quantitative and qualitative aspects of these atmospheric processes (Lohmann and Feichter 2005, Poschl 2005).

The spatial variability of ambient PM concentrations is regionally specific. For example, in Los Angeles there is considerable variation in PM concentrations in ambient air that is 40 kilometres apart, whereas there is far less spatial variability in the cities of Pittsburgh and Boston (US EPA 2009). The variability in Los Angeles relates to the mountainous topography surrounding the city and the extensive road networks. In close proximity to sources of PM and in built environments (such as street canyons), spatial variability can be extreme. In a study in Copenhagen, on working days when traffic was greatest, roadside PM levels were more than double those measured 500 metres away on a 20 metre high rooftop (Ketzel et al. 2003).

The temporal variability of PM concentrations is dependent on those influences that change over time. These temporal influences include:

- PM emission sources that are dependent on human activity, such as traffic and, PM emissions that are dependent on natural events[1], such as dust storms and sea swells; and
- atmospheric conditions including seasonal climatic factors and localised weather.

Despite this variability, extensive regional PM characteristics exist (Putaud et al. 2010). Long-term average PM mass concentrations have been used to assess the health impact of chronic exposure to PM (Dockery et al. 1993, Pelucchi et al. 2009). The impact of short-term exposures to PM have been investigated in time-series studies (Bell et al. 2004).

Exposure to ambient PM is associated with dose-dependent increases in morbidity and mortality (Anderson et al. 2012). The health effects of ambient PM are interrelated with some or all of:

[1] 'Natural' events such as dust storms and bushfires may be influenced by anthropogenic activities that change the land surface and climate.

- PM emission sources;
- PM composition;
- PM size;
- atmospheric conditions;
- climate;
- transformation of emitted PM in the atmosphere;
- human activity; and
- human physical condition (Poschl 2005).

Scientific research, which among other things, measures the impacts of PM (and other pollutants) on health, is the centrepiece of air quality management as described by Bachmann (Bachmann 2007). The variability of PM in ambient air and the atmospheric transformation of emitted PM are problematic to the determination of the health impacts of source-specific PM (Harrison and Yin 2000, WHO 2006b, Bell 2012). However, various methods are available to determine the health effects attributable to source-specific PM.

In summary, ambient PM is characterised by size, composition, origin, source and concentration. These characteristics are discussed in the following chapters.

3.1 PM size fractions

On the basis of aerodynamic diameter[2] PM is classified as <10 micrometres (µm) (PM_{10}), <2.5 µm ($PM_{2.5}$) or <0.1 µm ($PM_{0.1}$) (*Figure 3.1.1*). Coarse particles are between 10 and 2.5 µm in diameter ($PM_{10-2.5}$). $PM_{2.5}$ particles are known as "fine particles" and $PM_{0.1}$ particles are known as "ultrafine particles". Thus PM_{10} encompass coarse, fine and ultrafine particles whereas $PM_{2.5}$ includes fine and ultrafine particles. The term "total suspended particles" applies to particles of any size, suspended in ambient air. Since particles larger than 30 µm remain suspended for a relatively short period of time before deposition (compared to smaller particles) (de Kok et al. 2006), total suspended particles are in effect all ambient particles up to approximately 30 µm in diameter.

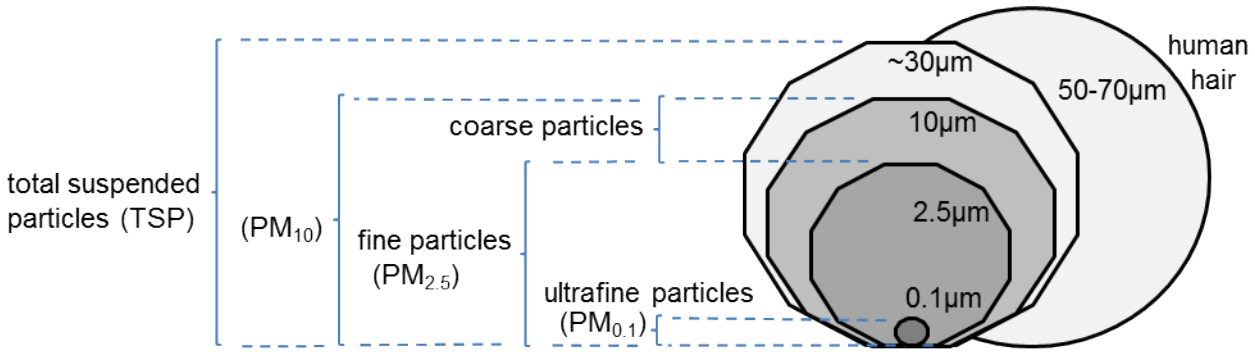

Figure 3.1.1 PM size fractions: showing the aerodynamic diameters in comparison to the diameter of a human hair (not to scale)

[2] Aerodynamic diameter is an expression of a particle's aerodynamic behaviour as if it were a perfect sphere with unit-density. It allows the comparison of particles with irregular shapes and different densities and defines particle transport in air and particle deposition. Chen W. and Fryrear D.W. (2001). Aerodynamic and geometric diameters of airborne particles. *Journal of Sedimentary Research* 71: 365-371.

In ambient air, rather than there being an even distribution of the different sized particles, particles are distributed into populations or "modes" (Whitby et al. 1972, Cao 2013). These modes represent different relative total masses, volumes, surface areas and numbers of particles (*Figure 3.1.2*). Coarse particles ($PM_{10-2.5}$) and fine particles larger than ultrafine ($PM_{2.5-0.1}$) are responsible for the bulk of PM mass in two distinct modes called "coarse" and "accumulation", respectively (Whitby et al. 1972, Heintzenberg 1989, Cao 2013). Fine particles ($PM_{2.5}$) are responsible for the majority of ambient particle surface area (Heintzenberg 1989). Ultrafine particles ($PM_{0.1}$) contribute by far the greatest number of particles to ambient PM, in what is called the "nucleation" mode (Whitby 1978, Heintzenberg 1989, Seinfeld and Pandis 2006). Regional variations in PM distributions do occur however, this basic multi-modal distribution of PM size in ambient air has been observed in urban (Whitby et al. 1972, Whitby 1978), remote (Bigg 1980) and source-specific environments (Morawska et al. 2008a).

Ambient air PM is usually measured on the basis of mass with units of $\mu g/m^3$. However, in situations where ultrafine particles are likely to impact human health it has been suggested that it may be more pertinent to measure particle number (Reche et al. 2011).

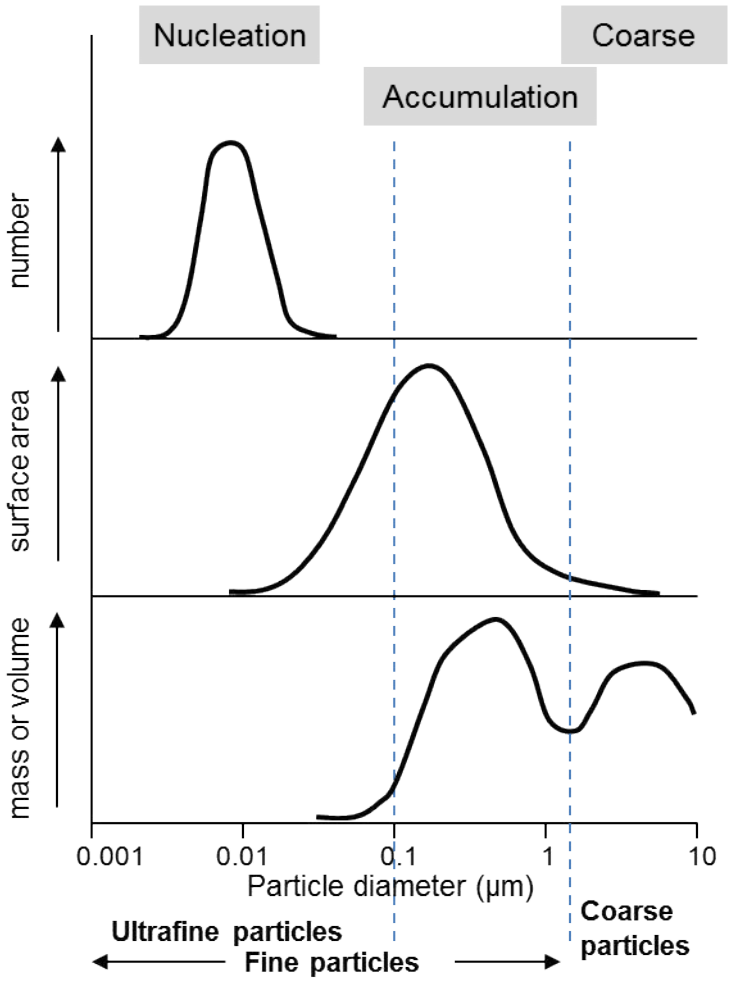

Figure 3.1.2 Number, surface area and mass/volume lognormal distributions of ambient air PM showing the different modes

PM modes are the result of particle emissions, *in situ* (atmospheric) particle formation (gas-to-particle), particle maturation, particle interaction and transformation (coagulation, condensation, evaporation and photochemical reactions) and, particle removal (settling, deposition and rainout). The particles in each of the modes have various characteristics (*Table 3.1.1*) that contribute to the extent of population exposure and the potential for health harms.

- **Coarse particles ($PM_{10-2.5}$)**

Coarse particles are produced by mechanical processes such as wind erosion, re-suspension by traffic and, agricultural and surface mining activities. Crustal dust, pollens, fungal spores, biological debris and sea salt are examples of coarse particles. Because of their large size, these particles exist in the atmosphere for only a few hours or days before gravitationally depositing onto surfaces (Hinds 1999). The relatively short time that coarse particles remain in the atmosphere means that the effects of these particles tend to occur close to source (<100 kilometres) (US EPA 2009). However, intercontinental transport of desert dust occurs and some of this dust is coarse particles (US EPA 2009). If weather conditions are conducive, dust storms in Australia can deposit coarse particles from central Australia into cities on the east coast and hundreds of kilometres out to sea (Chan et al. 2005). Inhaled coarse particles deposit in the upper airways.

- **Fine particles ($PM_{2.5}$)**

Fine particles are the direct result of emitted particles, condensation of sulphates, nitrates and gaseous organic molecules, reactions in water droplets and, coagulation of smaller ultrafine particles (John et al. 1990, Seinfeld and Pandis 2006). They include particles from combustion processes and, particles formed via photochemical reactions of VOCs and oxides in the presence of sunlight (Hinds 1999). As well as the aforementioned species, heavy metals, ammonium, elemental and organic carbon, bacteria, and viruses are components of accumulation mode particles. Fine particles can stay suspended in ambient air for days to weeks and be transported hundreds or thousands of kilometres (US EPA 2009). Consequently, fine particles are more homogeneously distributed on regional scales than either coarse or ultrafine particles. The aggregate surface area and mass of fine particles is large and there is great propensity for both adsorption (adhesion to the particle surface) and absorption (taking into the particle mass) of trace elements and potentially toxic molecules to these particles. Fine particles are removed from the atmosphere via deposition or the formation of cloud droplets and being rained out. Inhaled fine particles deposit in the lower airways.

- **Ultrafine particles ($PM_{0.1}$)**

Ultrafine particles are generated by high temperature combustion or formed from nucleation of atmospheric gases. It is hypothesised that ultrafine particles are dominated by primary anthropogenic combustion emissions in highly polluted urban settings and by nucleation of gases in remote sites (US EPA 2009). Organic and elemental carbon, trace metals, and sulphates are components of ultrafine particles from combustion sources. Sulphuric acid vapour and water vapour are the major nucleating gases (US EPA 2009). Ultrafine particles are quickly removed from the atmosphere (minutes to hours) via diffusion to surfaces or, coagulation, adsorption and condensing into fine particles. As a consequence, these particles are not transported far in ambient air and have great spatial and temporal variability. The ambient ultrafine particle

concentration can be significantly higher near to traffic without similar increases in PM_{10} and $PM_{2.5}$ levels (Morawska et al. 2004). Inhaled ultrafine particles are small enough to be able to deposit in tissues outside of the lungs.

Particle size is important to the potential for health harms because different sized particles deposit in different areas of the respiratory tract and elicit different biological responses. Smaller particles penetrate into the smaller airways. A crude distinction is that fine particles deposit in the terminal bronchioles and alveoli of the lung where gas exchange occurs, whereas coarse particles deposit primarily in the larger primary bronchi (Kelly and Fussell 2012). However, a particle diameter of 2.5 µm does not have biological significance. When the US EPA first set ambient air quality standards based on particle size, the selection of $PM_{2.5}$ was driven by consideration of what PM fraction could be measured at the time rather than any health significance of this specific sized particle (US EPA 2009, Cao 2013).

The surface area of particles available for adsorption of toxic chemicals also has the potential to influence the health impact of PM. For a given volume of inhaled ambient air there is a greater potential for the ingestion of adsorbed chemicals associated with fine than course particles (Kelly and Fussell 2012).

Table 3.1.1 Characteristics of coarse, fine and ultrafine particles

	Coarse particles ($PM_{10-2.5}$)	Fine particles ($PM_{2.5}$)	Ultrafine particles ($PM_{0.1}$)
Mode	Coarse	Accumulation	Nucleation
Formation and sources	Break-up of solids and droplets Erosion of land Suspension of dust Re-suspension of road debris (tyre/brake wear) Ocean spray Ash (black smoke) from uncontrolled combustion Construction and demolition Disturbance of surfaces (agriculture, mining, quarrying, unpaved roads) Biogenic emissions (pollen, fungal spores)	Condensation of atmospheric gases Coagulation of ultrafine particles Reactions of component gases of particles Evaporation of water droplets containing dissolved gases Combustion of fossil and biomass fuels Industrial processes (smelters, refineries, steel mills, mining)	Nucleation and condensation of atmospheric gases High temperature combustion (including vehicle exhausts)
Composition	Organic and elemental carbon Sulphates Nitrates Chlorides Oxides of crustal elements Sea salt Plant and animal debris Bacteria	Organic and elemental carbon Sulphates Nitrates Ammonium Metals Organic compounds Water Bacteria Viruses	Organic and elemental carbon Sulphates Metals Organic compounds
Physical characteristic of mode	Large mass	Large surface area	High particle number
Spatial/temporal variability	High	Low	Very high
Atmospheric life-time	Minutes to days	Days to weeks	Minutes to hours
Distance travelled	Usually <10's kms	100's-1000's kms	Usually <1's kms
Removal process	Gravitational deposition Scavenging by rain	Gravitational deposition Formation of cloud droplets and rain out	Coagulation, adsorption, condensation, diffusion to rain droplets
Extent of physiological deposition	Upper airways (primary bronchi)	Lower airways (terminal bronchioles and alveoli)	Extra-pulmonary organs

Modified from the *US EPA Integrated Science Assessment for Particulate Matter, December 2009* (US EPA 2009).

3.2 PM composition

Ambient PM is composed of biologic material, geologic material, hydrocarbons, ammonium, nitrates, sulphates, sea salt, acid aerosols, water and trace metals absorbed or attached to a carbonaceous core (de Kok et al. 2006, Seinfeld and Pandis 2006). Sulphate, nitrate, ammonium, organic and elemental carbon, and transition metals, are predominant in fine particles whereas crustal materials (silicon, calcium, magnesium, aluminium and iron), sea salt and biological material (pollen and spores) are more commonly present in coarse particles (Seinfeld and Pandis 2006). These constituents are the predominant constituents of PM and falsely imply a relatively simple mix of compounds. With the exception of volatile compounds that reside almost exclusively in the air surrounding particles, PM can be composed of almost any chemical compound or element. PM is usually composed of several main constituents that account for the majority of the measured PM mass and many trace constituents. *Figure 3.2.1* shows the composition of coarse and fine particles collected from an urban background site in the Netherlands. Even when the mass of seven constituents and 27 metals were accounted for, 33% of the mass of the coarse particles and 19% of the mass of the fine particles remained unaccounted for, highlighting the complexity of PM composition.

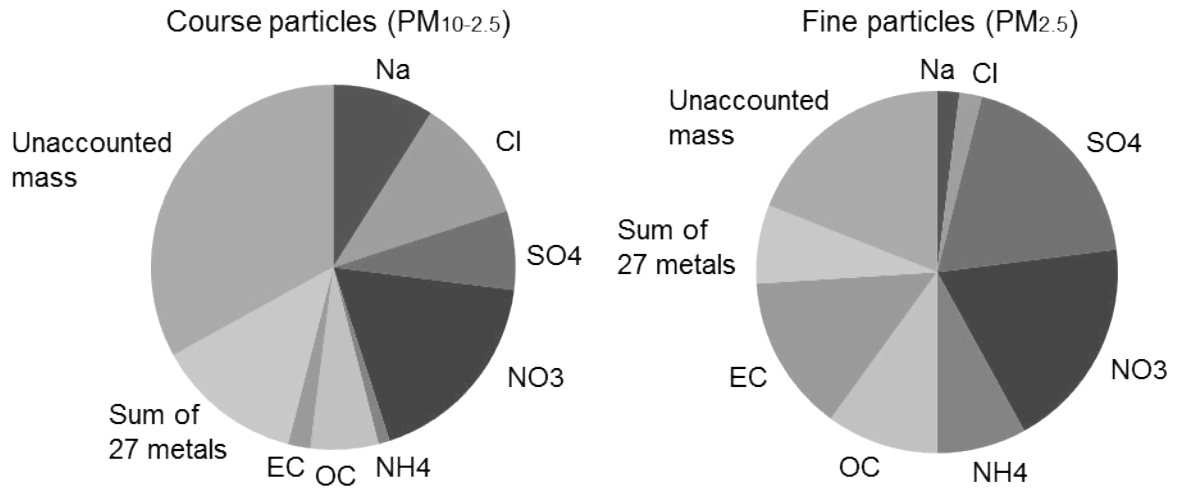

Figure 3.2.1 Percent mass concentration of course and fine PM from an urban background site in the Netherlands. Na, sodium; Cl, chloride; SO4, sulphate; NO3, nitrate; NH4, ammonia; OC, organic carbon; EC, elemental carbon.
Source: (Weijers et al. 2011).

Generally, hazardous inorganic pollutants in PM, such as metals, are relatively well characterised with respect to both their toxicity and abundance in the atmosphere compared to organic compounds, such as polycyclic aromatic hydrocarbons (PAHs), which may be equally (or more) hazardous (Finlayson-Pitts and Pitts Jr. 1997, Poschl 2002, de Kok et al. 2006). Organic compounds account for 20-60% of PM mass and include a wide variety of individual species (more than 200 have been identified), each one present at low concentrations (Perrino 2010, Kelly and Fussell 2012). Often investigations fail to identify most of the organic matter at the level of individual species

(McMurry 2000, Poschl 2005). Monitoring is generally concerned with identifying organic compounds known to be harmful or species that reflect specific emission sources.

Carbon in the form of elemental carbon, organic compounds or black carbon accounts for a significant proportion of the mass of PM. Black carbon is an operationally defined term that refers to carbon that is measured by light absorption, and is an indicator of a variable mix of material from combustion sources (Janssen et al. 2012). The terms elemental carbon and black carbon are often used interchangeably, however in terms of determining source apportionment it is more important that organic carbon is distinguished from these other forms of carbon. Organic carbon is primarily derived from biogenic sources whereas elemental or black carbon originates from anthropogenic sources (Szidat et al. 2004). The elemental carbon/total carbon ratio in $PM_{2.5}$ is as high as 50% in urban air samples and close to traffic, and approximately 30% in rural and alpine air (Poschl 2005).

The composition of PM varies spatially, reflecting the proximity of various sources of PM, atmospheric chemistry and, weather conditions. For example, $PM_{2.5}$ collected at a remote coastal site in Tasmania (Cape Grim) has a high salt content relative to $PM_{2.5}$ collected in urban Sydney (Mascot) reflecting the influence of ocean spray at the coastal site (*Table 3.2.1*). The $PM_{2.5}$ in Sydney has a higher proportion of black carbon compared to the $PM_{2.5}$ at the remote coastal site reflecting the influence of combustion emissions in Sydney.

Table 3.2.1 Average percent mass of $PM_{2.5}$ black carbon and salt at Cape Grim, Tasmania and Mascot, Sydney, 1998-2008

Site	Black Carbon	Salt
	% mass	
Cape Grim, Tasmania (*coastal, remote*)	5±3	43±21
Mascot, Sydney (*urban*)	21±10	16±15

Source: (ANSTO 2008)

Many PM emission sources influence the composition of ambient PM. Minerals and metals are a larger component of PM collected from roadsides as a result of the re-suspension by traffic of crustal dust and road and vehicle wear particles that are deposited on road surfaces (Putaud et al. 2004). Sulphates in PM are formed from sulphur dioxide emissions, of which electricity generation from fossil fuels is a major source. Nitrates are formed from oxides of nitrogen, with road traffic being a major source. Intensive agriculture is a source of ammonia emissions. As well as spatially, the composition of PM also varies temporally (including seasonally) reflecting both changes in atmospheric and climatic conditions, and human activity.

Many potentially toxic trace elements are associated with PM. The extent to which an element associated with PM will be a toxic hazard will depend on the chemical characteristics of the element, size of the particle and, the form in which the element exists. For example, trace elements of PM exist as oxides, sulphonates, nitrates, carbonates and, associated with organic molecules (Schroeder et al. 1987). These molecular forms influence the bio-availability of trace elements and consequently their toxicity. Trace elements in PM vary widely with location. In general, PM in remote areas contain elements of crustal origin (as well as some elements indicative of

anthropogenic sources) whereas PM in urban areas contain a far greater number of different trace elements from many different anthropogenic and natural sources (Schroeder et al. 1987).

The composition of PM undoubtedly has the potential to influence the capacity of PM to affect health and there is increasing evidence that PM constituents have health impacts. However, causative relations and mechanisms are little understood (Poschl 2005, Rohr and Wyzga 2012). Different chemical constituents of PM can induce the same biological response (Kelly and Fussell 2012). This makes associating a particular health effect with a specific constituent of PM a difficult proposition. Toxicological studies conducted under well-controlled conditions are important to determining whether constituents of PM have specific biological effects. If these studies suggest that a PM constituent has a biological effect, the constituent can then be investigated in epidemiological studies of ambient air exposures, however ambient exposures are subject to a multitude of co-pollutants that can have additive, synergistic or even antagonistic effects (Kelly and Fussell 2012). The relatively low concentrations of PM constituents in ambient air add to the difficulty in determining which constituents are associated with health effects. Furthermore, most studies that examine the health effects of ambient PM composition investigate a single particle component or constituent class (*e.g.*, metals). There are correlations among components of PM and this makes selection of a single component for study problematic. If a component of PM is found to have a health effect it is often not possible to establish whether the chosen component is responsible for the effect, whether the association is due to the component's correlation with another component that is the true toxic agent, or whether multiple components are jointly responsible (Stanek et al. 2011). Despite these limitations, when taken together, particle speciation studies that consider multiple components of ambient air PM can contribute data on the effects of individual components.

Epidemiological studies have found significant positive associations between health effects and various constituents of PM including: sulphate, nitrate, lead, potassium, organic carbon, elemental carbon, selenium, sodium, bromine, nitrates, iron, nickel, copper, chromium, scandium, titanium, vanadium, aluminium, arsenic, zinc, silicon, chloride, manganese and aldehydes (Kelly and Fussell 2012, Rohr and Wyzga 2012). Not all of the studies used multiple pollutant models to adjust for the effects of other pollutants and no single species was always associated with a health effect. Of 25 epidemiological studies examining the health effects of elemental and organic carbon (one of the most studied PM constituents), 12 studies reported significant associations for both elemental and organic carbon, three studies reported significant associations for elemental carbon only, and five studies reported significant associations for organic carbon only (Rohr and Wyzga 2012). While carbon has been a common species of interest in epidemiological studies, metals have been a focus of toxicological studies in which animals or humans were exposed to concentrated ambient particles. The metals most commonly observed as being associated with health endpoints in these studies were vanadium and nickel (Chen and Lippmann 2009, Rohr and Wyzga 2012).

Against the proposition that the composition of PM influences the health impacts of PM is the observation that relationships between morbidity/mortality and PM mass concentration are largely consistent across different locations despite differences in PM composition (Harrison and Yin 2000, Anderson et al. 2007).

Knowing the composition of PM enables the determination of the contribution of different sources of particle emissions to the pool of PM in ambient air (*source apportionment*). Proportions of various chemical compounds and elements in PM are a "fingerprint" of the sources that contribute to the ambient PM mixture at a given place and time. Composition fingerprints are used to identify the most likely source of ambient particles that are ultimately the result of particle emissions and physical and chemical reactions in the atmosphere. It is possible to identify with some assurance the contributing emission source(s) responsible for ambient PM collected at a time and place. A technique known as Positive Matrix Factorisation (PMF) incorporates both particle emission sources and the atmospheric modification of emitted particles into models to estimate the sources of ambient PM. PMF enables PM collected at a single site to be linked to various emission sources at different locations. PMF has been used to determine the source of ambient PM in NSW (Cohen et al. 2011, Hibberd et al. 2013, NSW EPA 2013b).

3.3 Origin (primary versus secondary particle formation) and atmospheric transformation of PM

PM emitted directly into the atmosphere are called ***primary* PM**. New particles that are formed in the atmosphere from gases via the processes of nucleation and condensation are called ***secondary* PM**. Although secondary PM is formed in the atmosphere, this does not exclude human involvement in the formation of such particles. Oxides of nitrogen and sulphur dioxide, both products of combustion, are involved in secondary PM formation (Nemmar et al. 2013a). Indeed, it has been estimated that on a global scale the mass of PM resulting from anthropogenic gaseous emissions is more than double the mass of directly emitted anthropogenic particles (Hinds 1999).

Ambient air PM is continually undergoing physical and chemical transformation. Particles change their size and composition via condensation of gases, by evaporation of gases from the particle surface, by colliding and adhering with other particles to become larger, by chemical reaction, or by activation in the presence of supersaturated water to become fog and cloud droplets (Seinfeld and Pandis 2006). The largely ultrafine particles emitted in diesel exhaust are a good example of the transformation that particles undergo in ambient air. Post-emission these particles rapidly disperse, quickly become larger though coagulation with other particles in the air and, on exposure to ozone and sunlight develop an affinity with water that increases their likelihood of removal from the atmosphere through precipitation (Maricq 2007). In another example of atmospheric transformation of PM, crustal dust particles can mix with anthropogenic sources of air pollution and increase their concentration of metals and sulphates (Huang et al. 2010).

The distribution of PM in ambient air at any given time and place is dependent on both primary PM emissions and secondary PM formation. These processes are dependent upon anthropogenic, biogenic, and geologic activity, climate and weather.

3.4 Sources of ambient PM emissions (including sources relevant to NSW)

There are many sources of ambient PM emissions.

Natural sources include:

- crustal dust eroded from the earth's surface or emitted from volcanoes;
- sea salt from ocean spray;
- incompletely combusted vegetative material from bushfires;
- gaseous precursors emitted from vegetation;
- microorganisms (*e.g.* moulds and bacteria); and
- plant and animal fragments (*e.g*. pollen and insect fragments).

Anthropogenic sources include:

- fuel combustion associated with power generation, residential heating and vehicles;
- construction and demolition activities;
- mining and quarrying;
- plant, animal and crustal material re-entrained through agricultural practices;
- prescribed burning to reduce fire hazards or clear land;
- waste disposal;
- other industrial processes;
- fugitive dust from unpaved roads;
- tyre, brake and road wear from traffic; and
- re-suspension of crustal dust by on-road and non-road vehicles.

Globally, the largest sources of ambient PM are crustal dust and sea salt. Anthropogenic sources contribute less to global ambient PM than these natural sources. Estimates range from <10% to 50% of total global PM emissions being from anthropogenic sources (Hinds 1999). However, in urban regions the human contribution to ambient PM often exceeds the contribution from natural sources and results in higher ambient PM concentrations compared to natural background sites (Querol et al. 2004, Cohen et al. 2010, Putaud et al. 2010, Cohen et al. 2011). It has been estimated that in 2008 in the Greater Metropolitan Region (GMR) of NSW, the great majority of PM emissions were of anthropogenic origin (*Table 3.4.1*).

Table 3.4.1 Anthropogenic and non-anthropogenic PM_{10} and $PM_{2.5}$ emissions in the Greater Metropolitan Region (GMR) and the Sydney region according to the NSW Air Emissions Inventory 2008

Source	Proportion of PM emissions (%)			
	GMR		Sydney region (within the GMR)	
	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Anthropogenic	72.8	81.2	80.9	91.9
Non-anthropogenic	27.2	18.8	19.1	8.1

Source: (NSW EPA 2012a)

The physical and chemical characteristics of ambient PM are in part determined by the emission sources. Thus, there is the potential for ambient PM from different sources to have different health effects. Sources of fine ($PM_{2.5}$) and coarse ($PM_{10-2.5}$) particles and common PM constituents are shown in *Table 3.4.2*.

Table 3.4.2 Sources of fine ($PM_{2.5}$) and coarse ($PM_{10-2.5}$) particles and common PM constituents

PM constituent	Primary $PM_{2.5}$		Primary $PM_{10-2.5}$		Secondary $PM_{2.5}$ (from gaseous precursors)	
	Natural	**Anthropogenic**	**Natural**	**Anthropogenic**	**Natural**	**Anthropogenic**
Sulphate	Sea spray	Fossil fuel combustion	Sea spray	-	Oceans, wetlands, volcanos, bushfires	Fossil fuel combustion
Nitrate	-	Vehicle exhaust	-	-	Soils, bushfires, lightning	Fossil fuel combustion, vehicle exhaust
Minerals	Crustal dust	Vehicular re-suspension, agricultural activity, forestry, construction, demolition	Crustal dust	Vehicular re-suspension, agricultural activity, forestry, construction, demolition	-	-
Ammonium	-	Vehicle exhaust	-	-	Wild animals, undisturbed soil	Vehicle exhaust, farm animals, sewage, fertilized land
Organic carbon	Bushfires	Prescribed burning, wood-heating, vehicle exhaust, tyre wear, industrial processes	Soil matter	Tyre and road wear	Vegetation, bushfires	Motor vehicles, prescribed burning, wood-heating, solvents, industrial processes
Elemental carbon	Bushfires	Vehicle exhaust (mainly diesel), biomass burning	-	Tyre and road wear	-	-
Metals	Volcanos	Fossil fuel combustion, smelting, vehicle brake wear	Crustal dust, organic debris	-	-	-
Bioaerosols	Viruses, bacteria	-	Plant and insect fragments, pollen, fungal spores	-	-	-

(-) minor source or no known source

Modified from the US EPA Integrated Science Assessment for Particulate Matter, December 2009 (US EPA 2009).

The contribution of various PM emission sources to ambient PM can be estimated using *particle speciation*, *source apportionment* and, *emission inventories*.

Particle speciation involves measuring the elemental and molecular constituents of ambient PM collected at a specific place and time. **Source apportionment** utilises sophisticated modelling incorporating real-time monitoring data (including ambient PM and gaseous pollutant concentrations, wind direction, and anthropogenic pollutant emission activity) to assign PM emission sources to specific chemical compositions of ambient PM. From the chemical composition of collected PM and modelling of real-time data, the contributions of known PM emission sources to ambient PM can be estimated (Viana et al. 2008). Different source apportionment models have been shown to give consistent source apportionment results (Hopke et al. 2006) and, when evaluated in health effects analyses the different methods have been shown to give comparable health risk estimates (Ito et al. 2006, Stanek et al. 2011). However, there are limitations in the methods to estimate the contribution of different sources to ambient PM collected at a point in time and place (Watson et al. 2008, Selvaraju et al. 2013). For example, it is not always clear which sources of PM should be included in a particular model. There is considerable uncertainty and inherent subjectivity in assigning sources of PM pollution to chemical combinations in ambient PM (Stanek et al. 2011). Ambiguity in source classification and misclassification of sources are potential problems (Grahame and Hidy 2007, Viana et al. 2008). Misclassification of source(s) is likely to be non-differential, biasing results towards the null. Therefore a measured significant association between a PM source and a health effect will likely reflect a real association. Methods that incorporate data from multiple sources and use multiple analysis techniques overcome some of the limitations of individual approaches (Viana et al. 2008, Selvaraju et al. 2013). Source contributions to ambient PM can be determined sufficiently to estimate whether a contribution is small, about the same as others, or dominant (Watson et al. 2008). Watson *et al* considers this degree of precision adequate to enable these estimates to be used to make decisions on emission controls.

Source apportionment was used in the Upper Hunter Valley Particle Characterization Study (Hibberd et al. 2013) to identify domestic wood heaters and emissions of sulphur dioxide (from sources such as power stations) as dominant sources of $PM_{2.5}$ at two different locations in the Upper Hunter Valley. Source apportionment has also been used to estimate sources of $PM_{2.5}$ and coarse PM in Melbourne, Sydney, Brisbane and Adelaide (Chan et al. 2008). Results were relatively similar for all four cities, with crustal dust and sea salt the dominant contributors to coarse PM and combustion a major contributor to $PM_{2.5}$. Ammonium sulphate contributed to $PM_{2.5}$ to a greater extent in Sydney than the other cities, likely reflecting a higher contribution from coal-fired power stations in that city.

Emission inventories list the amount of pollutants discharged into the atmosphere by source type in a given area and can be used to evaluate the impact of emission abatement strategies. The physical and chemical processes affecting PM emitted into the atmosphere are extremely complex and it cannot be determined from inventories alone what contribution a single source makes to ambient PM concentrations. Therefore, population exposure to source-specific PM cannot be precisely determined from emission inventories. In NSW an air emissions inventory covering the GMR is maintained by the NSW EPA (http://www.epa.nsw.gov.au/air/airinventory.htm). Inventories take several years to complete and the latest published inventory details emissions for 2008 (NSW EPA 2012a).

Sources of PM relevant to the population of NSW

As discussed above, particle speciation with source apportionment is preferred over emission inventories to estimate exposures to source-specific PM. However, in NSW only a few source apportionment studies have been conducted and they have measured specific chemical components of PM with a view to discerning the contribution of specific sources of PM in the vicinity of where the ambient PM was collected (Cohen et al. 2011, Hibberd et al. 2013, NSW EPA 2013b). Many sources of PM have not been investigated in source apportionment studies in NSW. Therefore the *NSW Air Emissions Inventory 2008 for the GMR* (NSW EPA 2012a) was utilised to determine which sources of PM pollution were to be reviewed in this report (*Table 3.4.3*). The PM emission sources chosen for review in this report are:

- coal mining (coal dust);
- coal-fired power stations;
- on-road vehicles;
- on-road diesel vehicle exhaust;
- non-road diesel exhaust;
- solid fuel (wood) domestic heating;
- bushfires and hazard reduction burning;
- crustal dust;
- sea salt; and,
- biogenic sources (volatile organic emissions from vegetation).

The GMR covers 57,330 km^2 that encompasses the sub-regions of Sydney, Newcastle and Wollongong. Given that the majority of the population of NSW resides in the Sydney region, consideration was given to both PM emissions in the Sydney region and the GMR when deciding which sources of PM were to be evaluated in this report. The total of the proportions of source-specific PM in *Table 3.4.3* for the GMR and Sydney region do not equal 100% because there are a variety of sources of PM not included in the table. Notable exclusions are iron/steel production (1.4% of PM_{10} and 3.1% of $PM_{2.5}$ in the GMR), waste disposal (6.0% of PM_{10} in the Sydney region) and ceramics production (4.1% of $PM_{2.5}$ in the Sydney region). Industry restructuring in NSW since the emissions data was collected in 2008 is likely to have decreased the contribution of emissions from some of these sources (Spoehr 2014). There are undoubtedly other sources of ambient PM, not evaluated in this report, which are of concern to local communities in NSW.

Table 3.4.3 Major sources of PM_{10} and $PM_{2.5}$ in the Greater Metropolitan Region (GMR) and the Sydney region according to the NSW Air Emissions Inventory 2008

Source	PM emission details[a]	Proportion of total PM emissions (%)			
		GMR		Sydney region (within the GMR)	
		PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Coal mining	Extraction of coal, transfer and loading of coal, removal of overburden and, wheel generated dust	42.5	22.6	2.0	0.4
Coal-fired power stations	Coal combustion for generation of electrical power, transport, crushing and loading of coal and, wheel generated dust	5.3	8.5	-	-
On-road vehicles (excluding diesel exhaust)	Tyre, brake and road wear and, petrol vehicle exhaust. The sum of four sources defined in the inventory: 1. All non-exhaust PM 2. Passenger vehicle petrol exhaust 3. Light duty commercial petrol exhaust 4. Others - exhaust	1.3	2.4	6.2	6.3
On-road diesel exhaust	The sum of two sources defined in the inventory: 1. Heavy duty commercial diesel exhaust 2. Light duty diesel exhaust	0.9	2.9	4.1	6.9
Non-road diesel exhaust	Non-road vehicle and equipment exhaust. The sum of the five largest users of diesel fuel in the GMR defined in the inventory: 1. Industrial vehicles & equipment 2. Locomotives 3. Commercial boats 4. Aircraft ground operations 5. Shipping	2.7	8.3	4.4	7.2
Solid fuel domestic heating	Wood fuel-fired residential space heaters	6.2	18.8	27.7	46.5
Bushfires and hazard reduction burning	Biogenic and geogenic emissions from bushfires and planned fires	2.8	7.6	2.7	4.0
Crustal dust and biogenic sources	Fugitive windborne PM from agricultural lands and unpaved roads (includes both biogenic & geogenic material) and, PM from quarrying. The sum of two sources defined in the inventory: 1. Fugitive-windborne 2. Land-based extractive activity	3.6	2.0	2.8	0.8
Sea salt	Sea salt aerosols from the action of surface wind on the open ocean	23.0	10.5	15.0	3.8
Total		88.3	83.6	64.9	75.9

[a] Emission details are as per the NSW Air Emissions Inventory Technical Reports 2008 (NSW EPA 2012b, NSW EPA 2012e, NSW EPA 2012f, NSW EPA 2012d, NSW EPA 2012c).

(-) Indicates not reported in the inventory

3.5 Ambient PM standards and PM concentrations in ambient air

In response to worsening air quality last century, specific air pollution events associated with a large number of excess deaths and, increasing evidence of the impact of PM on health, regulations were enacted in the major economic countries in an attempt to limit PM pollution (Bachmann 2007, Anderson et al. 2012). Current and previous air quality recommendations, guidelines and standards for PM_{10} and $PM_{2.5}$ from the WHO, major industrial countries/zones and Australia, are shown in *Table 3.5.1*. In *Table 3.5.1*, distinctions are not made between air quality guidelines (recommended concentration of PM based on health criteria), air quality standards (includes compliance criteria, monitoring procedures and may take into account economic and social considerations as well as human health protection) and, advisory reporting standards (required monitoring and assessment without compliance criteria) (Department of Sustainability 2010). Air quality standards consist of an indicator (*e.g.* PM_{10} or $PM_{2.5}$ mass), an averaging time (*e.g.* 24-hours or 1-year), a mass concentration level in ambient air and, a statistical form (*e.g.* limit not to be exceeded more than once per year) (Watson et al. 1995). The statistical form is not indicated in *Table 3.5.1*. The first air quality standards were published by the then newly formed US EPA in 1971 (Bachmann 2007). Based on health effects and air quality monitoring techniques, PM_{10} was put forward by the US EPA in 1987 as the best indicator of excessive PM exposure. $PM_{2.5}$ was considered for regulation by the US EPA in the mid-1980's but it took until 1997 to justify $PM_{2.5}$ as an indicator to be regulated (US EPA 1997). Currently the standard for $PM_{2.5}$ in Australia is an advisory reporting standard (NEPC 2003). However, the most recent NEPM review recommended the introduction of compliance standards for $PM_{2.5}$ (NEPC 2014).

The mass concentration of PM in ambient air broadly follows a gradient from low concentrations in natural and rural environments to high concentrations in urban and industrial sites (Monn et al. 1995, Querol et al. 2004, Poschl 2005, Putaud et al. 2010). In Europe, these gradients are superimposed on extensive regional variation and tend to be more evident for PM_{10} than $PM_{2.5}$ (Putaud et al. 2010). In NSW the natural/rural to urban site gradient does not occur for PM_{10}, with annual average concentrations in regional sites often exceeding urban sites (Department of Sustainability 2010, NSW EPA 2013b). This is likely due to the significant influence of dust storms, bushfires and agriculture on PM_{10} levels in NSW (NSW EPA 2013b). Indeed, the highest average maximum PM_{10} concentrations have been the result of the contributions of bushfires and dust storms in 2002 and 2003 and, dust storms in 2009 (Department of Sustainability 2010, NSW Department of Environment Climate Change and Water 2010). However, it would be expected that ambient $PM_{2.5}$, which is significantly influenced by anthropogenic sources, would follow a rural (lower concentration) to urban (higher concentration) gradient in NSW (*Figure 3.5.1*).

Table 3.5.1 Current and previous recommendations, guidelines and standards for ambient air PM_{10} and $PM_{2.5}$

Authority	Current			Previous		
	Year	PM_{10}	$PM_{2.5}$	Year	PM_{10}	$PM_{2.5}$
WHO	**2013**[a]	Limits should be maintained Short-term guideline for coarse particles ($PM_{10-2.5}$) may be considered	Likely to achieve health benefits through lowering limit below 2006 guidelines	**2006**[b]	Annual average: 20 µg/m^3 24-hour average: 50 µg/m^3	Annual average: 10 µg/m^3 24-hour average: 25 µg/m^3
US EPA	**2013**[c]	Annual average: none 24-hour average: 150 µg/m^3	Annual average: 12 µg/m^3 24-hour average: 35 µg/m^3	**2006**[d]	Annual average: none (revoked) 24-hour average: 150 µg/m^3	Annual average: 15 µg/m^3 24-hour average: 35 µg/m^3
European Commission	**2008**[e]	Annual average: 40 µg/m^3 24-hour average: 50 µg/m^3	Annual average: 25 µg/m^3 24-hour average: none	**1999**[f]	Annual average: 40 µg/m^3 24-hour average: 50 µg/m^3	Annual average: none 24-hour average: none
China, Ministry of Environment Protection	**2012**[g]	Annual average: Class I[h] 40 µg/m^3 Class II[i] 70 µg/m^3 24-hour average: Class I 50 µg/m^3 Class II 150 µg/m^3	Annual average: Class I 15 µg/m^3 Class II 35 µg/m^3 24-hour average: Class I 35 µg/m^3 Class II 75 µg/m^3	**1996**[g]	Annual average: Class I 40 µg/m^3 Class II 100 µg/m^3 24-hour average: Class I 50 µg/m^3 Class II 150 µg/m^3	Annual average: none 24-hour average: none
Australia, National Environment Protection Council	**2014**[j]	Annual average: consideration of 20 µg/m^3 24-hour average: consideration of 45 µg/m^3 and 40 µg/m^3	Convert annual average and 24-hour average advisory standards to formal standards at previous values	**2003**[k]	Annual average: none 24-hour average: 50 µg/m^3	Annual average: 8 µg/m^3 (advisory only) 24-hour average: 25 µg/m^3 (advisory only)

[a] (WHO 2013c)
[b] (WHO 2006a)
[c] (US EPA 2013)
[d] (US EPA 2006)
[e] (EU 2008)
[f] (EU 1999)
[g] (Cao 2013)
[h] Applies to natural areas
[i] Applies to residential/commercial areas
[j] (NEPC 2014)
[k] (NEPC 2003)

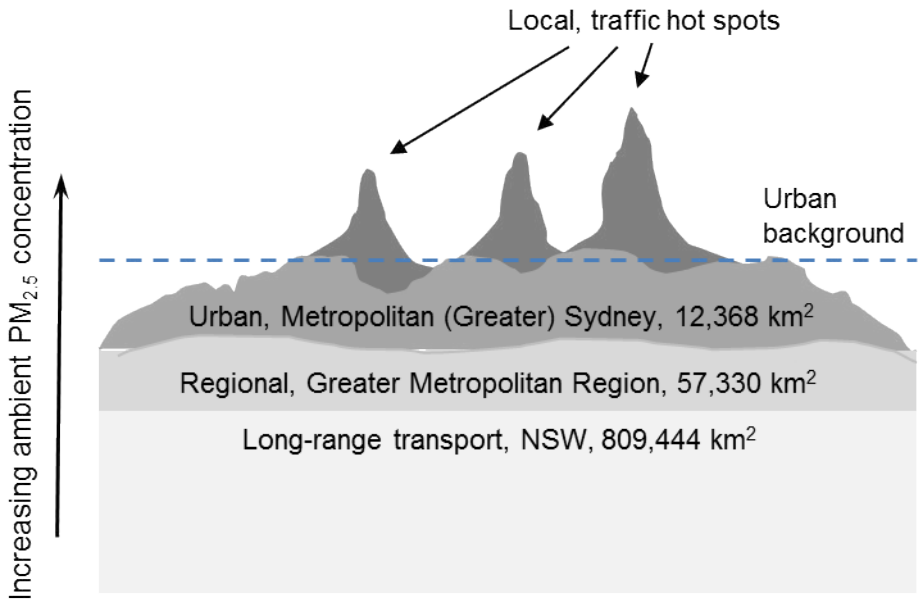

Figure 3.5.1 Schematic representation of expected ambient $PM_{2.5}$ levels in NSW.

Adapted from: *Health risks of particulate matter from long-range transboundary air pollution* (WHO 2006b). This is for illustrative purposes only, various sources of particles and atmospheric conditions can disrupt the general spatial concentration gradient.

In NSW 1997-2012, no trends in ambient PM concentrations were evident (Department of Sustainability 2010, NSW EPA 2013b). In Australian cities over the period 1999-2008, with the exception of Launceston and Hobart, where levels of PM_{10} have decreased largely as a result of reductions in domestic wood heating, no trends in ambient PM concentrations were evident (Department of Sustainability 2010). The lack of observed trends in ambient PM concentrations during this time has occurred despite the implementation of air quality standards, vehicle fuel quality standards (reductions in the sulphur content of petrol and diesel) and, vehicle emission standards (reductions in oxides of nitrogen and $PM_{2.5}$) (Department of Sustainability 2010). In NSW, the lack of a clear decrease in ambient PM may be a result of increases in coal mining, energy consumption from coal (which has only begun to decrease since 2008-09) and increased traffic off-setting these air quality improvement measures (ABS 2014c, ABS 2014b, NSW Trade and Investment Division of Resources and Energy 2014).

4. Health effects associated with ambient PM

Many health effects have been attributed to both long-term and short-term exposures to ambient PM (*Table 4.1*). Long-term exposure to ambient PM contributes to the initiation and progression of disease over months or years. Short-term exposure affects individuals who are particularly susceptible to the effects of PM; either because of existing chronic disease, compromised respiratory function in the developing lungs of children or, compromised physiological function in the elderly from the effects of ageing (Brook et al. 2010). Effect estimates (that is, the health effect per unit of PM exposure) in epidemiological studies are usually greater for long-term exposures than for short-term exposures, suggesting that long-term effects are not merely the sum of short-term effects (WHO 2013c). However, the health effects of long-term and short-term exposure are not independent. Repeated short-term exposures may result in the initiation and progression of chronic disease (WHO 2013c), while an acute event such as a heart attack or stroke that results from exposure during a day of high ambient PM concentration may be a consequence of chronic disease progression associated with long-term exposure.

The evidence of effects on mortality rates and, cardiovascular and respiratory morbidity effects from exposure to ambient PM are particularly strong, with consistency of findings across different populations and study types (US EPA 2012b).

Some people are more susceptible to experiencing specific health effects from exposure to PM than others. Elderly people are more prone to cardiovascular morbidity with PM exposure, while children are at increased risk of respiratory effects (US EPA 2009). There is also an increased susceptibility for individuals with underlying cardiovascular disease (hypertension, diabetes, ischaemic heart disease) and respiratory disease (asthma, chronic obstructive pulmonary disease (COPD)). These diseases are not independent. Controlled human exposure and toxicological studies have demonstrated increased PM-related cardiovascular effects in individuals with underlying respiratory conditions (US EPA 2009). Low socioeconomic status is also associated with increased susceptibility to PM exposure, an association that may be dependent on nutritional status (US EPA 2009). In the US, the Clean Air Act requires the US EPA to review national ambient air quality standards to determine the extent to which the standards provide an adequate margin of safety for those most vulnerable to the effects of ambient pollution (Cao 2013). Although some people are more susceptible to the effects of PM, there is no evidence that any segment of the population is free from the health effects of exposure to ambient PM. Furthermore, the effects of long-term exposure to ambient PM may go unnoticed in apparently healthy people because this exposure could potentially accelerate progression of an undiagnosed disease, or initiate disease (WHO 2013c).

Table 4.1 Health effects attributed to exposure to ambient PM

Long-term (months or years) PM exposure	Short-term (daily) PM exposure
All cause (non-accidental) mortality	*All cause (non-accidental) mortality*
Cardiovascular	*Cardiovascular*
Cardiovascular-related mortality	Cardiovascular-related mortality
Atherosclerosis	Ischaemic heart disease
Ischaemic heart disease	Ischaemic stroke
Complications of diabetes	Myocardial infarction
	Congestive heart failure
Respiratory	*Respiratory*
Respiratory-related mortality	Respiratory-related mortality
Asthma symptoms	Asthma symptoms
Reduced lung function in children	Respiratory infections
Reduced lung function in susceptible adults (elderly, people with COPD or asthma)	Bronchitis in children
	COPD symptoms
Respiratory infections in children	
Cancer	
Lung cancer mortality	
Neurological	
Neurological disorders in adults	
Impaired cognitive function	
Development	
Lung development	
Neurological development in children	
Reproduction	
Adverse birth outcomes	
Sperm quality and quantity	
Allergies	*Allergies*
Exacerbation of allergies	Exacerbation of allergies
Allergic sensitization	

Biologically plausible mechanisms to explain many of the health effects of ambient PM have been suggested from the results of controlled human exposure studies and toxicological studies using research animals *(Table 4.2*). In some of these studies, the relevance of responses is questionable, given that exposures to PM were significantly greater than usual ambient exposures (US EPA 2009). However, the aim of such studies is often not to provide direct evidence of the health effects of ambient exposure, but rather, to provide supportive evidence that it is biologically plausible for the health effects observed in epidemiological studies to have resulted from exposure to PM.

Table 4.2 Biologically plausible mechanisms by which PM could cause adverse health effects

Observed biological response to PM	Possible health effect
Pulmonary inflammation	Exacerbation of asthma and bronchitis
Systemic inflammation	Thrombosis Progression of atherosclerosis (including plaque rupture) Myocardial infarction Peripheral artery disease Stroke
Oxidative stress response	Initiation and progression of atherosclerosis Lung cancer Exacerbation of asthma, pneumonia, COPD
Protein modification	Cardiovascular diseases Respiratory diseases Cancer Developmental disorders Central nervous system disorders Adverse birth outcomes
Stimulation of the autonomic nervous system	Arrhythmia Heart disease Congestive heart failure Hypertension
Exaggerated response to environmental allergens	Enhanced allergic responses Asthma
Pro-coagulant activity	Myocardial infarction Thrombosis Peripheral artery disease Stroke
Suppression of immune defence in the lung	Respiratory infection Exacerbation of respiratory diseases

(Nel 2005, Poschl 2005, US EPA 2009)

Methods used to investigate the health effects of ambient PM

Various methods are used to investigate the health effects of exposure to ambient PM. Each type of study is designed to answer a specific question in relation to the potential for exposure to PM to cause health effects and, each type of study has specific strengths and limitations. The major study types are:

- epidemiological studies;
- controlled human exposure (chamber) studies; and,
- toxicological studies.

Epidemiological studies

Epidemiological studies examine the effect of long-term and short-term exposure to ambient PM on the health of populations. These studies determine the effects of exposure on health as people go about their everyday lives. The population includes individuals with a full range of susceptibilities to the potential effects of ambient PM. Since epidemiological studies are conducted in the community,

the population is exposed to the full mix of ambient PM pollution (and other health-affecting exposures). Epidemiological studies attempt to determine the degree to which diseases, other health conditions or death are influenced by exposure to ambient PM, even though none of these health endpoints are unique to exposure to PM. The determinations are usually made without knowing the exact (personal) exposures, rates of particle uptake by critical organs, or the relative contributions of co-existing agents that also affect health (Lipfert 1997). However, sophisticated analyses in well-defined epidemiological studies are able to confer with reasonable assuredness, the health effects associated with ambient PM exposures.

Long-term ambient PM exposure (cohort and ecological studies)

The effect of long-term ambient PM exposure is usually investigated in epidemiological cohort and ecological studies (Dockery et al. 1993, Pope III et al. 2002, Pelucchi et al. 2009). In cohort studies, individual health outcome data from people over a period of time (usually years) is assessed (regressed) against ambient PM (usually average annual concentration) where the people lived. In ecological studies, aggregate health outcome data from a specific place (*e.g.*, country, region or city) and over a period of time (usually years) is assessed against ambient PM (usually average annual concentration) at that location. Both studies attempt to answer the question:

> *What is (are) the health effect(s) of exposure to ambient PM over a long period of time? (i.e., is a higher average annual ambient PM concentration associated with more detrimental health outcomes?)*

Short-term ambient PM exposure (time-series, panel and case-crossover studies)

The effect of short-term ambient PM exposure is usually investigated in epidemiological time-series, panel and case-crossover studies (Katsouyanni et al. 1996, Samet et al. 2000a, Jaakkola 2003, Anderson et al. 2007). In time-series studies, daily health outcome data (usually routinely collected data such as mortality or hospitalisations) at a location is assessed against daily ambient PM data at that location. Panel studies also use daily data however the health outcome data is individual data, such as lung function and self-reported symptoms, from a group of people followed over time. In case-crossover studies the study population consists of subjects who have experienced an episode of the health outcome of interest. Comparisons are made between the number of health outcomes occurring during a period of high air pollution and a period of low air pollution. The case-crossover design has the advantage that each subject serves as his or her own control and therefore subject-specific confounding is limited. However, it is important that the time of onset of the health outcome is precisely known and therefore this type of study is only amenable to investigating health outcomes with abrupt onset, such as heart attacks or asthma episodes. These three types of study attempt to answer the question:

> *What is (are) the health effect(s) of exposure to ambient PM over a day (or few days)? (i.e., are days of high ambient PM associated with increases in the number of detrimental health outcomes above the daily average?)*

Strengths of epidemiological studies

The strengths of epidemiological studies are that (if conducted properly) they measure the effect of ambient PM exposure under usual living (exposure) conditions, on populations, including a proportion of people who may be particularly susceptible to the effects of PM pollution. Therefore,

epidemiological studies are directly relevant to public health because the findings from such studies reflect real life situations.

Limitations of epidemiological studies

In epidemiological studies other variables (*potential confounders*) can influence the observed relationship between PM exposure and health outcomes (*e.g.*, co-pollutants that are emitted with PM or are integral to the generation of secondary PM pollution and, the socioeconomic status of individuals). Confounders can give the appearance that ambient PM is having a greater or lesser effect on health than is actually the case. Confounders should be controlled for in study analyses however it is often unclear what the confounders are, and if the confounders cannot be measured directly then proxy measures may be used to adjust study results. The absence of careful consideration of potential confounders is a limitation of epidemiological studies. A further limitation of epidemiological studies is that personal exposure to ambient PM is rarely measured. Ambient PM is usually measured by fixed-site monitors that are in the vicinity of the population under investigation. No individual is exposed to the exact amount of ambient PM pollution as recorded by the monitor. The amount of personal exposure will depend on a person's activities and movements, as well as age and physical condition, all which affect lung function and the rate of respiration.

The more individual epidemiological studies (of different populations and using different analysis techniques) show similar estimates of effect, the greater the confidence in the collective findings. Each epidemiological study has specific strengths and weaknesses. For example, time-series studies that assess the association of short-term variation in air pollution and health outcomes within the same geographical area are less prone to confounding by population characteristics than cohort or ecological studies. The reason for this is that in time-series studies the population serves as its own control and population characteristics are relatively stable from day-to-day (Bell et al. 2004). However, daily climatic variations are important confounding factors in time-series studies, and therefore need to be controlled for. If similar estimates of effect are determined from a variety of epidemiological studies it can be concluded that the observed effect is the result of exposure to ambient PM and not solely a consequence of confounding variables.

Controlled human exposure (chamber) studies

Studies of the effects of controlled exposures of human volunteers to PM in sealed chambers (referred to as *chamber studies*) are useful for investigating specific, often subtle, health effects from well-defined exposure. The exposure could be PM from a specific emission source such as diesel engines, or PM with a specific composition or size. For ethical reasons, these studies usually examine the effects of short-term exposures in relatively healthy, young to middle-aged volunteers. The study subjects are intensively monitored during the study such that the effects on blood pressure, respiration, lung function, heart rate and levels of biomarkers of disease can be observed. These measurements are not usually obtainable in epidemiological studies. Because of the controlled nature of the exposure, health effects can be studied for exposures to very specific types and levels of PM. These studies can use higher than ambient levels of exposure. While the effects of such exposures may not be directly applicable to exposures in ambient air, the observations can suggest effects that are likely to occur in susceptible individuals at ambient exposures.

Strengths of controlled human exposure (chamber) studies

The advantage of chamber studies is that specific biological effects resulting from controlled PM exposures can be investigated. In such a controlled environment, and with study subjects with pre-defined characteristics for inclusion, the problem of confounding is largely removed.

Limitations of controlled human exposure (chamber) studies

For ethical reasons, the effects of long-term exposures cannot be investigated. Another limitation, also for ethical reasons, is that those people who are most vulnerable to the effects of ambient PM are usually not investigated in chamber studies.

Toxicological studies

Toxicological studies with research animals (and isolated cells) are conducted to investigate the molecular mechanisms by which PM pollutants can cause health effects. However, responses in animals and cells may vary from those in humans. As is the case for chamber studies, toxicological studies aim to provide supporting evidence (plausible mechanism of effect) for the observations seen in epidemiological studies.

Strengths of toxicological studies

Certain invasive investigations of the biological effects of PM pollution can only be conducted in animals or cells and therefore toxicological studies are the only means of gaining specific mechanistic insights.

Limitations of toxicological studies

A significant disadvantage of toxicological studies is that there is no certainty that responses to exposure that are observed in animals will reflect what occurs in humans. Furthermore, in order to observe a measureable effect, exposures in toxicological studies are usually an order of magnitude greater than ambient exposures and therefore are not directly comparable to ambient conditions.

At the population level, the health risk of PM can be high. For example, worldwide, ambient $PM_{2.5}$ is estimated to cause approximately 3-5% of cardiopulmonary deaths (WHO 2009, WHO 2013a). However, the risk to an individual in reasonable health is probably relatively low. For example, the risk of cardiopulmonary death from smoking (for an average smoker in the US) is approximately three times that of long-term exposure to an extremely high level of $PM_{2.5}$ (greater than usual ambient concentration) (Englert 2004). Health effects estimates associated with ambient PM exposure, as with other indicators of ambient air quality, are small. That is to say, that for each individual within a population there is a small, but measurable, health risk associated with exposure to ambient PM. This makes the determination of a causal relationship between ambient PM exposure and health effects, rather than simply an association, difficult. However, there is sufficient evidence to suggest that exposure to ambient PM *causes* adverse health effects.

Indications that exposure to ambient PM is likely to *cause* health effects:

- Consistency in findings across different populations and study types.
- Dose-response relationships observed, with greater health effects associated with higher ambient PM concentrations (Anderson et al. 2012).
- In time-series studies, health effects are observed after, but not before, PM exposures. Although it has been suggested that these observed effects do not always occur with a sufficient delay after exposure to accommodate known physiological processes (Gamble and Lewis 1996).
- Biologically plausible mechanisms have been demonstrated to explain observed health effects (*Table 4.2*).

Ambient PM is a ubiquitous air pollutant. Even spending a considerable amount of time indoors does not preclude exposure, because ambient particles infiltrate into buildings. Ambient PM (especially particles <1 µm in diameter) can constitute a large proportion of indoor levels of particulates (Chow et al. 2002). Everybody is exposed at variable frequency and duration to levels of ambient PM considered detrimental to health. Whether the health of an individual is substantially affected by this exposure depends on the susceptibility of the individual (age and current state of health). It has been suggested that in any population there is such a wide range in susceptibility that some individuals are at risk to even the lowest concentrations of ambient PM (WHO 2003). Thus, while the health risks to some individuals of 'usual' exposures to ambient PM may not be great, the population health risk overall is significant. The significance of this population health risk is demonstrated by estimates of the global burden of disease associated with PM pollution (Section *4.1*).

4.1 Morbidity and burden of disease (DALY estimates)

The WHO *Global Burden of Disease Study 2010* estimated the disease burden caused by different health risks based on disability-adjusted life years (DALYs) (Lim et al. 2012). One DALY can be thought of as one lost year of "healthy" life, and combines measures of years of life lost and years living with a disability. The *Global Burden of Disease Study 2010*, ranked ambient PM pollution ninth among 67 health risk factors contributing to global DALYs (Lim et al. 2012). The study revealed considerable regional heterogeneity in the contribution of ambient PM to the total burden of disease. The contribution of ambient PM to the total burden of disease was dependent upon both the ambient concentration of PM and the relative health burden of other health risk factors. It was estimated that in 2010, ambient PM was responsible for approximately 8% of DALYs (4^{th}-ranked risk factor) in East Asia. It was estimated that in Australasia, ambient PM was responsible for <1% of DALYs (26^{th}-ranked risk factor).

Ambient PM exposure could contribute to the burden of disease (DALYs) by:

1. increasing mortality rates associated with underlying chronic disease;
2. increasing the risk of acquiring disease;
3. increasing the risk of adverse birth outcomes; and
4. increasing mortality rates in frail, elderly individuals, unrelated to underlying disease.

Study results suggest that all four scenarios occur (Bell et al. 2004). It has been estimated that in Sydney in 2008, life expectancy was decreased by an average of 72 days for males and 65 days for females as a result of long-term exposure to $PM_{2.5}$ from anthropogenic sources (Morgan et al. 2013). In England and Wales in 2008, the estimated reduction in life expectancy from anthropogenic PM air pollution was six months (COMEAP 2010).

DALYs provide a comprehensive estimate of population morbidity. Other measures that have been used to indicate morbidity effects include:

- hospital admissions;
- GP consultations;
- presentations to hospital emergency departments;
- disease symptoms;
- number of cases of disease; and
- medication use.

Generally these measures have been applied to studies of the effect of PM exposure on specific diseases or disease classifications (*e.g.*, cardiovascular, respiratory). However, these measurable health outcomes, along with mortality, are only likely to represent the "tip of the iceberg" of the total health impact of PM pollution (*Figure 4.1.1*).

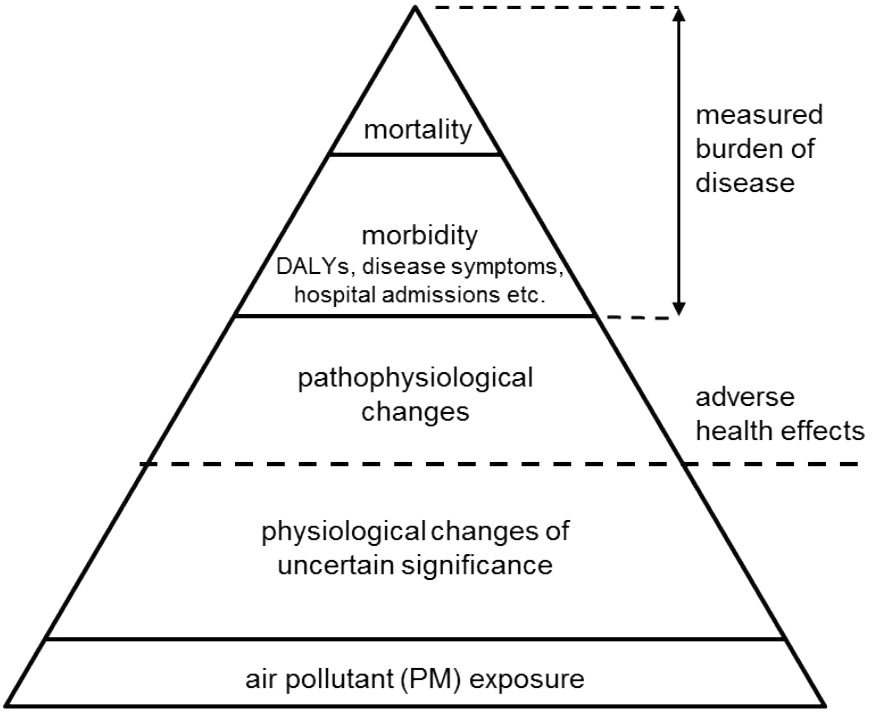

Figure 4.1.1 Schematic representation of the distribution of impacts of PM pollution on population health, adapted from (de Hollander et al. 1999).

4.2 All-cause mortality

Mortality has consistently been shown to be associated with both long- and short-term exposure to PM in ambient air (Bell et al. 2004, COMEAP 2009). Associations with all-cause mortality are strongest for $PM_{2.5}$. The USEPA (US EPA 2009), WHO (WHO 2013c) and the advisory committee to the UK Government, *Committee on the Medical Effects of Air Pollutants* (COMEAP 2009), all consider the strength of evidence sufficient to assign causality to $PM_{2.5}$ exposure for increased rates of all-cause mortality.

4.2.1 Long-term exposure studies

A number of landmark cohort studies have provided solid evidence of the effects on mortality of long-term exposure to ambient PM. The largest of these studies has been conducted on a cohort of adults from the *American Cancer Society Cancer Prevention Study II* (Stellman and Garfinkel 1986). This ongoing, prospective study began in 1982 and first reported results relating to $PM_{2.5}$ exposure for approximately half a million participants from 151 US metropolitan areas over seven years (Pope III et al. 1995). Subsequently, further analysis of the cohort has doubled the follow-up time to more than 16 years, tripled the number of deaths and, included an examination of the effects of exposure to coarser particles (Pope III et al. 2002). The data analyses controlled for many potential confounders including smoking, alcohol consumption, diet, education, occupational exposures and gaseous pollutants. Each 10 $\mu g/m^3$ increase in $PM_{2.5}$ was estimated to be associated with a 4% increase in all-cause mortality (Pope III et al. 2002). However, coarser particles (PM_{10}, PM_{15} and $PM_{15\text{-}2.5}$) were not consistently associated with mortality. The strengths of the study are the large sample size (which increases the precision of the effect estimate although not necessarily the accuracy) and, the adjustment for a large number of potential confounding mortality risk factors.

A cohort study conducted in the Netherlands, involving over 100,000 individuals over ten years, observed a similar estimate of effect as found in the American Cancer Society Study, with a 6% increase in mortality per 10 $\mu g/m^3$ increase in $PM_{2.5}$ (Beelen et al. 2008). However, the association was not statistically significant.

The *Harvard Six Cities Study*, a 15-year prospective study of approximately 8,000 individuals in the eastern US, observed associations between mortality rates and average multi-year ambient $PM_{2.5}$ concentrations in six cities (Dockery et al. 1993). The *Harvard Six Cities* and *American Cancer Society* studies have been updated several times with systematic re-analyses that have included additional years of air pollution and mortality data. These reanalyses have reaffirmed the association between long-term exposure to $PM_{2.5}$ and mortality (WHO 2013c). The latest extension to the *Harvard Six Cities Study* added an additional 11 years (1999-2009) of $PM_{2.5}$ exposure data at lower levels than previously owing to improvements in air quality over the subsequent years (Lepeule et al. 2012). These latest analyses indicate a statistically significant, 14% increase in all-cause mortality associated with a 10 $\mu g/m^3$ increase in the annual mean ambient $PM_{2.5}$ concentration. This is comparable to the 26% increase in mortality between the most- and least-polluted cities (19 $\mu g/m^3$ difference in mean annual $PM_{2.5}$) determined in the initial analysis (Dockery et al. 1993). Thus, the effect estimate is consistent over a range of $PM_{2.5}$ concentrations and time periods when a multitude of other factors such as standard of healthcare and health risk factors would have changed.

A recent body of work to identify concentration-response functions from a review of international and Australian reports recommended estimates of 4% and 6% increases in mortality per 10 µg/m^3 increase in long-term PM_{10} and $PM_{2.5}$ exposures, respectively (Jalaludin and Cowie 2012). A health risk assessment using these recommended concentration-response functions estimated that 4% and 2% of deaths in major Australian cities during 2006-2010 were attributable to long-term exposure to PM_{10} and $PM_{2.5}$ (Frangos and Di Marco 2013).

The evidence associating mortality with long-term exposure to PM_{10} is more limited than that for $PM_{2.5}$ (Abbey et al. 1999, Jalaludin and Cowie 2012). However, not all studies investigating the effects of long-term $PM_{2.5}$ exposure on all-cause mortality have observed significant associations (Abbey et al. 1999, McDonnell et al. 2000, Beelen et al. 2008, Puett et al. 2011). There is no strong evidence that mortality is associated with long-term exposure to coarse particles ($PM_{10-2.5}$) (Brunekreef and Forsberg 2005, US EPA 2009).

4.2.2 Short-term exposure studies

Time-series studies of large populations conducted across multiple locations in Europe and the US have examined the effect on daily mortality of short-term increases in ambient PM concentrations. The multi-city *Air Pollution and Health: a European Approach* (APHEA) project demonstrated that short-term exposure to PM_{10} and black smoke resulted in increases in the daily number of deaths (2.2% increase in daily mortality associated with a 50 µg/m^3 increase in daily PM_{10} concentration) (Katsouyanni et al. 1997). An extension of the APHEA database from 12 to 29 European cities (APHEA 2) confirmed these results and demonstrated a modification of the effect of PM on mortality that was explained by specific city characteristics such as other pollutants, temperature and the background mortality rate (Katsouyanni et al. 2001). A meta-analysis of 33 European studies by the WHO resulted in a summary estimate of a 0.6% increase in daily all-cause mortality associated with a 10 µg/m^3 increase in daily PM_{10} concentration (Anderson et al. 2004).

Analysis of daily mortality rates in the 20 and 90 largest US cities (the *National Morbidity, Mortality, and Air Pollution Study* (NMMAPS)) found effect estimates that were generally consistent with European studies; a 0.5% increase in daily all-cause mortality for every 10 µg/m^3 increase in ambient PM_{10} concentration on the day preceding death (Samet et al. 2000, Samet et al. 2000a, Daniels et al. 2004). Ambient $PM_{2.5}$ has also been associated with daily mortality. A time-series analysis of mortality data from the cities in the *Harvard Six Cities Study* indicated that a 25 µg/m^3 increase in daily $PM_{2.5}$ concentration was associated with a 3% increase in daily all-cause mortality (Bell et al. 2004). A recent analysis of over four million deaths from 2000 to 2006 in 75 US cities estimated that a 10 µg/m^3 increase in 2-day averaged $PM_{2.5}$ concentration was associated with a 1% increase in daily all-cause mortality (Dai et al. 2014). Similar effect estimates for $PM_{2.5}$ on daily all-cause mortality have been found in studies with data from 112 (Zanobetti and Schwartz 2009) and 27 (Franklin et al. 2007) US cities.

Data from the European APHEA and US NMMAPS studies were brought together (along with Canadian data) under a common protocol, *The Air Pollution and Health: A Combined European and North American Approach* (APHENA) study (Samoli et al. 2008, Katsouyanni and Samet 2009). The APHENA study found that the European and US mortality effect estimates for PM were generally comparable however, estimates were higher in Canada. In both Europe and the US, the daily

mortality risk associated with PM_{10} exposure was greater in cities with a higher proportion of elderly people and a higher rate of unemployment. Time-series studies have also identified associations between ambient PM and daily mortality in cities in Mexico, Thailand and Chile (Bell et al. 2004).

While many multi-city studies and several meta-analyses have demonstrated associations between short-term PM_{10} exposure and mortality, locations with higher $PM_{2.5}/PM_{10}$ ratios had stronger associations (Pope and Dockery 2006). This suggests that particles <2.5 µm may be responsible for a considerable component of the associations observed between daily mortality and ambient PM_{10} concentrations. There is currently limited evidence of mortality effects associated with short-term exposures to coarse PM ($PM_{10-2.5}$) (Brunekreef and Forsberg 2005, US EPA 2009).

In Australia, ambient PM has been shown to have a small effect on daily mortality rates in Sydney, Perth and Brisbane and, minimal impact on daily mortality in Melbourne (Simpson et al. 1997, Morgan et al. 1998, Simpson et al. 2000, Simpson et al. 2005). Meta-analyses of Australian studies have yielded increases in daily mortality of 0.2% and 0.9% per 10 µg/m^3 increase in PM_{10} and $PM_{2.5}$, respectively (Simpson et al. 2005). These estimates are comparable to US and European estimates and support a larger effect of $PM_{2.5}$ (than PM_{10}) exposure. A somewhat higher estimate of a 1.35% increase in all-cause mortality in response to short-term PM_{10} exposure has been reported for Sydney during the period 1994-2002 (Morgan et al. 2010). However, the mortality effect of PM_{10} appears to have declined in both Sydney and Brisbane since 2000, despite no reduction in ambient PM_{10} levels (Roberts 2013). This has speculatively been attributed to emissions regulations and standards resulting in less toxic PM (Roberts 2013).

Uncertainty in interpreting the public health impact of mortality associated with short-term PM exposures relates to the extent to which deaths resulting from days of high ambient PM concentration occur primarily in frail persons who, without such exposure, would have died imminently (Bell et al. 2004), a phenomenon called *mortality displacement*. Studies have assessed the degree to which mortality displacement is occurring by examining air pollution effects at multiple timescales. These studies have indicated that PM exposures advance mortality by more than just a few days and therefore ambient PM has a much larger public health impact on mortality than the mortality displacement theory would suggest (HEI 2003, Bell et al. 2004). The fact that PM increases daily deaths out of hospital more than daily deaths in hospital also suggests that PM-associated deaths are not merely displacing deaths (that would have otherwise occurred) by a few days (Schwartz 2000). In other words, mortality displacement may occur, but it cannot fully explain the mortality effects of PM exposure (Roberts 2011). In short, excess deaths on high-PM days are likely to be contributing to the higher long-term mortality seen in cohort studies and are therefore of high public health concern.

4.3 Respiratory health effects

Many epidemiological studies have demonstrated associations between PM exposure and respiratory morbidity and mortality (Ruckerl et al. 2011, Anderson et al. 2012). Child and adolescent respiratory health have been a focus of air pollution studies because lung and immune immaturity along with greater ventilation rates and outdoor activity make these groups particularly susceptible to the effects of ambient air pollution (Ruckerl et al. 2011, Schuepp and Sly 2012). Also, lung function in childhood is a strong predictor of lung function in adulthood and therefore an important

predictor of future health (Gilliland et al. 1999). Furthermore, from a methodological standpoint, studies involving children generally have fewer confounders (*e.g.*, smoking, occupational exposures) that may influence results and therefore epidemiological studies of children potentially provide a more accurate assessment of the health effects of environmental pollutants (Heinrich and Slama 2007).

Outcomes associated with PM exposures in children have included reduced lung function and growth; respiratory illness, symptoms and infection; and post-neonatal respiratory mortality (Pope and Dockery 2006, Heinrich and Slama 2007, Grigg 2011, Ruckerl et al. 2011, Brugha and Grigg 2014). In the *Harvard Six Cities* and *American Cancer Society* cohort studies, long-term PM exposure was associated with respiratory illness in children (Pope and Dockery 2006). The *Review of evidence on health aspects of air pollution (REVIHAAP)* conducted by the WHO found that since 2005, studies of birth cohorts in Europe and elsewhere have reported significant associations between exposure to $PM_{2.5}$ and respiratory infections, lung function and asthma in children (WHO 2013c). A US EPA review of the evidence of health effects associated with PM exposure concluded that long-term exposure to $PM_{2.5}$ was associated with decrements in lung function growth in children (US EPA 2009). In addition, meta-analyses of studies conducted in countries with widely varying socio-demographics provide strong evidence that exposure to $PM_{2.5}$ is causal for respiratory infections in children (Mehta et al. 2013) and that exposure to PM_{10} is causal for childhood asthma episodes (Weinmayr et al. 2010). In the past decade, evidence for incident asthma associated with PM exposure has proliferated, especially in children (Sava and Carlsten 2012).

Interventions that have reduced ambient PM concentrations have been accompanied by reductions in adverse respiratory health outcomes in children. Closure of a steel mill in a valley in Utah, US in 1986 resulted in a large decrease in ambient PM pollution and was associated with reductions in respiratory hospital admissions of children (primarily bronchitis and asthma) (Pope III 1989). Reductions in ambient PM pollution in East Germany following German reunification in 1990 were associated with reductions in the prevalence of bronchitis in children (Heinrich 2003).

Despite the emphasis on childhood studies, exposure to PM has also been shown to effect lung function in adults, primarily in susceptible populations (*e.g.*, the elderly and people with pre-existing respiratory diseases such as COPD) (Pope and Dockery 2006, Anderson et al. 2012). An analysis of data from eight cities from the APHEA 2 multi-European city study found associations between short-term exposure to PM_{10} and hospital admissions for adult asthma, COPD combined with asthma (>65 years) and, all respiratory diseases (>65 years) (Atkinson et al. 2001). Short-term exposure to $PM_{2.5}$ has been shown to be associated with hospitalisations for asthma and COPD (Sava and Carlsten 2012). The 2009 US EPA science assessment of the health effects of PM concluded that long-term exposure to $PM_{2.5}$ is associated with respiratory symptoms and disease in adults (US EPA 2009). Synthesising the evidence from both epidemiological and toxicological studies, the US EPA concluded that the relationship between both long- and short-term $PM_{2.5}$ exposures and respiratory health effects was likely to be causal. In relation to coarse PM ($PM_{10-2.5}$), the US EPA concluded that the evidence is suggestive of a causal relationship between short-term exposure and respiratory health effects but that the evidence is insufficient to conclude a causal relationship between long-term exposure and respiratory health effects.

Since the 2009 US EPA science assessment, several studies have reported associations between exposure to coarse PM and respiratory outcomes in adults, including respiratory, asthma and COPD hospital admissions and, respiratory mortality (WHO 2013c). While effect estimates for coarse PM were somewhat lower than those for $PM_{2.5}$, associations with respiratory and COPD hospital admissions remained when results were adjusted for ambient $PM_{2.5}$ concentration.

In Australia, associations have been demonstrated between daily ambient PM concentrations and respiratory (Petroeschevsky et al. 2001, Hinwood et al. 2006, Chen et al. 2007, Morgan et al. 2010), pneumonia (Hinwood et al. 2006) and asthma (Petroeschevsky et al. 2001, Hinwood et al. 2006) hospital admissions, respiratory symptoms in children (Lewis et al. 1998), physician consultations for asthma in children (Jalaludin et al. 2004) and, respiratory disease mortality (Simpson et al. 2000).

In the past decade toxicity studies have demonstrated that both coarse and fine particles induce inflammatory responses in the lungs of rats and mice (Gerlofs-Nijland et al. 2007, Gilmour et al. 2007, Happo et al. 2010, Halatek et al. 2011, Nemmar et al. 2013a). Inflammation has the potential to damage the lung epithelial lining, which may have important implications regarding pathogenic diseases, asthma and allergy (Nemmar et al. 2013a). It has been suggested that heightened airway hyper-responsiveness due to exposure to particulate air pollutants may provide an explanation for the increasing prevalence of allergic disease (Cacciola et al. 2002, D'Amato 2011).

4.4 Cardiovascular health effects

Long- and short-term exposures to PM have been associated with increases in cardiovascular disease endpoints in the US, Europe, Asia and Australia (Howie et al. 2005, Samoli et al. 2008, Guo et al. 2009, Brook et al. 2010, Chen et al. 2010b, Lippmann 2014).

Follow-up analyses of both the *American Cancer Society* and *Harvard Six Cities* cohort studies have shown that cardiovascular-related mortality was more strongly associated with long-term exposure to $PM_{2.5}$ than was respiratory-related mortality (Pope et al. 2004, Laden et al. 2006b). A synthesis of data from many studies suggests that the effect of long-term exposure to $PM_{2.5}$ on cardiovascular mortality risk is more than 3-fold greater than the effect on respiratory mortality risk (Hoek et al. 2013). However, data from the *Adventist Health Study*, another long-term cohort study from the US, suggests that cardiovascular mortality was not greater than respiratory mortality with regard to long-term PM_{10} exposure (Abbey et al. 1999).

The *American Cancer Society* and *Harvard Six Cities* studies observed that increases of 10 $\mu g/m^3$ in ambient $PM_{2.5}$ were associated with 8-28% increases in cardiovascular mortality (Pope et al. 2004, Laden et al. 2006b). More recent cohort studies in women have observed increases of 76% in cardiovascular disease mortality and 43% in coronary heart disease mortality associated with 10 $\mu g/m^3$ increases in $PM_{2.5}$ and PM_{10}, respectively (Miller et al. 2007, Puett et al. 2008). It is unclear whether the increased cardiovascular mortality in women compared to men reflects a real increase in risk in women or improved methods in risk determination in the more recent studies. However, in a follow-up of the *Adventist Health Study*, both PM_{10} and $PM_{2.5}$ were significantly associated with coronary heart disease mortality in women but not in men (Chen et al. 2005).

Long-term exposure to PM has also been associated with non-fatal cardiovascular events. Recent European studies found that long-term exposure to ambient $PM_{2.5}$ and PM_{10} was associated with the

incidence of myocardial infarction (heart attack), unstable angina (chest pain) and stroke (Cesaroni et al. 2014, Stafoggia et al. 2014). These associations were observed below the current European standards for annual average concentrations of $PM_{2.5}$ (25 µg/m^3) and PM_{10} (40 µg/m^3).

The evidence linking short-term exposure to PM with cardiovascular events is considerable (Franchini and Mannucci 2007, Bhaskaran et al. 2009, Donaldson et al. 2013, Martinelli et al. 2013, Lippmann 2014). The effect of short-term exposure to PM_{10} is weaker than for $PM_{2.5}$, but still exerts significant effects such as increased risk of cardiovascular mortality, myocardial infarction and stroke (Analitis et al. 2006, Donaldson et al. 2013, Teng et al. 2014). Associations between short-term exposure to ambient PM and cardiovascular hospital admissions (Barnett et al. 2006, Hansen et al. 2012) and cardiac arrests (Dennekamp et al. 2010, Straney et al. 2014) have been observed in Australian cities. The estimates of effect in Australian cities were greater for $PM_{2.5}$ than for PM_{10} exposure.

The reason that short-term exposure to PM is often associated with cardiovascular events may be a reflection of both the commonality of cardiovascular disease in Western countries and, the necessity of a "trigger" to cause a cardiovascular event. Cardiovascular disease is the leading cause of morbidity and mortality in Western countries and a high proportion of people have the vascular changes (*e.g.* narrowing of arteries in atherosclerotic disease) associated with increased cardiovascular disease risk. Thus, a relatively large number of people are vulnerable to cardiovascular events. For an event such as a heart attack or stroke to occur, atherosclerotic disease is usually necessary but a further acute stimulus can result in the complete occlusion of the artery and stop blood flow. PM air pollution is one possible stimulus or trigger and the biological effects of exposure discussed below support this hypothesis. Notwithstanding that associations with long-term PM exposure suggest that PM also has a role in the development of cardiovascular disease over time; the process is not well understood.

Cardiovascular-related biological changes associated with short-term PM exposures include heart rate changes, changes in vascular reactivity, increased blood pressure, changed blood flow and, increases in mediators of blood coagulation and vascular inflammation (Lippmann 2014). Many cardiovascular-related effects have been observed in controlled exposures of humans and animals to combustion-derived ultrafine particles less than 0.1 µm in diameter ($PM_{0.1}$) (*Table 4.4.1*). The cardiovascular toxicity of $PM_{0.1}$ may relate to the capacity of these particles to carry toxic compounds and the location of particle deposition. Compared to larger particles, $PM_{0.1}$ particles have a larger surface area per particle mass and hence a higher carrying capacity of potentially toxic substances. $PM_{0.1}$ are small enough to be able to translocate from the airways to the circulation and have been found in the blood and various organs of experimental animals (HEI 2013).

The various cardiovascular effects associated with exposure to $PM_{0.1}$ could potentially contribute to a cardiovascular event. The proposed mechanisms for $PM_{0.1}$ causing these effects are:

- lung inflammation spilling over to the cardiovascular system;
- translocation of particles into the cardiovascular system; and
- particles stimulating the central nervous system to affect the heart and blood vessels (Donaldson et al. 2013).

Table 4.4.1 Cardiovascular effects associated with exposure to combustion-derived $PM_{0.1}$

Heart

- decreased cardiac output
- restriction in blood supply (ischemia)
- altered heart rate (arrhythmia)

Blood vessels

- vasoconstriction
- increased blood pressure
- altered vascular reactivity to endogenous regulators of vascular tone
- increased atherosclerosis and, blood vessel lesions prone to rupture

Blood

- increase in pro-coagulant factors
- increased oxidation
- increase in inflammatory mediators
- pro-atherosclerotic blood lipid profile

Sources: (Weichenthal 2012, Donaldson et al. 2013, Li et al. 2013)

Exposure to $PM_{0.1}$ for as little as 24-hours causes changes in the microcirculation of healthy mice without any significant sign of inflammation in the respiratory tract (Khandoga et al. 2010). Even shorter exposures in humans change systemic blood circulation and markers of blood coagulation (HEI 2013).

Cardiovascular changes are not limited to exposure to $PM_{0.1}$. Exposure to $PM_{2.5}$ (and in some cases PM_{10}) has been associated with increases in vascular inflammation and accelerated atherosclerosis in mice (Martinelli et al. 2013), heart rate changes and increases in blood pressure in humans (Franchini and Mannucci 2007) and, increases in blood markers of inflammation, coagulation and oxidation in humans (Brook et al. 2010, Hajat et al. 2015). Long-term exposure to $PM_{2.5}$ has been associated with surrogate markers of atherosclerosis in humans (Brook et al. 2010, Adar et al. 2013).

On an individual level, the risk of PM pollution initiating a heart attack or stroke is small. This increased risk is principally to people with diagnosed cardiovascular disease or seemingly healthy people with unrecognised existing cardiovascular disease. It has been estimated that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ during the preceding day contributes on average to the premature death of approximately one susceptible person per day among 5 million people in the US (based on annual death rates in the US in 2005) (Brook et al. 2010). However, as everybody is exposed to ambient PM pollution, the burden to population health is significant. From the above estimation, daily increases in $PM_{2.5}$ levels lead to the premature mortality of 10,000's of people every year in the US (Brook et al. 2010). It has been estimated that in populations, PM exposure increases the incidence of myocardial infarction to a greater extent than does physical exertion or emotional state (Martinelli et al. 2013).

Ambient PM is unique among cardiovascular risk factors. Other cardiovascular risk factors such as diet and exercise can be modified by the individual. Ambient PM is ubiquitous and there is little an individual can do to reduce exposure. Through population level actions, including government action, levels of ambient PM can be reduced. In the most recent scientific statement by the American Heart Association on PM and cardiovascular disease, $PM_{2.5}$ exposure was deemed a modifiable risk factor for cardiovascular morbidity and mortality (Brook et al. 2010).

4.5 Cancer

Carcinogenic compounds are components of ambient PM and positive associations between both $PM_{2.5}$ and PM_{10} and, lung cancer risk have been observed in the US, Europe and elsewhere (Hamra et al. 2014). Across 17 cohorts, in 9 European countries, a 10 $\mu g/m^3$ increase in personal exposure to $PM_{2.5}$ and PM_{10} was significantly associated with increases of 40% and 22% in lung cancer incidence, respectively (Raaschou-Nielsen et al. 2013). Other studies that based exposure on fixed site monitoring of ambient PM have generally found smaller estimates of effect, approximately 9% increase in lung cancer risk for a 10 $\mu g/m^3$ increase in ambient PM (Hamra et al. 2014). The *American Cancer Society*, *Harvard Six Cities* and *Nurses' Health* cohort studies (among others) found significant associations between ambient $PM_{2.5}$ levels and lung cancer mortality (Pope III et al. 2002, Lepeule et al. 2012, Puett et al. 2014). Importantly, all of these cohort studies adjusted for the smoking status of individuals.

In 2012, the International Agency of Research on Cancer (IARC) classified diesel engine exhaust, silica and wood dusts and, some metals that are components of PM, as carcinogenic to humans (IARC 2012b, IARC 2012a). Exposure to silica and wood dusts is primarily occupational. Although exposure to diesel exhaust is not limited to occupational exposures, the IARC based their conclusions on the results of investigations of occupational exposures (and toxicity studies in animals). The conclusions of the IARC referred to *whole* diesel exhaust, that is, the mix of gases and particles. Thus, it was not concluded that the PM component of diesel exhaust specifically was associated with cancer.

Occupational case-control and cohort studies suggest that exposure to diesel engine exhaust increases lung cancer risk (Olsson et al. 2011, Vermeulen et al. 2014). Other investigators are not convinced that the evidence conclusively shows that diesel engine exhaust is a cause of lung cancer (Gamble et al. 2012, Sun et al. 2014). A significant deficiency of the occupational studies is the limited capacity to determine quantitative estimates of exposure, which are often based on work histories rather than environmental monitoring.

More recently the IARC has expanded what it considers causative for cancer and included evidence from air pollution exposures of the general population (non-occupational). In October 2013, the IARC announced that it classified outdoor air pollution and PM from outdoor air pollution as carcinogenic to humans(IARC 2013, Loomis et al. 2013). The IARC stated that there was "sufficient evidence" to conclude that "exposure to outdoor air pollution *causes* lung cancer," and that "exposure to outdoor air pollution was *positively associated* with an increased risk of bladder cancer." With regards to bladder cancer, there was limited epidemiological evidence, with most studies assessing potentially high occupational exposures. The evidence related to lung cancer was strong. It was noted by the IARC that:

> "The findings regarding carcinogenicity of outdoor air pollution as a mixture, and of particulate matter specifically, are remarkably consistent in epidemiological research, studies of cancer in experimental animals, and a wide range of studies of mechanisms related to cancer. Particularly, an increased risk of lung cancer was consistently observed."
>
> "Notably, virtually all of the studies were done in areas where annual average levels of $PM_{2.5}$ range from about 10-30 $\mu g/m^3$, which represents approximately the lower third of exposures worldwide" (Loomis et al. 2013).

It is thought that the mechanism by which PM air pollution causes cancer may involve oxidative stress. Ambient particles contain transition metals such as chromium and nickel, which have been classified by the IARC as carcinogenic to humans (Field and Withers 2012). Polycyclic aromatic hydrocarbons (PAHs) found in PM also have mutagenic properties. Both transition metals and PAHs are able to generate DNA-damaging reactive oxygen species that may lead to cancer (Risom et al. 2005). A number of studies have demonstrated associations between $PM_{2.5}$ exposure and biomarkers of DNA damage (Lewtas 2007). Moderate personal exposure to $PM_{2.5}$ (generally <25 $\mu g/m^3$) has been associated with increased oxidative DNA damage (Sorensen et al. 2003).

Various sources of PM emissions have been investigated for their carcinogenic properties. Most evidence relates to combustion sources. Human studies of both short- and long-term exposures to combustion emissions and ambient $PM_{2.5}$ have been associated with measures of genetic damage (Lewtas 2007). The most intensively investigated PM source is diesel engine exhaust. Many studies have observed associations between occupational exposure to diesel engine exhaust and lung cancer. However, it is unclear if there is a cancer risk from environmental (non-occupational) exposures to diesel exhaust.

Many toxicological studies in animals chronically exposed to diesel exhaust have shown that exposure is associated with a dose-related increase in lung tumours (Risom et al. 2005). There is overwhelming evidence from animal and cell culture experiments that exposure to diesel exhaust and diesel exhaust particulates causes oxidative DNA damage in a dose-response manner (Risom et al. 2005, Moller et al. 2008). There is also evidence indicating that exposure to traffic-related air pollution particles in general is associated with oxidative damage to DNA that could increase cancer risk (Moller et al. 2008).

The use of coal inside the home for fuel is clearly associated with an increased risk of lung cancer and the same may be true in European and North American homes that burn wood (Hosgood et al. 2010). However, there is little evidence relating outdoor exposure to these combustion sources to increases in cancer. A study from Spain found that populations living in the vicinity of coal-fired power stations had higher risks of mortality from lung, laryngeal and bladder cancer (Garcia-Perez et al. 2009). However no association with ambient air pollution levels was made. There is toxicological evidence that constituents of wood smoke are mutagenic, but the evidence from animal studies and epidemiological studies relating the burning of biomass with cancer is unconvincing (Lim and Seow 2012).

In sufficient quantities, inhalation of coal dust causes lung cancer in animals (Borm et al. 2004). However there is a lack of evidence of an increased lung cancer risk from coal dust in humans (Borm et al. 2004, Stayner and Graber 2010). Excess mortality from colorectal and lung cancer have been

associated with residential proximity to open-cut coal mining in Spain (Fernandez-Navarro et al. 2012). In the Hunter New England Area of NSW, an area of coal mining, higher incidence rates and death rates from cancer were observed compared to NSW rates (NSW Health 2010b). However, the higher rates were for cancers not known to be associated with air pollution (colorectal, prostate, melanoma), and lung cancer rates were not increased. These studies did not monitor air pollution and therefore the increases in cancer mortality associated with area of residence cannot be directly related to air pollution exposures. Animal studies suggest that silica dust (a hazard of various types of mining and quarrying), rather than coal dust, may be carcinogenic (Kolling et al. 2011).

Cancer as an outcome of environmental non-occupational exposure to ambient PM has not been investigated as intensively as have respiratory and cardiovascular disease outcomes. Studies to date indicate that ambient PM is carcinogenic (at least in regards to lung cancer) however these findings remain to be confirmed in further study.

4.6 Other health effects (central nervous system, developmental and reproductive)

There is suggestive evidence linking PM exposure to central nervous system, developmental and reproductive health effects.

4.6.1 Central nervous system effects

There is limited evidence that exposure to high levels of PM air pollution can be detrimental to the central nervous system (CNS) and perhaps contribute to neurodevelopmental and neurodegenerative diseases such as, autism spectrum disorders, Alzheimer's disease and Parkinson's disease. In Canada, the amount of manganese in ambient PM has been weakly associated with the number of diagnoses for Parkinson's disease, which is consistent with the theory that exposure to manganese adds to the loss of neurons during aging (Finkelstein and Jerrett 2007). Residential proximity to traffic and, gestational and early life exposure to traffic-related air pollution and diesel exhaust emissions have been associated with autism spectrum disorders (Costa et al. 2014).

Autopsy brain tissue from individuals who lived in areas with high air pollution levels is suggestive of air pollution exposure contributing to enhanced CNS inflammation and the pathological signs of Alzheimer's disease (Calderon-Garciduenas et al. 2004). Magnetic resonance imaging of the brains of children exposed to high ambient air pollution in Mexico City demonstrated an increase in brain lesions in these children (Calderon-Garciduenas et al. 2008). Children in Mexico City have signs of both neuro- and systemic inflammation (Calderon-Garciduenas et al. 2007). Young and old individuals appear to be particularly susceptible to air pollution-induced neurotoxicity (Costa et al. 2014). Closure of a coal-fired power plant in China was associated with a reduction in exposure to PAHs and improvements in neurological and cognitive development in children (Tang et al. 2014).

The primary mechanisms of air pollution neurotoxicity appear to be related to oxidative stress and inflammation. The brain is vulnerable to both of these processes (MohanKumar et al. 2008). Animals exposed to concentrated PM have enhanced inflammation in the brain and neuropathology (Campbell et al. 2005, Kleinman et al. 2008, Block and Calderon-Garciduenas 2009). Dogs from an urban area exposed to high levels of air pollution demonstrated accelerated Alzheimer's-type

pathology and CNS inflammation compared to dogs from a cleaner air location (Calderón-Garcidueñas et al. 2003). The PM-associated metals nickel and vanadium were detected in the brains of the dogs from the polluted city. Initial evidence from animal studies suggests that exposure to diesel engine exhaust particulates may increase CNS inflammation and cause developmental neurotoxicity (Levesque et al. 2011, Costa et al. 2014).

Systemic inflammation can cause CNS inflammation and neurotoxicity and, is implicated in neurodegenerative disease (Block and Calderon-Garciduenas 2009). Thus it may be that the neurological effects of PM air pollution are a result of systemic inflammation. It is also possible that particles in the systemic circulation translocate into the CNS (Oberdorster et al. 2004, Peters et al. 2006, Calderon-Garciduenas et al. 2008). The evidence in humans, albeit limited, suggests that exposure to PM may adversely affect the CNS.

4.6.2 Developmental and reproductive effects

Exposure to air pollution (and specifically PM) during pregnancy appears to retard foetal growth, as evidenced by associations between exposure and low term birth weights, albeit with considerable variability in results from different regions (Wilhelm and Ritz 2005, Heinrich and Slama 2007, Millman et al. 2008, Parker et al. 2011, Dadvand et al. 2013). Exposure to $PM_{2.5}$ from traffic has been associated with low term birth weights in Los Angeles (Wilhelm et al. 2012). Exposure to PM during pregnancy has also been associated with an increased risk of pre-term birth (Wilhelm and Ritz 2005, Rappazzo et al. 2014). It is not clear whether PM directly affects the foetus or whether effects on the health of the mother are responsible for these adverse birth outcomes. However, the presence of PAHs attached to DNA in umbilical cord blood indicates that air pollutants can transfer to the foetus (Tang et al. 2006). High PAH-DNA adducts in cord blood have been associated with decreased body weight in the first few years of childhood (Tang et al. 2006). Pre-natal exposure to PAHs in air has been associated with morphological changes related to cognitive deficits and behavioural problems in the brains of children aged 7-9 years (Peterson et al 2015).

Lung development continues well into childhood and postnatal exposure to $PM_{2.5}$ is associated with impaired lung growth and decreased lung function later in life (Gauderman et al. 2004, Schuepp and Sly 2012). Children who moved to locations with higher ambient PM have demonstrated a slowing in lung function development, whereas children who have moved to areas with cleaner air have demonstrated an increase in lung function development (Avol et al. 2001).

Pre- and post-natal exposure to diesel exhaust particulates in animals has been associated with a variety of developmental and reproductive effects including retarded growth of offspring, abnormal development of the female and male reproductive systems, altered sperm development and, increased mutation rates in male germline cells (Ema et al. 2013). It has been suggested that inhalation of PM could result in heritable mutations by causing mutations in sperm (Samet et al. 2004), however there is no human evidence for this. There is the suggestion that exposure to air pollution may be associated with a reduction in sperm quality (Selevan et al. 2000) and this could reduce fertility rates. This effect may be related to the oxidative stress effects of PM as increased oxidative stress is associated with decreases in sperm motility (Ruckerl et al. 2011).

4.7 Health effects associated with ambient PM - Summary

There is ample evidence, from a variety of different studies, demonstrating that exposure to ambient PM adversely impacts human health. On a global scale, the health burden of ambient PM is high relative to most other contributors to health burden. Both long- and short-term exposure to ambient PM is associated with increases in mortality. The evidence is strong that ambient PM impacts respiratory and cardiovascular health and, lung cancer risk. There is also strong evidence of biological plausible mechanisms to explain these health effects.

Exposure to ambient PM is also associated with central nervous, developmental and reproductive effects however the weight of evidence is less than for respiratory and cardiovascular health. However, these differences may reflect the quantity of investigations for the various health effects and the relative commonality of respiratory and cardiovascular diseases, rather than an indication of which areas of health are most impacted by ambient PM. It would appear that the inhalation of PM and the resulting biological response mechanisms can impact most systems of the human body.

5. Health impacts of source-specific PM relevant to NSW

5.1 Coal dust

Coal dust is a fine powdered form of coal that is created during mining, processing and transportation of coal. The brittle nature of coal makes it susceptible to forming fine dust. Most of the fugitive coal dust emissions are in the coarse particle fraction ($PM_{10-2.5}$) rather than in $PM_{2.5}$ (Hibberd et al. 2013). Just how much dust is generated during mining and coal processing depends on weather conditions, local geology, mining/industrial activity, and methods of dust suppression (Reynolds et al. 2003).

Communities near to coal mining operations can be exposed to increased dust levels. A study conducted in the UK reported that PM_{10} levels in communities near to open-cut coal mines were, on average, 14% higher than in communities further from the mines (Pless-Mulloli et al. 2000). Mineral identification confirmed that the coal mines contributed to the increased PM_{10} load in the UK communities. However, coal dust was not specifically identified as a component of PM_{10} in the surrounding communities. Mineral matter from an open-cut coal mine in Wales has been found in airborne particles over a mile from the mine, supporting the view that exposure to coal dust extends well beyond mine boundaries (Jones et al. 2002). Coal transportation can be a significant source of coal dust. A study of PM emissions in a valley in India with three open-cut coal mines found that transportation of coal was the main source of suspended PM (Chaulya 2004). Mineral analysis of the suspended PM estimated that 78% of the dust in the valley was of coal origin. A study conducted in a rural community in the US, through which mined coal was regularly transported by truck, found that people who lived beside the road used for coal transport were exposed to coal dust (Aneja et al. 2012).

Coal is classified by *rank*, which reflects the percentage of carbon in the coal. The higher the carbon content, the higher the rank. As well as being composed of coal, which is primarily carbonaceous rock, coal dust is composed of minerals such as quartz (silica) and clays (Harrison et al. 1997). Coal dust from open-cut mining has a much higher mineral component compared to coal dust from underground coal mining (Reynolds et al. 2003). In mining operations dusts are generated not only from the coal but also from adjacent rock strata. These emissions may increase the quartz component of airborne dust to about 10% of the total mixed dust (IARC 1997).

Crystalline silica in the form of quartz dust has been classified by the IARC as carcinogenic to humans (IARC 2012b). However, the IARC has considered the evidence inadequate to classify coal dust as carcinogenic (IARC 1997). High level (occupational) exposure to respirable quartz dust may cause silicosis (Harrison et al. 1997) and coal worker's pneumoconiosis (Schulz 1997). Collectively, these occupationally-acquired forms of lung fibrosis are known as pneumoconiosis. In toxicological studies, both silica and coal dust have been shown to be cytotoxic, with damage to lung cells resulting in eventual lung fibrosis (scarring) (Castranova and Vallyathan 2000). Many researchers consider coal as an inert material mixed with active quartz (Borm 2002) however it remains to be determined whether quartz *is* the toxic component of coal dust. Hydroxyl radical generation (that can potentially induce cancer and other diseases) by coal mine dust is enhanced when the dust has a high quartz content (Borm 2002). Dusts from coal mines with high-rank coals have a stronger propensity for pulmonary fibrotic activity, although paradoxically low-rank coal is more cytotoxic (Schulz 1997).

Health consequences of significant coal dust exposure have been suspected ever since an increase in the prevalence of pneumoconiosis became evident among coal workers in South Wales in the 1930's (Heppleston 1992). Given the long history of coal mining, there have been many studies of coal mine workers over the past century. Occupational exposure to coal dust has been associated with pneumoconiosis, chronic bronchitis, emphysema and loss of lung function (Heppleston 1992, Wouters et al. 1994, Petsonk et al. 2013). Data indicate a dose-response relationship between coal dust inhalation and the incidence and severity of pneumoconiosis (Finkelman et al. 2002). There has also been a strong dose-dependent relationship observed between occupational exposure to coal dust and the development of emphysema and chronic bronchitis (Cohen et al. 2009). In cohort studies, among almost 18,000 British and 9,000 US coal workers, death from COPD was related to cumulative, respirable, coal dust exposure (Miller and MacCalman 2010, Graber et al. 2014). Occupational exposure has also been associated with lung cancer mortality although the relationship is controversial, with some studies reporting a decrease in lung cancer among coal workers (Stayner and Graber 2010). Occupational exposure to coal dust has also been associated with heart disease mortality and, the incidence of multiple myeloma, a cancer of white blood cells (Ghosh et al. 2011, Landen et al. 2011).

As early as 1939, an Australian Royal Commission into the safety and health of workers in coal mines concluded that coal dust might have a role in disease causation (Kinnear 2001). An increase in the prevalence of chronic cough and mucus hyper-secretion among NSW coal miners has been associated with the duration of coal dust exposure (Leigh et al. 1986). An examination of the lungs of deceased NSW underground coal miners who died between 1966 and 1983 revealed that the extent of emphysema (adjusted for age and smoking) and lung fibrosis were strongly associated with the coal and silica content in the lung, respectively (Leigh et al. 1994). Mortality from pneumoconiosis in coal miners has declined in Australia in recent decades, as it has in other developed countries, primarily due to improved work safety practices (Smith and Leggat 2006).

The prevalence of disease due to occupational exposure to coal dust has not only been related to the quantity of coal dust inhaled but also to the mineralogical features of the dust including silica (quartz) content, coal rank and iron content (Finkelman et al. 2002, Huang and Finkelman 2008, McCunney et al. 2009). Cumulative quartz exposure, rather than coal dust exposure, has been shown to be associated with markers of lung inflammation in coal mine workers (Kuempel et al. 2003). Thus, there is the suggestion that the quartz component of coal dust is responsible for its toxic effects. However, published studies have variously associated the prevalence of pneumoconiosis with exposure to coal with high or low quartz content or, have found no association between the quartz content of coal and the presence of lung disease (McCunney et al. 2009). Consistent positive associations between exposure to coal dust from high-rank coals and the prevalence of pneumoconiosis suggest that coal dusts with high carbon content may have a greater capacity to cause lung damage. It has been speculated that this may relate to longer dust retention time in the lung, greater surface area and, more tissue damaging molecules on the surface of dust particles from high-rank coals compared to low-rank coals (McCunney et al. 2009). Coal mines in NSW extract high-rank coals (Geoscience Australia 2014). A correlation exists between the bioavailable iron content in coal from different regions of the US and the prevalence of pneumoconiosis and, it has been speculated that iron, not quartz, is the active agent within coal (McCunney et al. 2009). Overall, the relative importance of quartz content, coal rank and, iron content in determining the toxicity of airborne coal dust has not been resolved.

Significant occupational exposure to coal dust can cause serious, sometimes fatal, respiratory disease. However the effects of occupational exposure cannot be directly extrapolated to the effects of non-occupational exposures in the general community. Occupational exposure is often substantially greater than community exposure due to the proximity of workers to the emission source and the dispersion of coal dust in the atmosphere. Even within the mining occupation, exposures and health outcomes differ. Not surprisingly, pneumoconiosis and chronic bronchitis are more prevalent among underground coal miners than miners in open-cut mines (Leigh et al. 1986, Lockwood et al. 2009). Although occupational exposures to coal mine dust are often greater than non-occupational exposures, mine workers may reduce their exposure through work safety practices such as wearing personal protective equipment, actions not practiced by the general population.

Another significant reason why the results of occupational studies are not applicable to the wider population is that workers tend to be healthier than the population as a whole, known as '*the healthy worker effect*'. There are a variety of reasons why people who work are generally healthier, but principle among them is that the workforce is deficient (compared to the population as a whole) in elderly people, children and, people with severe chronic disease. These are the very people who are most vulnerable to the effects of air pollution. Occupational studies importantly demonstrate the potential for coal dust to cause health effects. However, to determine whether exposure to coal dust has health effects on the general population it is important to conduct studies in communities exposed to coal dust emissions. These studies are discussed in sections *5.1.2* and *5.1.3*.

5.1.1 Nature of the contribution of coal dust to PM in NSW

In the NSW EPA Air Emissions Inventory 2008, *mining for coal* was the greatest source of both PM_{10} (42.5%) and $PM_{2.5}$ (22.6%) in the GMR (NSW EPA 2012a). Sources of emissions covered under the *mining for coal* category in the emissions inventory include: coal extraction activities (*e.g*. blasting, drilling, crushing *etc.*), wind erosion of coal and overburden, loading trains, dumping coal and overburden and, wheel generated dust from paved and unpaved roads. Some of these activities include emissions of PM that are not coal dust. Thus, not all PM in the inventory that is attributable to *mining for coal* is coal dust. Coal processing and coke production are sources of coal dust in NSW that are additional to those covered under *mining for coal* in the emissions inventory (NSW EPA 2012d). However, the contribution of these coal works to PM emissions in the inventory is considerably less than those sources related directly to mining activities covered under *mining for coal*.

The vast majority of PM emissions from coal mining in the GMR (96%) are outside of the urban centres of Sydney, Newcastle and Wollongong (NSW EPA 2012a). In NSW, coal is mined near the eastern and western edges of the Sydney-Gunnedah Basin that extends from the NSW south-coast to the Queensland border. Mining along the western edge in the Wollongong-Appin-Bulli area and in the Lithgow-Mudgee area is generally from underground mines (Geoscience Australia 2014). Mines near the eastern edge of the basin are spread along the Hunter Valley from Newcastle in the south to Muswellbrook in the north. Many of these mines are open-cut. There were 58 active coal mining operations (29 open-cut, 29 underground) in NSW in 2013-2014 (NSW Trade and Investment Division of Resources and Energy 2014).

The highest concentration of open-cut coal mines in NSW is in the Hunter Valley region. Open-cut mines, as opposed to underground mines, have a higher propensity to emit coal dust into the atmosphere since the coal is exposed to the open air environment and blasting is used to remove the overburden (Lockwood et al. 2009). The recently conducted, CSIRO *Upper Hunter Valley Particle Characterisation Study* provided estimates of the sources of ambient $PM_{2.5}$ in the two main population centres (Singleton and Muswellbrook) in the Upper Hunter Valley (Hibberd et al. 2013). Most coal dust particles are in the coarse range of $PM_{2.5-10}$. The study did not identify a unique fingerprint for fugitive coal dust emissions, and so was not able to specifically quantify the contribution of coal dust to ambient PM. However, an indication of its contribution was assumed using the results for black carbon in the soil fingerprint, which was 1% of total $PM_{2.5}$ at Singleton and 4% of total $PM_{2.5}$ at Muswellbrook. The results did suggest that vehicles and industry make a greater contribution to $PM_{2.5}$ at Singleton, which is closer to the coal mines, than Muswellbrook. The highest contributions (to ambient $PM_{2.5}$) from vehicles and industry resulted when winds came from the direction of the mines (Hibberd et al. 2013). Furthermore, the $PM_{2.5}$ collected at Singleton had a higher content of the element silicon (a component of coal and other mineral dusts). A joint project of the NSW EPA, NSW Office of Environment and Heritage, NSW Ministry of Health, CSIRO and, the Australian Nuclear Science and Technology Organisation that aims to determine sources contributing to $PM_{2.5}$ and PM_{10} at population centres in the Lower Hunter Valley and near the Newcastle coal port, commenced in 2014 (Hibberd et al. 2014). Using source apportionment techniques, that study aims to quantify the contribution of major emission sources to ambient PM in the region, which includes coal handling operations at the Port of Newcastle.

A few studies have recently investigated the contribution of dust from coal trains in the Hunter Valley to ambient PM concentrations. A study conducted by a conglomeration of community environmental action groups found that levels of PM increased in close proximity to passing coal trains, however the contribution of coal dust to ambient PM could not be distinguished from that of diesel exhaust emissions or crustal dust resuspended via locomotive wheels (Higginbotham et al. 2013). Another study, commissioned by the Australian Rail Track Corporation, conducted 61 days of continuous PM monitoring at a single rail corridor site passed by Hunter Valley coal trains (Katestone Environmental 2013). That study concluded that there was a small increase in PM levels from unloaded coal trains compared to passenger trains, but that the passing of loaded coal trains did not elevate PM levels any more than did the passing of other trains. An independent re-analysis of the data from the study concluded that coal trains (loaded and empty) caused modest increases in PM (Ryan 2015).

Another community-driven study of ambient PM levels in the Hunter Valley region found regular exceedances of the NEPM 24-hour average PM_{10} standard (50 $\mu g/m^3$) within residential properties in Newcastle and the Lower Hunter Valley region (Rogers et al. 2013). However, the monitoring only occurred over seven days in summer and may not necessarily be representative of usual PM_{10} levels at those locations. Also, summer is when windblown crustal dust and bushfires make greater contributions to ambient PM_{10}.

The NSW EPA Air Emissions Inventory 2008 reports that in the Upper Hunter Valley, 88% of PM_{10} emissions (and 66% of $PM_{2.5}$) were generated by coal mining activities (NSW EPA 2013c). Annual average ambient PM concentrations in Muswellbrook and Singleton have only been reported since 2011, however early indications are that PM concentrations in these two Upper Hunter Valley towns

are higher than in other regions of the GMR (NSW EPA 2013b). The current NEPM goal of not having more than five days per year above the PM_{10} 24-hour average standard was not met in the Upper Hunter Valley in 2012 despite this goal being met in urban and regional sites throughout NSW in that same year (NSW EPA 2013b). The annual average $PM_{2.5}$ concentration in the Upper Hunter Valley did not meet the NEPM advisory reporting standard (8 µg/m^3) in 2011 and 2012 (NSW EPA 2013b). Monitoring of PM_{10} close to coal mining activities (*diagnostic sites*[3]) in the Hunter Valley clearly indicates that coal mining contributes significantly to ambient PM levels in the region (NSW EPA 2013a).

It is important to recognise that while coal dust may contribute to emissions of PM from open-cut mines, many processes associated with coal mining and transport activities generate PM pollution that is not coal dust. PM emissions that do not include coal dust are generated from land preparation prior to coal mining, drilling, blasting and removal of overburden, on- and non-road vehicular transport, rail transport and, mined land rehabilitation (Katestone Environmental 2011). Coal dust is generated during coal recovery (excavators digging coal and dumping coal into haul trucks, wheel generated PM, bulldozing coal, truck or loader dumping of coal into stockpiles and, wind erosion of coal stockpiles), coal processing (coal crushing and screening, transfer of coal from processing plant to stockyard by conveyor, wind erosion from product stockpile, unloading coal from the product stockpile, transfer of coal to truck or rail loading facility) and, coal transport (dumping coal into rail wagons or trucks, transport of uncovered coal, transfer and disposal of coal rejects, loading of coal onto ships at port) (Katestone Environmental 2011). Measurement of PM in the vicinity of coal mining and coal processing activities can indicate that these sources contribute to increased ambient PM; however this does not determine the specific contribution of coal dust. The current *Lower Hunter Dust Deposition Study* aims to identify both the composition and likely sources of visible dust deposited adjacent to the Lower Hunter rail corridor and other areas within the region (NSW EPA 2014b). This will potentially give an indication of the contribution of coal dust to PM air pollution in the local community.

5.1.2 Australian evidence of health effects

No Australian studies have specifically examined the health effect of non-occupational exposures to coal dust. However there have been studies of the health of coal mining communities. NSW Health has analysed routinely collected health and mortality data for the population encompassed by the Hunter New England Area Health Service (HNEAHS) and, reported on general practitioner (GP) presentations in the Upper Hunter Valley towns of Singleton, Muswellbrook and Denman (NSW Health 2010b, NSW Health 2010a). A significant number of open-cut coal mines are located within the regions that were investigated. However the studies did not include assessment of air quality. The studies determined whether health outcomes were different in communities where coal mining occurred. The lack of air pollution data meant that health outcome and air quality associations could not be analysed. The HNEAHS covers more than 130,000 square kilometres and coal mining activities are clustered in only a few small areas (NSW Health 2010b). The studies compared the health status of communities that were close to coal mines with other populations in the HNEAHS and in NSW. Data were stratified by age to accommodate differences in the age distribution

[3] Monitoring PM at diagnostic sites assists in determining the sources of PM but does not provide information about community air quality.

between regions. However there were no data that allowed adjustment for other potential confounders such as socioeconomic status, smoking, levels of obesity and the prevalence of other chronic diseases. Thus the study results need to be interpreted with some caution.

Residents of Muswellbrook and Singleton had higher rates of respiratory illness and presentations to hospital emergency departments for asthma compared to the HNEAHS region (NSW Health 2010b). Muswellbrook, but not Singleton, had higher rates of hospital admissions for respiratory disease and asthma compared to both the HNEAHS and all of NSW. This may be a cause for concern. However, the higher rates of respiratory illness might not be attributable to coal mining but to the large contribution of wood smoke to $PM_{2.5}$ in Muswellbrook during the colder months (Hibberd et al. 2013).

Respiratory disease-related mortality in males was actually lower in the Lower Hunter (where most coal-mining activity occurred) compared to the rest of the HNEAHS and all of NSW (NSW Health 2010b). Both Muswellbrook and Singleton had higher rates of hospital admissions for cardiovascular disease compared to both the HNEAHS and all of NSW. Cardiovascular disease mortality was higher in the Lower Hunter compared to all of NSW. The incidence of lung cancer was not higher in coal mining areas compared to either the HNEAHS or all of NSW. Self-reported health status and asthma were not different in the Hunter Valley region compared to either the HNEAHS or NSW. Hence, overall the findings were mixed.

Survey data suggest that people in the Hunter Valley do not consider their health to be worse than do people in the rest of NSW (NSW Health 2010b). Poor health outcomes in the 25 local government areas within the HNEAHS generally did not correspond with the location of coal mines, with the exception of hospital admissions for cardiovascular disease, where the highest rates of admission coincided with areas with operating coal mines (NSW Health 2010b). Compared to non-metropolitan NSW, there were no differences in the rate of GP encounters for any disease classification in the Hunter Valley region (NSW Health 2010a).

Investigations of the health of communities living in the vicinity of NSW coal mines have predominantly been cross-sectional studies. The studies recorded the health of communities during a specific point or period of time and did not follow the health of individuals over time. It is possible that some families who developed health problems while living near coal mines moved away from the area for the sake of their health. People with pre-existing health conditions may also have moved into the area. Thus, it is possible that health problems associated with living near coal mines may be either over or under reported in cross-sectional studies.

None of these investigations of health outcomes in the Hunter Valley region included air pollution data in the analyses. Therefore it is not clear whether any differences in health outcomes in areas surrounding coal mining activity, compared to populations elsewhere, are the result of exposure to locally emitted PM. Furthermore, the fact that health data from these studies were not adjusted for other possible causes of chronic disease such as rates of smoking and dietary habits, make it problematic to assign any observed poor health outcomes to the environmental health effects of coal mining. Given the limitations to the research conducted to date, there is insufficient evidence that reported adverse health outcomes in Australian communities surrounding coal mining operations are related to exposure to coal dust. However, observations of higher rates of respiratory and cardiovascular outcomes in some coal mining areas warrant further investigation to

determine associations between health outcomes (adjusted for known causes of disease) and exposure to PM derived from coal mining activities.

A review of evidence of the health effects of coal mining on local communities, in the context of the Hunter Valley region of NSW, concluded:

> "*there is no direct research evidence available on coal related disease clusters in the Hunter Region and the evidence from analyses of routine monitoring data shows variable and inconclusive results*" (Colagiuri et al. 2012).

The only Australian evidence of health effects related to exposure to coal dust come from studies of occupational exposure by coal miners (outlined earlier in *section 5.1*). As discussed earlier, the health effects from occupational exposures do not reflect the effects associated with ambient environmental exposures.

5.1.3 International evidence of health effects

The results of a small number of environmental health studies conducted in communities surrounding coal mining in countries other than Australia have been published. The majority of studies have examined the health status of the Appalachian population in the eastern US, where coal mining constitutes a major industrial activity.

In three Appalachian states, the rates of hospitalisation for hypertension and COPD were weakly associated with the amount of coal produced, by county and year (Hendryx et al. 2007). The results were adjusted for a variety of potential confounders including poverty. The ecological study design (county-level data), rather than individual-level data, is a limitation of the study. Hospitalisations for conditions assigned as *insensitive* to coal mining by the study investigators were not increased in coal mining counties. This finding was used to support the notion that increased hospitalisations for *coal sensitive* conditions were unlikely to be explained by other social and environmental factors. However, hospitalisations of two *coal-sensitive* conditions, kidney disease and lung cancer, were negatively associated with the amount of coal the county produced. Hospitalisations by hospital location (rather than residential location of patients) are a poor indicator of exposure to coal mining pollution, particularly for cases of complex chronic disease, where patients may reside a considerable distance from a specialist tertiary hospital.

Health surveys conducted throughout the US (Hendryx and Zullig 2009) and within the Appalachian state of West Virginia (Hendryx and Ahern 2008) have examined the prevalence of self-reported illness in relation to residing within counties with coal mining activities. Self-report of cardiopulmonary disease, all cardiovascular disease, angina, coronary heart disease, heart attack, hypertension, COPD and kidney disease were all associated with living in Appalachian and West Virginian coal production counties (Hendryx and Ahern 2008, Hendryx and Zullig 2009). The West Virginian study relied on county-level data for potential confounders including smoking, obesity and poverty level. Self-reported health data was not confirmed by medical record review. There is the potential for bias in such data, especially if opposition to coal mining is widely reported. Another limitation is that these are cross-sectional data. The coal mining activity occurred coincidently with the reported health outcomes. Ideally, investigation of the association between environmental exposures and the prevalence of chronic disease, such as coronary heart disease, require cohort

studies, in which exposure is assessed many years prior to the measurement of health outcomes. For an environmental exposure to be causal for a disease that progresses slowly over time, the exposure must occur with sufficient time prior to the development of symptoms. However, it is possible for exposure to air pollution to exacerbate already existing chronic disease.

A study which utilised US national health survey data adjusted results for individual-level data on smoking, body mass index and alcohol consumption (Hendryx and Zullig 2009). The study found that living in Appalachia (both coal mining and non-coal mining areas) was associated with poor cardiovascular disease-related outcomes. Residents of coal mining areas outside of Appalachia also had higher rates of cardiovascular disease. The US national survey also indicated that residents of coal-mining counties, both inside and outside of Appalachia, reported fewer healthy days and poorer self-rated health compared to non-coal mining counties (Zullig and Hendryx 2010). The health disparities were greatest for Appalachian coal mining areas, possibly related to mountaintop mining, a particularly environmentally destructive form of surface mining that was independently associated with poor self-rated health (Zullig and Hendryx 2011).

Higher mortality rates (total, cardiovascular, stroke, respiratory, cancer, lung cancer and kidney disease) have also been associated with living in Appalachian coal mining counties (Hendryx et al. 2008, Hendryx 2009, Hendryx and Ahern 2009, Hendryx et al. 2010, Hitt and Hendryx 2010, Christian et al. 2011, Esch and Hendryx 2011, Sergeev 2011). Each of these studies relied on county-level data to adjust for possible confounders of disease rates. The use of county-level, rather than individual subject data, to adjust mortality rates for health risk factors including smoking and socioeconomic disadvantage, is an imprecise method to account for confounding variables and is a limitation of these studies. The Appalachians are an area of severe socioeconomic disadvantage within the US, with poor public health outcomes associated with this disadvantage (Hendryx and Ahern 2009). In Appalachian states, coal mining areas closely align with those areas with the greatest poverty (Esch and Hendryx 2011, Pollard and Jacobsen 2012). It is not possible to discount the possibility that Appalachian coal-mining health disparities are primarily the consequence of socioeconomic disadvantage and the risk factors that accompany this disadvantage. Tellingly, while lung cancer mortality was increased in Appalachian open-cut coal mining counties, it was not increased in non-Appalachian open-cut coal mining counties (Hendryx et al. 2008). This suggests that there may be some aspect specific to *Appalachian* open-cut coal mining counties, either specific to the mining itself or something else, which is responsible for the increased lung cancer mortality in these regions.

It cannot be determined whether, or to what extent, exposure to coal mine pollution contributed to the health disparities found in these studies because no air quality data was reported. It has been reported that ambient PM_{10} levels are marginally higher at mining sites in West Virginia compared to non-mining sites (Kurth et al. 2014). However, the differences between mining and non-mining sites were small compared to seasonal variations in PM.

Except where mountaintop mining was identified (Esch and Hendryx 2011, Zullig and Hendryx 2011, Ahern et al. 2011a), health outcomes in Appalachian studies were assessed against underground and open-cut coal mining combined. It is possible for communities to be exposed to coal dust from coal derived from underground mining (*i.e.*, during coal transport, storage and processing) however it is expected that exposure would be greater from open-cut mining operations. The non-differentiation

of the types of coal mining within these studies limits the conclusions that can be made about the likely coal dust exposures. An analysis of lung cancer mortality rates based on the type of mining found that lung cancer deaths were similarly increased in coal mining regions regardless of whether the mines were underground or open-cut (Hendryx et al. 2008).

It has been reported that mothers living in Appalachian coal mining areas during pregnancy have an increased risk of having children with birth defects and low birth weights (Ahern et al. 2011a, Ahern et al. 2011b). The studies controlled for a variety of gestational risk factors at the individual subject level, including mother's age, education, and smoking and alcohol consumption. The fact that elevated rates of birth defects and low birth weight in coal mining areas remained after adjustment for other risk factors, and that the rate of low birth weight followed a crude dose-response relationship in areas of low and high levels of coal mining, supports the view that some aspect of coal mining contributes to poor birth outcomes. An increase in neural tube defects (a congenital malformation) has been identified in villages in northern China that are within six kilometres of coal mines (Liao et al. 2010). However, few coal mines in the area were equipped to treat mine water and sewage to prevent seepage of pollutants such as arsenic into the environment. There was no evidence that the increase in neural tube defects was associated with exposure to air pollutants from the mines.

Associations between living close to coal mines and poor health outcomes have been reported elsewhere. In Spain, increased mortality from a variety of cancers (lung, colorectal, thyroid, liver, gallbladder, brain) has been associated with living less than five kilometres from a coal mine (Fernandez-Navarro et al. 2012). As with the Appalachian studies, the Spanish study used area-level data, rather than individual subject data, and did not control for other important causes of cancer such as smoking. Exposure to coal mine emissions by the population of each municipality was estimated using distance between the municipalities and coal mines. This is an improvement on the Appalachian studies, which did not take into account the location of individual mines when estimating exposure. However, exposure to mine emissions will depend on more than linear distance. Mining operations, prevailing winds and geographic landforms will influence environmental exposure. Furthermore, it was not known how long people had lived at their current residence prior to their death from cancer. Because of these limitations, causality could not be assigned.

In an evaluation of mortality patterns in the historically polluted and socioeconomically depressed Cape Breton County in Nova Scotia, Canada it was found that years of life lost from both respiratory diseases and lung cancer was greater in a coal mining community than elsewhere in the county (Veugelers and Guernsey 1999). This finding might be attributed to environmental and occupational exposures associated with local coal mining. However, the results were not adjusted for other health risk factors. Hence, a causal associated between living in a coal mining community and the risk of respiratory diseases and lung cancer cannot be inferred from this study; (single studies are rarely sufficient to assign causation).

A study conducted during the period of the opening of an open-cut coal mine in Wales found that the number of new episodes of asthma presenting to a nearby general medical practice increased coincidently with the mine opening (Temple and Sykes 1992). The increase in new asthma episodes was consistent and sustained for more than two years after the mine started operations. It is not

clear how many, if any, of the asthma cases related to occupational exposures from the mine. New weekly episodes of asthma were reported as a cumulative sum and not as a proportion of patient presentations or the medical practice population as a whole, which are more accurate measures of asthma presentations. The consistent, sustained increase in new asthma episodes may suggest an effect but it cannot be discounted that publicity surrounding the opening of the mine may have resulted in more people presenting to the practice with health concerns.

Studies in communities in northern England found that small increases in respiratory symptoms in children coincided with increases in ambient PM_{10} levels but that the strength of these associations were similar regardless of whether the communities were near or far from open-cut coal mines (Howel et al. 2001a). While GP consultations for children with conditions considered likely to result from exposure to air pollution (respiratory, skin and eye conditions) were generally higher in open-cut coal mine communities than control communities (Pless-Mulloli et al. 2000, Howel et al. 2001b, Pless-Mulloli et al. 2001), there was no difference in the prevalence of respiratory illness or asthma severity despite PM_{10} levels being higher in coal mining areas (Pless-Mulloli et al. 2000). These data suggest that children in coal mining areas are exposed to a small but significant amount of additional PM_{10}. It is possible that the increase in GP consultations in the coal mining communities may have been a consequence of raised awareness of the potential health effects of the mines.

Another study in England reported an increase in respiratory symptoms and absenteeism (for respiratory symptoms) in children in schools located near a Liverpool dock where coal was handled, compared to control schools located near the docks but away from the coal terminal (Brabin et al. 1994). A higher dust burden (British Standard measurement of dust deposition) was reported at the *exposed* schools. Children at the exposed schools had increased respiratory symptoms irrespective of their parents being smokers or non-smokers.

A recent review of the international evidence of the health effects of coal mining on local communities concluded that there are clear indications that there are serious health harms from coal mining to surrounding communities (Colagiuri et al. 2012). However, the report did not consistently comment on the limitations of individual studies, nor report negative results. The authors speculated on reasons why health impacts in communities surrounding coal mines may be "under-estimated" or "under-reported" but epidemiological study designs that could result in false positive results were not discussed. In an objective evaluation of scientific evidence it is equally as important to consider whether a reported health effect is truly related to an exposure as it is to speculate on why an exposure-health effect relationship was not found when it was expected. The report of Colagiuri *et al* stated "much of the evidence comes from the Appalachian region of the US, where coal mining has been conducted for many years and which has higher morbidity and mortality rates than the rest of the US." Although not implicitly saying so, this statement implies that the high morbidity and mortality is a consequence of the coal mining. There are many possible causes for the health disparities in the Appalachian region and these confounders have not been adequately addressed in studies.

The results of animal toxicity studies suggest that coal dust can harm health. Studies have shown that the inhalation of coal dust generates high levels of reactive oxygen species (chemically reactive molecules that can cause inflammation, damage cells and DNA, and are considered to have a role in a variety of chronic diseases) (Schins and Borm 1999, Pinho et al. 2004). The notion that inhaled coal

dust can cause oxidative tissue damage is reinforced by studies showing that antioxidants reduce lung inflammation in rats exposed to coal dust (Pinho et al. 2005). Evidence that coal dust elicits similar biological responses in humans comes from measurements of blood biomarkers of oxidative stress in community residents and coal miners in Brazil (Avila Junior et al. 2009). Biomarkers of oxidative stress were increased not only in the blood of coal workers but also in the blood of residents that lived near coal mines. Biomarkers of DNA damage have been found in workers who are involved in coal transport or mine equipment maintenance (Schins et al. 1995, Leon-Mejia et al. 2011). DNA damage could predispose individuals to a variety of diseases including cancer.

Animal studies suggest that inhalation of coal dust increases lung cancer risk when exposure is sufficiently great to cause a breach in a threshold level of chronic inflammation (Kolling et al. 2011). Coal dust causes inflammation in the lungs of experimental animals (Brown and Donaldson 1989, Ernst et al. 2002, Kania et al. 2014) and elicits inflammatory cells to express cytokines (molecules involved in inflammatory responses) (Schins and Borm 1999). Instillation of particles collected from a coal mine site into the upper respiratory tract of rats resulted in the dysfunction of small blood vessels, similar to that which occurs in cardiovascular disease (Knuckles et al. 2013). Administration of quartz to the upper airways of mice results in a significant increase in levels of intercellular adhesion molecule-1 (ICAM-1) in lung tissue (Nario and Hubbard 1996). ICAM-1 mediates translocation of inflammatory cells and ICAM-1 levels are increased in a wide variety of inflammatory diseases including cardiovascular disease, asthma and COPD (Kotteas et al. 2014). Thus animal toxicity studies provide supportive evidence of biological responses to coal dust inhalation consistent with the development of respiratory and cardiovascular diseases.

5.1.4 Summary

The summation of evidence from epidemiological studies of communities surrounding coal mines, occupational epidemiology studies and, animal toxicity studies show that exposure to coal dust has the potential to cause chronic respiratory diseases and, possibly cardiovascular disease and cancer. However, limitations of the methods used in the health studies of communities living near coal mines severely restrict what can be inferred from the study results. Furthermore, air pollutants other than coal dust (*e.g*. diesel exhaust emissions and crustal dust) are associated with coal mining and this makes it difficult to assign health effects specifically to coal dust exposure. Thus, the health effects of ambient (non-occupational) exposure to coal dust are unclear, and at this stage, there is insufficient evidence to suggest that coal dust is more hazardous to health than PM from other sources.

The vast majority of community studies have been conducted in the Appalachians in the eastern US, a region of low socioeconomic status and poor health outcomes. Most of these studies have been conducted by the same group of researchers, and the studies have been limited by the lack of individual-level health outcomes data, the absence of adjustment of health outcomes for important potential confounders (*e.g*. smoking) and, poor exposure classification. These studies have failed to collect data on coal dust (or ambient PM) and therefore observed health effects could not necessarily be attributed to exposure to coal dust. The health effect of coal dust on communities surrounding coal mines requires further investigation. Toxicity studies suggest that some components of coal dust, such as quartz, can cause adverse health effects although it is not known if coal dust is more hazardous than other ambient PM.

While there is no conclusive evidence that coal dust specifically and independently affects the health of communities, coal dust and other PM emitted by coal mine operations will contribute to the overall burden of PM that residents are exposed to. Given that there is strong evidence that increased ambient PM mass is associated with increased adverse health effects, PM emissions from coal mining operations are likely to impact the health of populations exposed to these emissions.

The characteristics of coal mining emissions vary from region to region, depending upon coal characteristics, mining techniques (including air pollution reduction measures), local topography and climate. The susceptibility of communities to the health effects of emissions also varies depending upon the health status and demographics of surrounding populations. If the health effects of PM emissions from coal mining in NSW are to be understood, it would be useful for studies to be conducted in this specific context. For such studies to occur, they would need to incorporate source-speciation techniques to estimate the contribution of coal mining operations to ambient PM levels and measure both health outcomes and possible confounders to the exposure-health outcome relationship, at the individual level. A major barrier to undertaking such a study is the need to study large populations over periods of time to detect an effect. Currently communities located in proximity to coal mining in NSW have relatively small population sizes. Studies conducted in small populations may not have the statistical power to detect small changes in the health of that population (not all people will demonstrate a health effect), and any study not adjusting at the individual level for major potential confounders (such as cigarette smoking) may lead to either a bias toward or away from the true effect estimate. Care needs to be exercised when calling for such studies in small communities.

- Occupational epidemiology studies and, animal toxicity studies suggest that PM emissions from coal mining have the potential to adversely impact respiratory health, and possibly cardiovascular health and cancer risk in communities surrounding coal mines.
- Due to limitations in study methods and the small number of studies conducted, there is insufficient evidence that exposure to coal dust is associated with adverse health effects in communities surrounding coal mines.
- At this stage, there is insufficient evidence to indicate whether coal dust is more hazardous to health than PM from other sources.
- The activities of coal mining increase the concentration of ambient PM and this may impact on the health of surrounding communities.
- Appropriately designed and statistically powered studies regarding the health impacts of emissions from coal mining operations in NSW would be useful but care needs to be taken to ensure feasibility, adequate sample size and statistical power.

5.2 Coal-fired power station emissions

Coal is the major fuel for electricity production, accounting for more than 40% of worldwide electricity production and 75% of Australian production (Grubler et al. 2012, Energy Supply Association of Australia 2013). Despite a considerable increase in electricity generation from non-fossil fuels (nuclear, hydro and non-hydro renewables) over the past two decades, the rapid increase in global energy growth has resulted in an increase in power generation from coal that far exceeds the increase in electricity generation from all non-fossil fuel energy sources combined (International Energy Agency 2012). Ambient PM resulting from coal combustion in power stations includes both primary PM emitted directly from power station smoke stacks and secondary PM formed in the atmosphere from sulphur and nitrogen oxide gases emitted by power stations (*Table 5.2.1).*

Table 5.2.1 Ambient PM resulting from coal-fired power station emissions

Origin of PM	PM	Coal-fired power station emissions
Primary PM emissions	Mostly $PM_{2.5}$ with surface-bound metals	PM containing metals and other trace elements of coal combustion
Secondary PM formation in the atmosphere	$PM_{2.5}$ containing sulphates and nitrates	Gases - sulphur dioxide, oxides of nitrogen, PAHs

PM emitted directly from the combustion of coal is often called *fly ash*. Fly ash is a generic term used to describe PM derived from mineral and metal contaminants that are present in organic fuels (BeruBe et al. 2007). In Australia, as in many other countries, power stations employ emission control processes that are capable of removing over 99% of PM before gases are emitted into the atmosphere (Kumar et al. 2013). The efficiency of the emission control devices decreases with decreasing particle size and some small particles escape into ambient air. In Europe, the US and some other countries (but not Australia) additional clean-up to remove sulphur and nitrous oxides is often installed (Azzi et al. 2013). Coal is chemically complex and fly ash from coal combustion contains over 50 elements and oxides (Giere et al. 2003). Many toxic elements and heavy metals are concentrated in fly ash, including arsenic, cadmium, lead, chromium and mercury (Azzi et al. 2013). As much as two-thirds of global anthropogenic emissions of mercury are from fossil fuel combustion (includes electricity generation, industrial processes and domestic heating), with coal combustion being the largest source (Pacyna et al. 2006). Australia is the fifth largest global emitter of mercury from anthropogenic sources (Pacyna et al. 2006) and coal-fired power stations are the second largest anthropogenic source of mercury emissions in Australia, behind gold smelting (from a single location in Western Australia) (Nelson et al. 2012).

The characteristics of PM emitted from coal-fired power stations are dependent on the type of coal and the clean-up technologies used to reduce emissions (Yoo et al. 2005). The composition of trace elements in coal from different areas varies and consequently so does the level of different elements in fly ash. Australian fly ashes generally have the same or lower levels of traces elements than fly ashes elsewhere (Azzi et al. 2013). Emission control processes remove most trace elements although a small proportion are emitted into the atmosphere either as gases or attached to fine

particles that pass through the particulate control devices (Azzi et al. 2013). The finer particles emitted from coal-fired power stations are enriched in heavy metals (Yoo et al. 2005). Due to the potentially bio-reactive nature of metal-bearing particles, there is likely to be an increased health risk associated with their inhalation (Silva and da Boit 2011).

Coal-fired power stations are the largest source (approximately 67% in the US) of sulphur dioxide emissions and a significant source of oxides of nitrogen (vehicle emissions are the major source) (Reiss et al. 2007). Complex chemical transformations of these gases in the atmosphere result in PM containing sulphate and nitrate molecules. Because the largest source of the precursor to sulphate, sulphur dioxide, is coal-fired power stations, sulphate is often used as a marker for coal-fired power station emissions. In the US, concentrations of PM-sulphate are highest in Midwestern and eastern cities where coal-fired power stations are concentrated. In six of these cities sulphate accounts for 30% or more of $PM_{2.5}$ mass (Reiss et al. 2007). State laws requiring reductions in pollutant emissions from coal-fired power plants in North Carolina on the east coast of the US resulted in significant declines in both sulphur dioxide emissions and $PM_{2.5}$ sulphate concentrations (Li and Gibson 2014).

The effects of emissions from coal-fired power stations on ambient PM pollution can occur hundreds of kilometres from the emission source. This is particularly so for secondary PM that exerts its effects downwind from where gaseous precursors are emitted (Smith et al. 2013). However, $PM_{2.5}$ emitted directly from power stations also has a great propensity to remain suspended in the atmosphere for long periods of time and travel large distances. PM derived from high temperature fossil fuel combustion has been found in the sediments of a pristine Tasmanian lake coincident with the period of increasing coal-fired power generation on mainland Australia from 1955 (Cameron et al. 1993, Brady 1996). It has even been suggested that Australian coal combustion sources contribute to low-level background contamination of sub-Antarctic islands off the coast of South America (Rose et al. 2012). Toxins emitted by coal-fired power stations travel long distances attached to PM and potentially could have health consequences elsewhere. Some studies have indicated that the contribution of anthropogenic mercury emissions from Asia (including emissions from coal combustion) to mercury deposition in the US is equivalent to the contribution from anthropogenic emissions from North American sources (Wang et al. 2014c).

The potential of coal combustion to affect the health of populations has been dramatically demonstrated in landmark episodes of sudden increases in mortality and respiratory illness from relatively short-term increases in air pollution in Belgium's Meuse River Valley in 1930; in Donora, Pennsylvania, in 1948; and in London in 1952 (Townsend 1950, Logan 1953, Nemery et al. 2001). These events primarily resulted from intensive burning of coal and human exposure to high pollution levels that were enhanced by local geography and weather. At the time of these historical episodes coal was used much more widely, than it is today, as a fuel for a variety of industrial and domestic processes. Furthermore, controls on emissions did not exist as they do today. Consequently the severity of these past air pollution episodes was much greater than is usually currently experienced (Bell and Davis 2001).

The consequences of current emissions from coal combustion are most evident in developing economies, which have high levels of PM pollution containing heavy metals and other toxins derived from industrial and domestic sources (Zhang et al. 2010, Duan and Tan 2013, Pui et al. 2014). Models have estimated that $PM_{2.5}$ emissions from coal-fired power station emissions in India during

the period 2010-2011 were responsible for approximately 100,000 premature deaths and 20 million asthma exacerbations (Guttikunda and Jawahar 2014). In India and rural China, indoor domestic coal combustion for cooking and heating is associated with severe adverse health effects (including reduced child growth, lung disease, developmental impairment and increased cancer risk) from exposure to high levels of toxic trace elements (Millman et al. 2008, Epstein et al. 2013, Chen et al. 2014). The introduction of domestic coal-burning regulations in Dublin in 1990 reduced PM air pollution, resulting in reductions in both respiratory and cardiovascular deaths (Clancy et al. 2002). The burning of coal domestically indoors and the very high ambient concentrations of PM from coal combustion in developing economies do not equate with exposures from regulated emissions from coal-fired power stations in countries like Australia (the focus of this review). However, these examples of the toxic potential of emissions from coal combustion underscore the importance of minimising exposure to these sources. Furthermore, while greater coal-fired power station emissions in China and India relates to the sheer number of power stations compared to Australia, in the past few years both countries have surpassed Australia in terms of the percentage of power derived from high-efficiency, low-emissions power generation (International Energy Agency 2012). Unlike Australia, emissions of $PM_{2.5}$ per unit of power generated are decreasing in these rapidly developing economies.

5.2.1 Nature of the contribution of coal fired power stations to PM in NSW

The NSW Air Emissions Inventory indicates that in 2008, in the GMR, coal-fired power stations contributed 5.3% and 8.5% to the total PM_{10} and $PM_{2.5}$ emissions, respectively (NSW EPA 2012a). Since 2008 there has been a decline in electricity generated from coal. However in 2012-13 coal remained the major fuel type for electricity generation in NSW at 81% of all electricity generation (Bureau of Resources and Energy Economics 2014).

In 2008, there were eight operational coal-fired power stations in NSW (NSW Department of Primary Industries 2009). Two power stations have subsequently shutdown, citing decreased demand and economic considerations as reasons for their closure (Wallerawang in 2014; Munmorah in 2010). Of the six currently operational coal-fired power stations, one is located west of the Sydney basin near Lithgow, three are in the Hunter Valley and, two are on the NSW Central Coast near Newcastle (NSW Department of Primary Industries 2009). Although these power stations lie well outside the Sydney metropolitan region, $PM_{2.5}$ and gaseous emissions (primarily sulphur dioxide) have a great propensity to disperse over large distances and, therefore, will probably contribute to PM exposure for Sydney residents. Source apportionment modelling has estimated that coal-fired power station emissions were responsible for 14-18% of $PM_{2.5}$ mass in an outer-suburban Sydney site (Cohen et al. 2012). Source apportionment studies have also suggested that the contribution of coal-fired power station emissions to $PM_{2.5}$ is greater in Sydney than it is in Melbourne, Brisbane or Adelaide (Chan et al. 2008). This reflects the high energy use and contribution of coal to the electricity supply of Sydney. The wide dispersion of power station emissions will result in spatially homogeneous concentrations of PM derived from these emissions and therefore there are unlikely to be particularly high ambient PM concentrations close to source. The Upper Hunter Valley Particle Characterisation Study concluded that although primary $PM_{2.5}$ emissions from power stations contributed to 13% of the Upper Hunter emissions inventory, power stations would contribute much less than this to ambient

$PM_{2.5}$ concentrations measured in the study (in the Upper Hunter Valley) because the emitted $PM_{2.5}$ would quickly disperse to other regions (Hibberd et al. 2013). Secondary $PM_{2.5}$ generated via gaseous emissions may be substantial in NSW. Of the total sulphur dioxide emissions produced each year in NSW, over 80% are from coal-fired power stations (Cohen et al. 2012).

5.2.2 Australian evidence of health effects

There is no Australian evidence directly linking exposure to PM from coal-fired power stations to health effects.

Surveys conducted over 25 years ago in the NSW town of Lake Munmorah (then located between two coal-fired power stations) and a control town (Nelson Bay), suggested that wheeze and bronchial hyper-responsiveness (a hallmark of asthma) were more prevalent in children in the town near the power stations (Henry et al. 1991a). These data were adjusted for smoking in the home, which was significantly more prevalent in Lake Munmorah, but not for indoor fuel use. Households in Lake Munmorah were significantly more likely to use wood or gas for heating and fuel other than electricity for cooking, compared to the control town. Indoor fuel use, along with socioeconomic status, which was also significantly different between the two towns (and was not accounted for in data analysis), may have impacted these results. A few years later, another study was conducted comparing children of Lake Munmorah with children from another town, Dungog, where the socioeconomic status of the inhabitants was more similar (Halliday et al. 1993). The greater prevalence of wheeze in Lake Munmorah was confirmed but the prevalence of airway hyper-responsiveness did not differ between the two towns. A study of a cohort of children from Lake Munmorah with a history of wheeze found no association between daily ambient concentrations of sulphur dioxide or nitrous oxides, and respiratory symptoms (Henry et al. 1991b). PM concentrations were not measured in the cohort study.

These study results provide weak evidence of poorer respiratory health in children residing near coal-fired power stations, as the results may have been confounded by factors other than air pollution that impacted on children's respiratory health in the town of Lake Munmorah.

5.2.3 International evidence of health effects

In many industrialised countries, PM from coal-fired power station emissions (represented by PM-sulphur) is a significant contributor to ambient PM. Thus there have been many investigations of the associations between PM-sulphur and health outcomes. A time-series study of approximately 4.5 million deaths in 75 US cities over six years found that a higher proportion of sulphur in $PM_{2.5}$ was associated with a 1.4% increase in daily all-cause mortality and a 4.5% increase in daily mortality from respiratory diseases (Dai et al. 2014). Among 13 constituents of $PM_{2.5}$, sulphur was the only species significantly associated with respiratory disease mortality. $PM_{2.5}$-sulphur was not significantly associated with daily deaths from cardiovascular disease. This large data set, and the fact that data were obtained from many cities across the US, each with their own demographic, social, and environmental peculiarities, provide strong evidence that $PM_{2.5}$-sulphur is detrimental to respiratory health. This analysis of the association between county-level ambient PM and mortality was adjusted for a variety of behavioural and other health risk factors including smoking, alcohol consumption and diabetes.

PM-sulphur comes from several sources and does not solely reflect coal-fired power plant emissions. Thus any measure of health effects associated with PM-sulphur cannot be wholly attributed to exposure to coal-fired power station emissions. Nevertheless, sulphate, the primary form of sulphur in PM, is considered a reliable marker of coal-fired power station emissions.

In New York, $PM_{2.5}$-sulphate and selenium (an indicator element for emissions from coal combustion) were significantly associated with both daily cardiovascular disease hospitalisations and mortality (Ito et al. 2011). In a study from Seoul, South Korea, $PM_{2.5}$-sulphate was moderately associated with daily cardiovascular disease mortality but not with all-cause or respiratory disease mortality (Son et al. 2012). PM-sulphate has been moderately associated with daily respiratory disease hospitalisations (Ostro et al. 2009, Atkinson et al. 2010, Kim et al. 2012b). The study of Kim *et al* found a particularly strong association between $PM_{2.5}$-sulphate and hospitalisations for asthma. The studies of Atkinson *et al*, Kim *et al* and Son *et al*, measured ambient PM concentrations from a single location, which is unlikely to provide a good assessment of exposure for the urban populations of London, Denver and Seoul, respectively, from which the health outcome data were sourced. Considerable spatial variability of $PM_{2.5}$ concentrations in urban environments indicate that there is the potential for exposure misclassification (Pinto et al. 2004).

A meta-analysis of ten single-city time-series studies (eight from North America and two from Europe) determined that PM-sulphate was significantly associated with all-cause mortality (Smith et al. 2009). Analysis of data from the *American Cancer Society Cancer Prevention* cohort of over 300,000 people suggests that long-term exposure to PM-sulphate has a small impact on all-cause mortality and a larger impact on cardiopulmonary mortality (Smith et al. 2009).

A comprehensive review of the health impacts of PM-sulphate found that epidemiological studies which showed a significant positive association between ambient $PM_{2.5}$-sulphate and health effects (usually mortality rates) generally also demonstrated an association between total $PM_{2.5}$ mass and the same health effect (Reiss et al. 2007). Therefore, it was difficult to differentiate the impact of exposure to $PM_{2.5}$-sulphate from the impact of exposure to total $PM_{2.5}$. However, where a significant association between $PM_{2.5}$ and a health effect existed, the relative risk associated with $PM_{2.5}$-sulphate was nearly always greater than the relative risk for total $PM_{2.5}$ mass. This suggests that $PM_{2.5}$-sulphate has greater health impacts than does total $PM_{2.5}$. An analysis of daily mortality in the Netherlands over nine years found that ambient particulate sulphate was more consistently associated with mortality than was PM_{10} (Hoek et al. 2000). A WHO report noted that sulphate is associated with a number of other PM constituents from the combustion of fossil fuels, such as transition metals and organic compounds (WHO 2013c). The WHO report stated that "sulphate could be considered to be an indicator of harmful constituents from oil and coal combustion" and, "it is still unclear whether removal of sulphur dioxide (a precursor for sulphate) from the emissions of oil and coal combustion would lead to a significant reduction in the health effects associated with these sources". Regardless of which components of PM from coal-fired power station emissions are responsible for adverse health impacts, there is considerable evidence suggesting that exposure to these particles is associated with hospitalisations and mortality.

The US EPA *Integrated Science Assessment for PM* concluded that while short-term exposure to secondary sulphate and $PM_{2.5}$ from power stations have been associated with cardiovascular and respiratory health effects, the evidence is not consistent or robust (US EPA 2009). The UK

Government, *Committee on the Medical Effects of Air Pollutants* (COMEAP) concluded from an analysis of results from time-series and panel studies that "there is reasonably strong evidence of a positive effect" of short-term exposure to PM-sulphates on both cardiovascular and respiratory disease outcomes and an especially strong effect on mortality (COMEAP 2009). COMEAP further concluded that long-term exposure studies support a positive (adverse) association between the sulphate concentration in PM and adverse health effects. COMEAP suggested that "sources of particles that are related to sulphur-containing fuel combustion may have adverse health effects" (COMEAP 2009). Interestingly, while COMEAP reported that epidemiological studies have shown reasonably consistent positive associations between ambient sulphur dioxide or PM-sulphate concentrations and effects on cardiovascular health, they also stated there was insufficient toxicological evidence (primarily due to a lack of studies) to suggest that long-term exposure to sulphates may impact on the cardiovascular system (COMEAP 2009). Since that time, further particle characterisation studies on PM-sulphate have been conducted as indicated above.

However, not all epidemiological studies have reported positive associations between PM-sulphate and adverse health outcomes. Two recent studies observed that PM-sulphate was not associated with cardiovascular disease hospitalisations (Atkinson et al. 2010, Kim et al. 2012b). A limitation of these studies was the possibility of exposure misclassification because ambient PM was only measured from a single location. Exposure misclassification can bias results towards the null (*i.e.* show negative results when an association might actually exist). However, the studies did observe moderate associations between PM-sulphate and respiratory disease hospitalisations. A review by the WHO of health effects associated with exposure to PM noted that the source category "secondary inorganic particulate air pollution", which is typically indicated by ambient sulphate, has been associated with cardiorespiratory health effects in most studies published since 2005 (WHO 2013c). The WHO report noted that this category of PM includes particles not only from coal and oil combustion, but also from vehicle exhausts.

An alternative approach to relying on a single PM species such as sulphate, which can come from multiple sources, to indicate coal-fired power station emissions is to use a number of chemical and physical properties of PM that are typically associated with PM from this source (*source apportionment*). The following paragraphs describe results from source apportionment studies.

Source apportionment was applied to $PM_{2.5}$ collected from Washington DC over nine years, and resulted in the determination of nine different particle types that were characteristic of nine different sources of $PM_{2.5}$ (Hopke et al. 2006). When the source-apportioned PM were analysed against daily mortality data from Washington DC, the largest and most significant increase in mortality was associated with increased levels of $PM_{2.5}$ related to the secondary formation of sulphate (*Table 5.2.2*) (Ito et al. 2006). The increase in daily mortality occurred with a three-day lag after the measurement of secondary sulphate $PM_{2.5}$. Consistent results were obtained when the same data were analysed by different investigators using different source apportionment methods. $PM_{2.5}$ derived from traffic was associated with a smaller, non-significant, increase in mortality (Ito et al. 2006). When the same investigators analysed similar data from the city of Phoenix, increased secondary sulfate-$PM_{2.5}$ was associated with a significant increase in daily cardiovascular mortality, but not all-cause mortality (Mar et al. 2006).

Table 5.2.2 Epidemiological studies of PM linked to coal-fired power station emissions

Location	PM pollution	Health outcome	Health effects*		Reference
Short-term exposure		*Daily*			
Washington DC	Source apportioned secondary sulphate-$PM_{2.5}$	All-cause mortality Cardiovascular disease mortality	**6.7%↑** 6.0%↑	per 90 percentile increase in source apportioned $PM_{2.5}$	(Ito et al. 2006)
Phoenix	Source apportioned secondary sulphate-$PM_{2.5}$	All-cause mortality Cardiovascular disease mortality	2.0%↑ **16.0%↑**	per 90 percentile increase in source apportioned $PM_{2.5}$	(Mar et al. 2006)
6 US cities, mid-west & eastern states	Source apportioned coal combustion $PM_{2.5}$	All-cause mortality COPD mortality Pneumonia mortality IHD mortality	**1.1%↑** 4.5%↑ **7.5%↑** not significant	per 10 $\mu g/m^3$ increase in source apportioned $PM_{2.5}$	(Laden et al. 2000)
Atlanta	Source apportioned secondary sulphate-$PM_{2.5}$ and <u>total</u> $PM_{2.5}$	Respiratory disease hospital ED visits Cardiovascular disease hospital ED visits	**1.2-2.0%↑** 0.8%↑ 0.5-1.0%↑ **2.5%↑**	per IQR increase in source apportioned $PM_{2.5}$ per IQR increase in <u>total</u> $PM_{2.5}$ per IQR increase in source apportioned $PM_{2.5}$ per IQR increase in <u>total</u> $PM_{2.5}$	(Sarnat et al. 2008)
Barcelona	Source apportioned secondary sulphate-$PM_{2.5}$ and <u>total</u> $PM_{2.5}$	All-cause mortality Cardiovascular disease mortality	1.4%↑ 2.0%↑ **7.0%↑** 4.0%↑	per IQR increase in source apportioned $PM_{2.5}$ per IQR increase in <u>total</u> $PM_{2.5}$ per IQR increase in source apportioned $PM_{2.5}$ per IQR increase in <u>total</u> $PM_{2.5}$	(Ostro et al. 2011)
Long-term exposure		*Study period*			
100 US metropolitan sites	$PM_{2.5}$ with coal trace elements	All-cause mortality IHD mortality Respiratory disease mortality Lung cancer mortality	**1.0-1.8%↑** **2.2-3.5%↑** 1.0%↓ **1.0-1.8%↑**	per IQR increase in speciated $PM_{2.5}$	(Thurston et al. 2013)

* Health effects were reported as relative measures and do not indicate absolute changes in health outcomes. Some values have been estimated from figures in original sources.

↑ indicates an increase in the relative measure; health effects in bold indicate a statistically significant effect.

COPD, chronic obstructive pulmonary disease; IHD, ischaemic heart disease; ED, emergency department; IQR, interquartile range

Source apportionment applied to an analysis of data from the *Harvard Six Cities Study* found that a 10 μg/m^3 increase in $PM_{2.5}$ derived from coal combustion was associated with a significant increase in daily all-cause mortality (Laden et al. 2000). This is a similar effect to that observed for total $PM_{2.5}$ in the *Harvard Six Cities Study* (1.5% increase in daily mortality per 10 μg/m^3 increase in $PM_{2.5}$) (Schwartz et al. 1996) and so it is debatable whether, in this study, $PM_{2.5}$ derived from coal combustion has any effect on mortality beyond that of $PM_{2.5}$ in general. It may be that impacts on mortality from $PM_{2.5}$ exposure in studies conducted where coal combustion is a major source of $PM_{2.5}$ (*i.e.*, eastern US) is primarily the result of exposure to PM generated from coal-fired power station emissions. However, against this proposition, source apportionment has determined that $PM_{2.5}$ from mobile sources (vehicle exhaust) had an even greater effect on daily mortality (mortality increased 3.4% per 10 μg/m^3 increase in $PM_{2.5}$) (Laden et al. 2000). PM from vehicle exhaust was strongly associated with deaths due to ischaemic heart disease but not respiratory deaths, whereas PM derived from coal combustion was more strongly associated with respiratory deaths than ischaemic heart disease deaths (Laden et al. 2000). The study investigators proposed that these source-specific particles may affect health by different mechanisms. The original analysis of the *Harvard Six Cities Study* (Dockery et al. 1993), a cohort study designed to examine the effects of long-term exposure to PM, was unable to differentiate between $PM_{2.5}$ and sulphate effects because concentrations of $PM_{2.5}$ and sulphate across cities were highly correlated (Reiss et al. 2007).

Interquartile range increases in sulphate-rich secondary $PM_{2.5}$ (determined by three different source apportionment methods) were associated with significant increases in daily respiratory disease-related visits to hospital emergency departments in the city of Atlanta (Sarnat et al. 2008). Five other source apportioned species of $PM_{2.5}$, as well as total $PM_{2.5}$, were not associated with daily respiratory disease-related visits. Approximately 40% of total $PM_{2.5}$ mass during the four year study period was identified as sulphate-rich secondary $PM_{2.5}$. Increases in sulphate-rich secondary $PM_{2.5}$ were not associated with daily cardiovascular disease-related emergency department visits, however, increases in $PM_{2.5}$ apportioned to petrol vehicle emissions, diesel vehicle emissions and wood smoke were (Sarnat et al. 2008). A time-series study conducted in Barcelona found that increases in source apportioned secondary sulphate-$PM_{2.5}$ were significantly associated with increases in daily cardiovascular mortality but not all-cause mortality (Ostro et al. 2011).

The health impacts of long-term exposure to $PM_{2.5}$ emitted during coal combustion has been examined in an expanded analysis of the *American Cancer Society's, Cancer Prevention Cohort Study* of annual mortality of approximately half a million adults from 100 metropolitan sites in the US. Particle speciation using trace elements associated with coal (arsenic and selenium) determined that 6-year average concentrations of $PM_{2.5}$ from coal combustion were more strongly and consistently associated with all-cause mortality and death from ischaemic heart disease than $PM_{2.5}$ attributed to seven other sources (Thurston et al. 2013). Furthermore, $PM_{2.5}$ from coal combustion was the only $PM_{2.5}$ species associated with lung cancer mortality. This cohort study was able to control for a number of health risk factors at the individual level and therefore provides strong evidence that long-term exposure to $PM_{2.5}$ from coal-fired power stations is detrimental to health. Noticeably, the associations between $PM_{2.5}$ from coal combustion and annual mortality were as strong as the previously determined associations between total $PM_{2.5}$ mass and annual mortality. It was concluded by study investigators that "long-term exposure to $PM_{2.5}$ from combustion sources, especially coal combustion, explain most associations between $PM_{2.5}$ and mortality risk from all causes, ischaemic heart disease and lung cancer, found in earlier studies of the *American Cancer*

Society cohort" (Thurston et al. 2013). This conclusion is supported by similar, and significant, increases in relative mortality risk estimates associated with 10 μg/m^3 increases in either $PM_{2.5}$ or $PM_{2.5}$-sulphate from earlier analyses of data from this cohort (Reiss et al. 2007).

In conclusion, many studies that have apportioned ambient PM to emissions from coal-fired power stations have found associations with all-cause mortality and adverse respiratory and cardiovascular outcomes. However, total ambient PM is often highly correlated with PM derived from coal-fired power station emissions, which can make it difficult to disentangle the effects specific to PM from coal-fired power stations.

A few studies have investigated the impact of local coal-fired power stations on lung function. However, all studies were limited in their ability to attribute deficits in lung function specifically to emissions from coal-fired power stations. Only two studies have included measurements of ambient PM (Aekplakorn 2003, Peled et al. 2005). One of these studies found that impaired lung function in children with asthma was associated with ambient concentrations of PM_{10} and $PM_{2.5}$ in two Israeli towns that were both located near power stations (one coal-fired and one oil-fired) (Peled et al. 2005). The contribution of the coal-fired power station emissions to the ambient PM concentration was not measured. Another study, in Thailand, found an association between 10 μg/m^3 increases in daily ambient PM_{10} levels and reduced lung function in children with asthma, but not in children without asthma, residing near to a coal-fired power station (Aekplakorn 2003). There were no significant associations between increases in ambient sulphur dioxide concentrations and reduced lung function. Again, the contribution of the coal-fired power station emissions to the ambient PM concentration was unknown. Another study from Israel examined the association between lung function development and the estimated individual-level exposure to air pollution of children living in the vicinity of a coal-fired power station. Individual exposure was based on children's place of residence and, air pollution "excessive events" that were attributed to power station emissions (Dubnov et al. 2007). Ambient sulphur dioxide and nitrous oxide concentrations, although not exceeding local pollution standards, were associated with reduced lung function development. The effect estimates were most pronounced in children who had chest symptoms (Yogev-Baggio et al. 2010). The emitted gases will have contributed to secondary PM formation however due to a lack of monitoring PM concentrations were unable to be included in effect estimates.

Colagiuri *et al.*, in a review of the health and social harms of coal mining, cited several studies that have associated living near coal-fired power stations with adverse health effects (Colagiuri et al. 2012). The majority of studies did not measure ambient pollution levels but instead used the distance of residence from the power station(s), biological concentrations of trace elements or, periods of power station operation and closure, as surrogates for exposure. These studies found that the prevalence of laryngeal and bladder cancer (Garcia-Perez et al. 2009), non-melanoma skin cancer (Pesch et al. 2002, Bencko et al. 2009) and, respiratory symptoms (Karavuş et al. 2002) were associated with living near power stations. Exposure to emissions from coal-fired power stations during pregnancy have been related to low birth weight (Mohorovic 2004) and, miscarriage and stillbirth (Mohorovic et al. 2010) in Croatia and, slow child physical development in China (Tang et al. 2006). The effect of socioeconomic confounders needs to be considered when interpreting the results of these studies.

While there is some evidence that living near coal-fired power stations is associated with impacts on health, it is unclear what exposures are relevant. The available evidence is not readily comparable to the situation in Australia because air pollution levels were usually not measured and, the types of coal and power station technologies differ from place to place.

There have been few investigations of the biological mechanisms underpinning the health effects of exposure to PM generated from coal-fired power stations. There have been many more investigations of the toxicity of residual oil fly ash (ROFA), which is emitted from power stations that burn oil. Strong coherence between epidemiological and animal toxicological studies indicate that soluble metals are an important component of PM that are relevant to PM toxicity (Lippmann and Chen 2009). ROFA has been used in toxicity studies because of its elemental similarity to ambient PM (ROFA is chemically complex containing metals, sulphates, acids, fuel contaminants and an insoluble particulate carbon core) and because it is rich in metals and is therefore a useful surrogate to test the hypothesis that metals mediate the biologic effects of PM (Antonini et al. 2002, Ghio et al. 2002). ROFA has a high capacity to induce lung injury in experimental animal models (Antonini et al. 2002, Ghio et al. 2002, Lippmann and Chen 2009). After either intra-tracheal administration or inhalation of ROFA, injury to the lung is evident within 24 hours, with a dose-dependent recruitment of inflammatory cells (Chen and Lippmann 2009). This is accompanied by airway hyper-reactivity and an increase in susceptibility to infections. ROFA exposure also affects heart function (arrhythmias, bradycardia, ischaemia, cardiac inflammation, fibrosis) (Chen and Lippmann 2009). Various studies support the hypothesis that the toxicity of PM is dependent upon the bioavailability of the metal component and the capacity for metals to produce reactive oxygen species and induce inflammatory injury (Ghio et al. 2002). Vanadium and nickel are two toxic metal components of ROFA that have been extensively studied. $PM_{2.5}$ containing vanadium and nickel have been associated with increased cardiovascular and respiratory disease morbidity and mortality (Zhang et al. 2009). Vanadium and nickel were more strongly associated with mortality than other metals in an intervention study in Hong Kong in which the sulphur content of fuels was restricted (Hedley et al. 2002). These metals, and others found in fly ash, have variously been shown to be responsible for many biological effects (Chen and Lippmann 2009). Coal fly ash contains many of the components found in ROFA; however, the chemical characteristics of ROFA are qualitatively and quantitatively different from coal fly ash (Al-Malack et al. 2013) and therefore the health effects from ROFA exposure may not equate to the effects from exposure to coal-fired power station emissions.

Exposure of experimental animals to coal fly ashes show either mild or no fibrosis of the lung at exposure levels that are an order of magnitude greater than levels found in ambient air (Borm 1997). Biomarkers of pulmonary toxicity (oedema, cell injury, inflammation, pulmonary capillary membrane damage) are increased to a greater extent by intra-tracheal instillation of ultrafine coal fly ash compared to coarse fly ash particles and, the type of coal burned affects the response (Gilmour et al. 2004). There have not been sufficient, controlled experiments to generate a hierarchy of toxic potential for different types of coal. A general conclusion is that higher-sulphur (bituminous) coal, the type burned in NSW power stations, produces more toxic emissions than lower-sulphur, sub-bituminous coal and lignite (Gilmour et al. 2004).

Few studies have examined the mechanisms of the toxic effects of inhalation of coal fly ash and there is no consensus on how these particles mediate their biological effects. There is some evidence that inhalation of fly ash suppresses the ability of macrophages and plasma cells to

function and therefore the body may not mount a suitable immune response to inhaled PM (or other respiratory pathogens) (Borm 1997). Exposure of animals to coal fly ash increases bacterial growth and impairs the ability of the immune system to clear bacteria from the lungs (Borcherding et al. 2013). The unifying hypothesis for the toxic effect of combustion-derived particles in general is that these particles cause inflammation via oxidative stress and activation of redox-sensitive transcription factors that lead to adverse health effects such as fibrosis, chronic inflammatory lung disease and cancer (BeruBe et al. 2007). It is not known whether a specific component of fly ash is responsible for toxicity or whether it is the PM as a whole that exerts a biological response. When exposures of ambient PM and fly ash were normalised to give equivalent metal content the biological effects were similar. On a per unit mass basis, PM derived from ambient air exhibits less of an inflammatory effect than PM derived from combustion sources (Chen and Lippmann 2009). This suggests that the bioavailable metal content of fly ash, at least in part, is responsible for the biological effects of exposure. In Taiwan, urinary levels of chromium and arsenic, but not nickel, in children in a school adjacent to a coal-fired power station have been shown to be associated with increased oxidative DNA damage (Wong et al. 2005). In support of the hypothesis that exposure to coal-fired power station emissions generates tissue-damaging reactive oxygen species, workers and surrounding residents of a coal-fired power station in Brazil were shown to have elevated levels of markers of oxidative stress in their blood (Possamai et al. 2010). Given that the vast majority of particulates generated in modern coal-fired power stations are now removed via control technologies prior to emission into the atmosphere, the relevance of coal fly ash to population exposures in Australia is unclear.

The majority of ambient air particles generated from coal-fired power station emissions are secondary PM formed in the atmosphere from sulphur dioxide (and nitrous oxide) emissions. Because of the complexity of atmospheric reactions involved in secondary particle generation it is difficult to determine what the toxic effects of such particles will be. When coal-fired power plant emissions were photochemically aged and "atmospherically transformed" in a laboratory to simulate downwind power station PM pollution, inhalation of these particles by rats produced mild effects (increase in cell number in broncho-alveolar lavage, increase in reactive oxygen species in the heart and lung) that were greater than the effects of "primary" particles (Godleski et al. 2011).

Exposure to PM-sulphate has been associated with impaired blood vessel function in humans that equates to an increase in cardiovascular risk which was especially pronounced in people with diabetes (O'Neill et al. 2005). While the measurement of PM-sulphate does not specifically confer coal-fired power station emissions, the study was conducted in the Boston area, a region exposed to power station emissions. It was concluded by study investigators that diabetes confers vulnerability to PM associated with coal-burning power stations and traffic (O'Neill et al. 2005). An analysis of the biological effects of instilling PM collected from three urban sites and one rural site in the US into the lungs of mice found that coarse PM from a site heavily influenced by emissions from coal-fired power plants caused the greatest increase in lung inflammatory cells (Gilmour et al. 2007). Coarse PM from this site also resulted in the largest increase in a biomarker for cardiac damage however, it was not possible to attribute these changes to coal-fired power station emissions as the site was also heavily influenced by mobile source emissions. The fact that biological effects were greater for exposure to coarse particles, compared to fine particles, and that effects correlated with iron content, but not with zinc or sulphate content, suggests that PM from car brake wear could be contributing to the biological response. COMEAP concluded that either chronic or acute exposure to

sulphur-containing particles generally has little impact and that it is more likely that sulphate interacts with other pollutants to induce a toxicological response (COMEAP 2009). Overall, toxicity studies indicate that specific components of PM derived from coal-fired power stations do not predict health effects as well as PM subjected to simulated atmospheric transformation (US EPA 2012b). Hence, secondary PM derived from gases emitted from coal-fired power stations may be responsible for most of the adverse health effects attributed to emissions from this source.

Some evidence from bacterial and human cell culture assays and, from occupationally exposed populations shows that exposure to coal fly ash is associated with a moderate genotoxic risk (Kleinjans et al. 1989, Stierum et al. 1993, Dwivedi et al. 2012). PAHs are considered the primary human cancer causative agents present in coal combustion emissions (Borm 1997, Lewtas 2007). However, bacterial mutagenic tests have identified hundreds of chemical compounds in ambient PM that contribute to its carcinogenicity and PAHs cannot account for the majority of mutagenic activity of PM (Claxton et al. 2004). Transition metals induce reactive oxygen species, which may result in DNA damage and contribute to the carcinogenic potential of fly ash. With regard to secondary PM-sulphate, far fewer sulphate-containing airborne contaminants have been found to have genotoxic properties compared to hydrocarbons (Claxton et al. 2004). Studies have identified that photochemical and oxidation reactions can increase or decrease the mutagenicity of organic compounds, or even produce mutagenic products from non-mutagenic compounds (Claxton et al. 2004). Emissions from coal combustion in China have been found to have carcinogenic activity but there is little data on the carcinogenicity of emissions from coal-fired power stations in other countries (US EPA 2009). Toxicological studies have shown that inhalation of poorly-combusted industrial coal emissions can cause cancer in research animals. However, these emissions are not equivalent to emissions from current, modern day coal-fired power stations (Lewtas 2007).

It is difficult to quantitatively compare the toxicity of exposure to PM generated from coal-fired power stations and PM from other emission sources. The complexity of atmospheric, secondary PM formation, along with regional variability in the physical and chemical characteristics of PM from coal-fired power station emissions, make it impossible to establish toxicological findings that are applicable to all coal-fired power station exposure situations. Exposure-response studies in research animals have compared responses to chronic inhalation of diesel and petrol exhausts, wood smoke and simulated downwind coal combustion emissions (Mauderly et al. 2014). These studies did not aim to establish relative toxicities of source-based PM, but rather to identify whether specific PM components were associated with different biological responses. The simulated downwind coal combustion emissions were a mixture of components based on historical data of widespread downwind coal combustion emissions and their reaction products. Thus, this does not represent any specific location or set of atmospheric reactions. The major conclusion from these studies is that engine exhaust emissions elicit greater biological effects on the respiratory and cardiovascular systems than either coal combustion emissions or wood smoke (Mauderly et al. 2011, Mauderly et al. 2014). Similar conclusions were obtained from repeated intra-tracheal instillation in research animals of urban air PM from six European cities (Happo et al. 2010). Inflammatory responses were greatest for exposure to PM containing oxidised organic compounds (exhaust emissions), transition metals and soil minerals, whereas PM with compounds recognised as tracers of biomass and coal combustion, reduced the inflammatory response to PM.

Risk assessments in the US and Turkey suggest that the control of sulphur-species emitted from coal-fired power plants has reduced premature deaths and hospitalisations (Buke and Kone 2011, Li and Gibson 2014). It has been estimated that $PM_{2.5}$ generated from coal-fired power station emissions are responsible for approximately 2,500 premature deaths per year in the UK (Yim and Barrett 2012). A figure second only to $PM_{2.5}$ from road transport (4,900 premature deaths). Road transport in the UK uses low sulphur fuels. It was estimated that sulphate contributes 1% of the impact on mortality of $PM_{2.5}$ from road transport whereas sulphate contributes 62% of the impact on mortality of $PM_{2.5}$ from power stations (Yim and Barrett 2012). An intervention in Hong Kong to reduce the sulphur content of fuels in power plants and road vehicles coincided with significant reductions in all-cause, respiratory and cardiovascular mortality (Hedley et al. 2002). Interestingly, this intervention resulted in a sustained reduction in ambient sulphur dioxide concentrations however PM-sulphate concentrations were only reduced for the first year and half after the intervention. Unfortunately ambient $PM_{2.5}$ concentrations were not measured in the Hong Kong study (PM_{10} concentrations did not change through the intervention). Although these results cannot be attributed to changes in power station emissions (since fuels for vehicles were also changed) they do provide evidence that pollution from sulphur-rich fuels has impacts on health.

The National Ambient Air Quality Standard for sulphur dioxide has been found to be one of the most financially beneficial of all US Federal regulations over the past ten years based on the health benefits of improvements in ambient air quality (US Office of Management and Budget 2011). It was noted that more than 99% of the total financial benefits from ambient sulphur dioxide regulations were attributable to reductions in $PM_{2.5}$. Although not all sulphur dioxide emissions are from coal-fired power stations (they are the major source in the US), this report highlights the benefits to population health obtained from reducing $PM_{2.5}$ generated from coal-fired power station emissions.

5.2.4 Summary

There is considerable evidence that exposure to PM derived from coal-fired power station emissions, either emitted directly or formed secondarily in the atmosphere from emitted gases, has adverse impacts on health.

There is some evidence that PM derived from coal-fired power station emissions adversely impacts population health to a greater extent than PM mass in general, however further well designed, sufficiently statistically powered research are needed to confirm or refute this relationship.

Sulphur dioxide emissions from coal-fired power stations are a source of secondary $PM_{2.5}$. Total $PM_{2.5}$ mass is often highly correlated with the mass of $PM_{2.5}$-sulphate and this makes it difficult to differentiate the health effects associated with exposure to total $PM_{2.5}$ versus $PM_{2.5}$-sulphate. Not all epidemiological and toxicological studies have demonstrated that exposure to $PM_{2.5}$-sulphate is associated with health effects. However, in general, where a study has found a significant association between total $PM_{2.5}$ mass and an adverse health outcome, the association is stronger between the health outcome and $PM_{2.5}$-sulphate. This suggests that PM derived from coal-fired power stations impacts population health.

Many of the studies of the health effects of PM-sulphate are from the eastern US, where power stations contribute significantly to ambient PM. The physical and chemical properties of PM derived

from coal-fired power stations depend upon the type of coal burned, emissions controls and, the techniques of coal combustion. Therefore, the health impacts of PM emissions from coal-fired power stations in eastern US may not necessarily be directly applicable to NSW.

Metals that are found in PM emitted from coal-fired power stations have been associated with adverse health effects.

PM derived from coal-fired power stations are dispersed widely and the health effects of these particles may occur at considerable distances from the emission source. There is insufficient evidence that people living close to coal-fired power stations are at an increased risk of the health effects of power station emissions.

- There is strong international evidence that exposure to PM generated from coal-fired power stations is associated with adverse health effects.
- There is some evidence that PM derived from coal-fired power station emissions adversely impacts population health to a greater extent than PM mass in general, however further well designed research is needed to confirm or refute this relationship.
- PM derived from coal-fired power station emissions are dispersed widely and, there is insufficient evidence that people living close to coal-fired power stations are at an increased risk of the health effects of power station emissions.

5.3 On-road vehicles

On-road vehicles are one of the major contributors to potentially toxic urban air pollution. Studies of air pollution within road tunnels (where the majority of PM is traffic-related) have shown that traffic-generated PM contains many metals and PAHs, some of which have toxic effects (Kuykendall et al. 2009). Trace amounts of other toxic substances (dioxins, furans and vehicle-derived refrigerant compounds) have also been found in tunnel air.

Traffic-generated particulates account for the majority of PM in urban areas in highly industrialised countries (as much as 80% in London) (Han and Naeher 2006, Friend et al. 2012). Tailpipe exhaust emissions alone account for up to 30% of $PM_{2.5}$ in urban areas (WHO 2005) and constitute the major source of ultrafine PM (<0.1 µm) in urban environments (Morawska et al. 2008b). On-road vehicles generate PM via tailpipe emissions from fuel combustion (natural gas, petrol and diesel) and from non-exhaust emission sources; including road, tyre and brake wear and road dust re-suspension (*Table 5.3.1*). Tyre and brake wear are an important source of trace metals in urban environments, while particle re-suspension from the road surface can be a significant source of ambient PM, especially in dry climates (Abu-Allaban et al. 2003, Aryal et al. 2011). Secondary particles are formed in the atmosphere from hot exhaust gases expelled from vehicle tailpipes. These secondary particles are generally less than 0.03 µm in diameter and the number of particles is highly unstable and unpredictable (Morawska et al. 2008b).

Table 5.3.1 On-road vehicle primary PM emissions

Source	PM size	PM composition (principal components)
Tailpipe exhaust	$PM_{2.5}$ (predominantly $PM_{0.1}$)	Elemental carbon Organic compounds
Non-exhaust (tyre and brake wear, road dust)	PM_{10} (predominantly $PM_{10-2.5}$)	Metals Organic compounds Crustal material Biogenic material

Sources: (Abu-Allaban et al. 2003, Zhang et al. 2005)

Other substances emitted from motor vehicles, known as mobile-source air toxics, include benzene, formaldehyde, acetaldehyde, 1,3-butadiene, antimony and lead. These substances can combine with emitted PM and can cause adverse health effects (Smichowski et al. 2007, HEI 2010a). Technological innovations in automotive engine design, increased exhaust system efficiency, control technologies that treat exhaust (catalytic converters and particulate filters) and, changes to fuel compositions, have decreased emissions of these substances. Few countries outside of North Africa and the Middle East still allow the sale of petrol that contains lead and all economically advanced countries have motor vehicle emission regulations (HEI 2010a). Improvements in fuel efficiency (primarily for economic reasons and to combat growing greenhouse gas emissions) have also had a downward impact on motor vehicle PM emissions.

More than two decades ago the United Nations estimated that over 600 million people living in cities and towns world-wide were exposed to unhealthy levels of traffic-related air pollutants (Cacciola et

al. 2002). Population exposure to pollution from on-road vehicles is high because the level of pollution from this source tends to be correlated with population density. Ultimately exposure is dependent upon pollution emissions and the time-activity patterns of individuals. Location of residence or work relative to busy roads and time spent commuting in traffic, are critical to dictating the level of exposure (WHO 2005). People with high exposure to traffic-related air pollution include those who live near busy roads, those whose homes are ventilated with air drawn from heavily trafficked road canyons, regular road users (drivers, commuters and pedestrians) and, people whose occupations require them to spend long periods of time on or near roads. In most cities, based on these criteria, many people are highly exposed to traffic-related air pollution.

Improvements in fuel and engine technologies have reduced motor vehicle exhaust emissions of PM in some countries (34-37% reduction in the United States from 1990 to 2007) (HEI 2010a). However the increasing urbanisation of global populations and the expansion of the vehicle fleet in rapidly developing economies (*Table 5.3.2*) have increased the proportion of the global population exposed to traffic-related air pollution.

Table 5.3.2 Growth of motor vehicle fleets 2000-2012, showing the increasing contribution of China to the growing global motor vehicle fleet

Years	Average annual percent increase in motor vehicle registrations[a]		
	Global	China	United States
2000-2005	22	3	5
2005-2010	34	9	2
2010-2012	41	16	2

[a] Combined car, truck and bus registrations

Source: (Davis et al. 2014)

Technological improvements that have reduced traffic-related air pollution in developed economies only apply to emissions from vehicle exhaust. The non-exhaust sources of traffic PM can contribute as much as tailpipe exhaust to ambient PM in cities, particularly in cold climates with winter road sanding and studded tyres (Querol et al. 2004, Thorpe and Harrison 2008). There are no actions currently in place to reduce these non-exhaust emissions (Amato et al. 2014). As vehicle exhaust emission standards have been implemented and strengthened and, vehicle kilometres travelled (VKT) have grown, the proportion of PM traffic emissions arising from non-exhaust sources has increased. In Europe, from 1999 to 2010, the fraction of road transport PM_{10} emissions arising from non-exhaust sources increased from 23% to 36% (Denier van der Gon et al. 2013). US EPA emission inventories indicate that in 2007, on-road vehicle exhaust emissions contributed 0.9% of total PM_{10} emissions. However if the contribution of paved road dust is included, the total PM_{10} contribution from motor vehicles would be approximately 16% (HEI 2010a). Although available data restricts the possibility of making precise estimates of exposure, traffic can still be said to be responsible for an increasing proportion of the population's exposure to PM air pollution (WHO 2005).

Despite the considerable contribution of non-exhaust PM emissions to total aerosol particulates, the introduction of on-road vehicle exhaust emission standards have had beneficial effects. Not only have per-vehicle emissions of mobile-source air toxics been reduced, but so have emissions of ozone precursors, greenhouse gases and $PM_{2.5}$ (including ultrafine PM). On-road vehicle exhaust emissions make a greater contribution to ambient air $PM_{2.5}$ than to ambient air PM_{10}. Given that $PM_{2.5}$ are considered to have a greater impact on health than PM_{10}, the improvements in vehicle exhaust emissions are likely to have had a significantly greater effect on population health than increases in non-exhaust vehicle emissions.

On-road vehicle emissions are complex in terms of chemical composition, time and space. Despite limited systematic chemical characterisation of roadside environments, the US EPA has identified more than a thousand chemical compounds in motor vehicle emissions that might pose potential health risks (HEI 2010a). Atmospheric transformations of on-road vehicle PM emissions include nucleation, coagulation, particle growth, and gas-particle interactions. Diurnal variations in PM concentrations around roadways closely follow temporal variations in traffic density, with the highest levels observed on weekdays during rush hours (Morawska et al. 2008b). The spatial dispersion of PM from roadways is complicated and dependent upon wind speed and direction relative to the road, atmospheric stability, road elevation, and the surrounding terrain.

In urban environments the major source of ultrafine PM is on-road vehicle exhaust emissions (Kumar et al. 2014a). The concentration of ultrafine PM is considerably higher than background levels for a distance of hundreds of metres from roadways (*Table 5.3.3*) (Johnston et al. 2013b). Steep gradients of ambient ultrafine PM concentrations result in substantial spatial variation in ultrafine PM in urban environments. In locations where traffic-related pollution concentrates, such as street canyons and road tunnels, ultrafine PM can be up to 7-16 times higher than the urban background level (Morawska et al. 2008b). Ultrafine PM typically constitute approximately 90% or more of the number of particles in areas influenced by vehicle emissions (Knibbs et al. 2011). This is significant given that these small particles have been identified as having a role in various health effects (Hoek et al. 2010, HEI 2013). $PM_{2.5}$ concentrations are marginally above background concentrations at roadsides, and with a very shallow spatial concentration gradient there is little change in concentration with distance (Karner et al. 2010). The uniformity of $PM_{2.5}$ relates to $PM_{2.5}$ in urban areas typically being well mixed (HEI 2010a). PM_{10} concentrations near traffic are higher than background concentrations but are efficiently removed by settling within approximately one hundred metres of roadways. Other air pollution components that are often in greater abundance near roads include PM-bound PAHs and metals (copper, iron, tin, barium, zinc), nitric oxides, carbon monoxide, benzene and formaldehyde (HEI 2010a).

Table 5.3.3 Concentrations of PM around roadways

PM pollutant	Increase above urban background at roadside	Distance from roadside to reach urban background levels
Ultrafine PM ($PM_{0.1}$)	4-7×	200-900 metres
$PM_{2.5}$	Marginally above background	Essentially no variation with distance
PM_{10}	1.3-1.4×	Approximately 100 metres

Source: (Karner et al. 2010), using data from 41 studies with wide geographic variation

Spatial variability in the concentration of traffic-related PM presents an opportunity and a challenge to determining the health impacts of exposure. Variation in pollutant concentration is essential to investigating whether exposure is related to health outcomes, however high spatial variability make average concentrations derived from measurements taken at different locations around a city uninformative. Population-orientated central, ambient air pollution monitors cannot capture spatial variability. Thus methods often used to determine exposure to ambient PM are not appropriate for determining the exposure to PM from traffic. High spatial variability in traffic–related PM exposure in urban environments is a potential source of exposure misclassification (discrepancy between an individual's assessed exposure and true exposure). It is reasonable to expect that misclassification will result in both over- and under-estimation of exposure (non-differential misclassification). This will dilute any measured exposure-health outcome relationship such that the true relationship will likely be stronger than that which is measured.

Exposure to traffic-related PM pollution is often estimated using the surrogates, distance of residence from roadways and detailed traffic density data (number and type of vehicles, driving speeds *etc*.) (HEI 2010b). However living close to high traffic density is also associated with low socio-economic status and, exposure to noise and air pollutants other than PM. Each of these factors also impacts health. If measured, these confounders can be adjusted for during data analysis. Another approach to assessing traffic-related PM pollution exposure is to use a component of PM such as elemental carbon to represent exposure. The problem with the use of these "marker substances" is that there is no universal indicator that is specific to traffic-generated PM. Since virtually all PM emission sources, especially combustion sources such as vehicle exhausts, emit many different types of pollutants (including gases), a study based on ambient data will almost certainly suffer from co-pollutant confounding (Lipfert and Wyzga 2008). Mathematical dispersion or line models that predict emissions from on-road motor vehicles are a means of estimating exposure levels (HEI 2010a). These models incorporate other parameters, besides linear distance and traffic data, which influence the level of exposure to traffic-related air pollution, such as local weather, climate, time-activity data and land use. These data account for the dispersion of traffic-related air pollution and the contribution of other pollution sources. The validity of a model is dependent, in part, upon the accuracy of the data inputs. No single method of estimating exposure to traffic-related PM is without significant limitation, however findings obtained using a variety of methods can amount to considerable evidence of an exposure-response relationship.

5.3.1 Nature of the contribution of on-road vehicles to PM in NSW

According to the 2008 NSW Air Emissions Inventory, on-road vehicles (including passenger and light duty commercial petrol vehicles and, light and heavy duty diesel vehicles) contributed 10.3% and 13.2% to the total PM_{10} and $PM_{2.5}$ emissions in the Sydney region, respectively (*Table 5.3.4*). Emissions from vehicles powered by natural gas are not included in these figures however there were few of these vehicles in the NSW fleet and liquefied petroleum gas and compressed natural gas engines produce very low levels of PM (Department of Transport and Regional Services 2005).

Table 5.3.4 Contribution of on-road vehicles to PM_{10} and $PM_{2.5}$ emissions in the Greater Metropolitan Region (GMR) and the Sydney region

Source	PM emission details	Proportion of PM emissions (%)			
		GMR		Sydney region (within the GMR)	
		PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
On-road vehicles (excluding diesel exhaust)	The sum of four sources defined in the inventory as: 1. All non-exhaust PM 2. Passenger vehicle petrol exhaust 3. Light duty commercial petrol exhaust 4. Others - exhaust	1.3	2.4	6.2	6.3
On-road diesel exhaust	The sum of two sources defined in the inventory as: 1. Heavy duty commercial diesel exhaust 2. Light duty diesel exhaust	0.9	2.9	4.1	6.9
Total		**2.2**	**5.3**	**10.3**	**13.2**

Source: (NSW EPA 2012a)

The relatively low contribution of on-road vehicles to total PM emissions does not reflect levels of exposure likely to be experienced by urban populations in close proximity to high-density traffic. The high spatial variability of vehicle emissions means that within urban environments there will be some localised areas where the contribution of traffic to ambient air PM concentrations will be very high. For example, motor vehicles' share of PM_{10} in the Sydney central business district has been estimated at approximately 55% (Department of Transport and Regional Services 2005). Thus, much of the urban population of NSW is likely to be exposed to significant levels of traffic-related PM for some of the time, depending upon personal activities in relation to traffic patterns.

The NSW EPA has estimated that PM_{10} emissions from motor vehicles in the NSW GMR (both exhaust and non-exhaust) are currently decreasing and will reach a minimum at approximately the year 2026, after which they are projected to slowly increase (*Figure 5.3.1*). This increase in PM_{10} emissions from 2026 onwards is projected to be largely due to increasing non-exhaust vehicle emissions.

In Australia, new petrol vehicles have only been subjected to emissions standards incorporating PM since 2013 (Department of Infrastructure and Regional Development 2014). Although PM emissions from recently manufactured vehicles are reduced compared to older vehicles, many older petrol vehicles manufactured prior to the latest version of the Australian Design Rules (with accompanying PM emission standards) remain on Australian roads.

Approximately 50% of copper, lead and zinc deposited in Sydney from the atmosphere are attributed to traffic and elsewhere in Australia daily traffic volume correlates with the build-up of heavy metals and organic compounds on road surfaces (Davis and Birch 2011, Mahbub et al. 2011a, Mahbub et al. 2011b). Traffic is likely to be a significant contributor to the exposure of urban populations in NSW to air toxics.

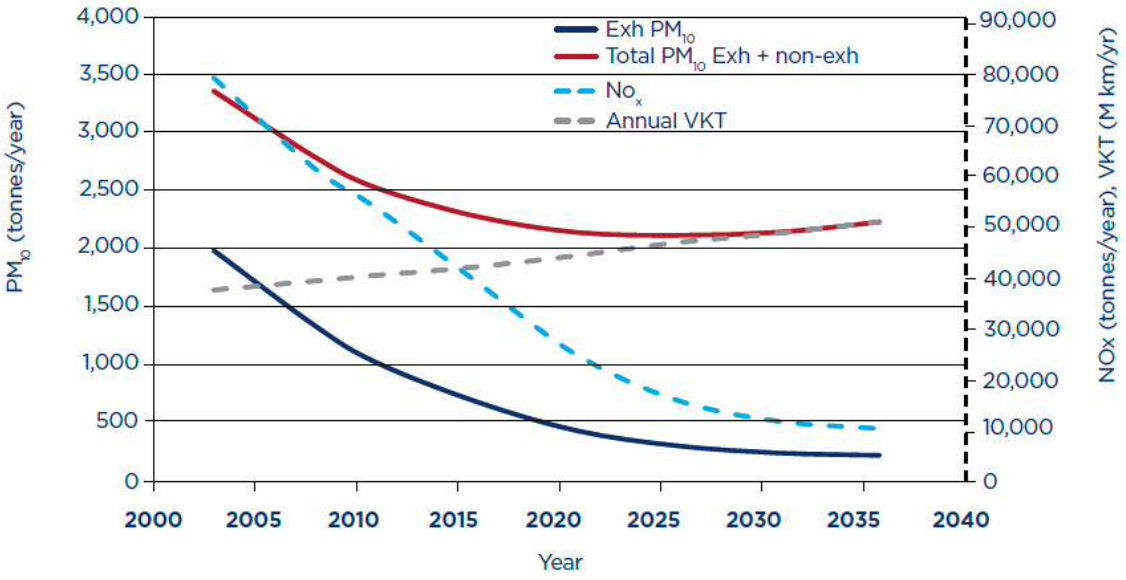

Figure 5.3.1 Projected NSW GMR motor vehicle emissions (NSW EPA 2014d)

Given the proximity of the majority of the NSW population to traffic-related air pollution, on-road vehicles are one of the most important sources of PM exposure in NSW.

5.3.2 Australian evidence of health effects

A few Australian studies have investigated the health impact of traffic-related air pollution. A population-based cohort study in Tasmania, in which both exposures and outcomes were measured by self-report (questionnaire), found that being frequently exposed to heavy vehicles near to home was associated with increased asthma severity, but not with an increase in the prevalence of asthma (Bui et al. 2013). In the absence of objective measurements of exposure to traffic-related air pollution, it is possible that exposure was misclassified in that study. The frequency of intense traffic noise was not associated with asthma severity, indicating that it may well have been traffic-related air pollution, although not necessarily PM, that was associated with increased asthma severity. Traffic-related air pollutants (nitrogen dioxide and carbon dioxide) have been associated with daily childhood (0-4 years of age) asthma presentations to hospital emergency departments in Perth (Pereira et al. 2010).

Estimates of traffic-related air pollution in Perth from measurements of ambient nitrogen dioxide suggested that exposure to traffic-related air pollution during pregnancy is associated with an increased risk of pre-eclampsia (Pereira et al. 2013). A greater number of freeways and main roads around the home have been associated with shorter gestation time in Queensland mothers, without any measured impact on birth outcomes (Barnett et al. 2011). The effect of disturbed sleep due to traffic noise may have been a contributing factor.

No measurements of PM were made in these Australian studies and therefore health outcomes were not specifically associated with exposure to traffic-related PM. In Brisbane, associations between ambient air PM_{10} concentrations and hospital emergency department respiratory

admissions were found to be stronger in those geographic areas with heavy traffic (Chen et al. 2007). This suggests that PM from traffic may impact respiratory health to a greater extent than PM from other sources.

In Sydney, a randomised cross-over study found that short-term exposure (2 hours) of participants to a heavily trafficked road was associated with increased chest and eye symptoms and, increased exhaled nitric oxide (a marker of respiratory airway inflammation) compared to exposure to emissions from a road tunnel ventilation stack (Cowie et al. 2012). The study measured higher PM_{10}, $PM_{2.5}$ and oxides of nitrogen at the heavily trafficked site, but the study did not distinguish which of the specific pollutants were associated with the health impacts. Nevertheless, these findings are consistent with studies conducted elsewhere showing adverse respiratory health effects of acute exposures to traffic-related air pollution.

5.3.3 International evidence of health effects

International evidence indicates that living along busy urban roads is associated with many adverse health outcomes. After adjustment for a range of possible confounders (including socioeconomic status, which is often lower in communities near busy roads), excess health risks associated with close residential proximity to roads have been demonstrated for: mortality, myocardial infarction, sudden cardiac death, cardiovascular disease, atherosclerosis, hypertension, diabetes, asthma hospitalisation, respiratory symptoms, reduced lung function, lung cancer, arthritis, childhood cancer, autism, low birth weight and, cognitive performance in children (Boothe and Shendell 2008, Hart et al. 2009, Hoffmann et al. 2009, HEI 2010a, Nuvolone et al. 2011, Volk et al. 2011, WHO 2013c, Grahame et al. 2014, Hart et al. 2014). Living near a road is a proxy measurement for exposure to traffic-related air pollution but it is not clear whether all of these health outcomes are actually related to exposure to traffic-related air pollution (let alone PM). However many studies with air pollution data specifically indicate that exposure to traffic-related air pollution is associated with increased mortality, respiratory and cardiovascular morbidity and adverse birth outcomes (Heinrich and Wichmann 2004, WHO 2005, HEI 2010a, Laumbach and Kipen 2012, WHO 2013c, Grahame et al. 2014).

The increased risk of adverse health outcomes that is associated with living in close proximity to traffic is unlikely to be explained by $PM_{2.5}$ mass since this component of air pollution is only slightly elevated near roads. Levels of ultrafine PM and certain components of PM such as PAHs and metals are elevated near roads and it is possible that these contribute to elevated health risks. Despite the comprehensive investigation of the health impacts of traffic-derived PM in a variety of settings and utilising different methods, the recent WHO *Review of evidence on health aspects of air pollution - REVIHAAP Project* concluded that because of limited data and large variability in outcomes and source indicators among studies, the harmfulness of traffic-related PM cannot be ranked relative to other particle sources (WHO 2013c).

An analysis of source apportionment data from the *Harvard Six Cities* cohort study in the US showed that ambient concentrations of $PM_{2.5}$ from traffic are positively associated with mortality (Schwartz et al. 2002). Each 10 $\mu g/m^3$ increase in traffic-related $PM_{2.5}$ was associated with an increase in mortality that was double that observed for the same increase in total $PM_{2.5}$. In the same cohort, neither $PM_{2.5}$ from coal combustion nor crustal sources were associated with mortality rates (Laden

et al. 2000). Over a four-year period in Seoul, South Korea, $PM_{2.5}$ apportioned to either petrol or diesel motor vehicles were both significantly associated with daily respiratory mortality whereas other source apportioned $PM_{2.5}$ were not associated with respiratory mortality (Heo et al. 2014). In a study conducted in Barcelona, Spain, $PM_{2.5}$ apportioned to traffic was the source-specific $PM_{2.5}$ that was most strongly associated with daily cardiovascular and all-cause mortality (Ostro et al. 2011). An examination of associations between daily source-specific $PM_{2.5}$ and hospital admissions in New York City found that $PM_{2.5}$ apportioned to traffic was consistently associated with same-day cardiovascular disease, stroke and heart failure admissions, but not respiratory admissions (Lall et al. 2011). The New York study observed that cardiovascular admissions were not associated with total ambient $PM_{2.5}$ or $PM_{2.5}$ from other sources.

In the European multi-country APHEA2 study, PM from areas with relatively high ambient nitrogen dioxide concentrations (a marker of traffic emissions) had larger acute health effects, suggesting that PM emitted by traffic is more toxic than PM from other sources (WHO 2005).

In a review of the health effects of combustion emissions, among studies that measured associations between centrally-monitored components of $PM_{2.5}$ and, mortality and cardiovascular hospital admissions, elemental carbon (considered a measure of traffic emissions) was the $PM_{2.5}$ species most consistently associated with these health endpoints (Grahame et al. 2014). The reviewed studies were conducted in different geographical areas, using different study designs. The consistency of health-exposure associations obtained by multiple study designs in multiple settings is suggestive of a causal association. However, elemental carbon is a particle constituent that is strongly correlated with $PM_{2.5}$ mass and therefore elemental carbon may appear more closely related to adverse health outcomes than other particle constituents even if it is not inherently more toxic.

Evidence published between 1980 and 2008 on the health effects associated with exposure to traffic-related air pollution, has been extensively reviewed by an expert panel for the Health Effects Institute in the US (HEI 2010a). Epidemiological studies were included in the review if they:

- investigated associations between primary emissions from traffic and health; and,
- provided data on local air pollution from a specific traffic source.

The expert panel concluded that:

1. Exposure to traffic-related air pollution is causally associated with the exacerbation of asthma in children;
2. The evidence is suggestive (but not sufficient to fully support) that traffic-related air pollution is causally associated with the onset of childhood asthma, respiratory symptoms, impaired lung function, all-cause mortality, and cardiovascular morbidity;
3. The evidence is inadequate to support exposure to traffic-related air pollution causing cancer, allergies, COPD, adult-onset asthma or poor birth outcomes.

The review identified that many health effects have been associated with exposure to traffic-related air pollution and yet the authors concluded that only evidence relating to the exacerbation of childhood asthma was sufficient to assign causality. Appropriately, the authors applied stringent criteria for causation. The authors cited the following reasons for classifying associations as "suggestive but not sufficient" to infer causation:

- the paucity of evidence;
- health effects could be attributed to confounders;
- inability to compare results from studies with different methods;
- the difficulty in disentangling the effects of traffic-related air pollution from those of general urban air pollution.

The "*suggestive but not sufficient*" label should not be taken as inferring that traffic-related air pollution is not of public health concern. It is more a reflection of the difficulties in designing, implementing, analysing and interpreting studies of the health effects of source-specific air pollution in complex urban environments, than a lack of evidence linking traffic-related air pollution with health outcomes. For example, although there is little epidemiological evidence to link traffic-related air pollution with allergies, human chamber studies and animal experiments indicate that traffic-related air pollution can increase the risk of allergy development and exacerbate allergic reactions (WHO 2005).

The Health Effects Institute review did not delineate which component(s) of traffic-related air pollution (PM, gases or mobile-source air toxics) was associated with the various health outcomes. Animal studies and epidemiological assessment of short- and long-term exposure suggest that not only particles, but also the gaseous fraction of vehicle exhaust (particularly nitrogen dioxide) is associated with adverse health effects (WHO 2013c). Given that ambient PM has been associated with the same health effects that are associated with exposure to traffic-related air pollution it is likely that the PM component of traffic-related air pollution is at least a contributing factor to adverse health effects.

In another evidence review, the US EPA concluded that observed associations between $PM_{2.5}$ and cardiovascular disease hospitalisations may be primarily due to particles from traffic (US EPA 2009). The US EPA assessment implicated traffic-related air pollution generally as a risk factor for myocardial infarction. Evidence from controlled human exposure chamber studies and animal studies is suggestive of a causal relationship between ultrafine PM exposure and cardiovascular effects (US EPA 2009). On-road vehicles are a significant source of ultrafine PM in urban areas and initial evidence suggests that ultrafine particle exposures incurred while commuting are associated with adverse health effects (inflammation, oxidative DNA damage, reductions in lung function and, irregular heart rate) (Knibbs et al. 2011). In Austria, France and Switzerland, it has been estimated that approximately 50% of the mortality attributed to outdoor air PM pollution is attributable to traffic-related PM (Kunzli et al. 2000).

The effect of air pollution from on-road vehicles has been a particular focus in studies of child health (Mejía et al. 2011). An analysis of data from 51 paediatric populations found similar significant increases in asthma symptoms in response to exposure to PM_{10} and nitrogen dioxide, 2.8% and 3.1% per 10 $\mu g/m^3$ increase in PM_{10} and nitrogen dioxide, respectively (Weinmayr et al. 2010). The effects of PM_{10} on asthma symptoms were found to be greater when concentrations of nitrogen dioxide were higher. This suggests that PM specifically from traffic exacerbates childhood asthma. Exposure to traffic-related air pollution and PM have also been associated with new cases of asthma in both adults and children, although it is still unclear whether PM from traffic is responsible for the development of asthma (Gowers et al. 2012, Jacquemin et al. 2012, Guarnieri and Balmes 2014). There is some evidence that genetically susceptible children and adults have an increased risk of

developing asthma in response to exposure to traffic-related air pollution (Castro-Giner et al. 2009, MacIntyre et al. 2014a). Traffic-related air pollution has been associated with bronchitis symptoms in children although the effect was greater for nitrous oxides than it was for PM (Kim et al. 2004). Traffic-related air pollution, but not specifically PM from traffic, has been associated with reduced lung function (Morales et al. 2015), respiratory symptoms (Janssen et al. 2003) and, the prevalence of allergic sensitisation in children (Kramer et al. 2000, Janssen et al. 2003). Exposure to $PM_{2.5}$ from on-road vehicles by mothers during pregnancy has been associated with term low birth weights in Los Angeles (Wilhelm et al. 2012). Other adverse birth outcomes including premature birth, spontaneous abortion and preeclampsia have been associated with exposure to traffic emissions however few studies have attributed these outcomes specifically to PM (Grahame et al. 2014).

Long-term exposure of European populations to traffic-related air pollution has been associated with increases in blood pressure however this relationship was not attributed to PM (Fuks et al. 2014). In a cohort of over 100,000 women in the US, the incidence of lung cancer was associated with exposure to ambient air PM but not with the proximity of a person's residence to roadways (Puett et al. 2014). There is some evidence that long-term exposure to traffic-related PM is associated with lung function decline in the elderly (Lepeule et al. 2014). Repeated exposures to ultrafine particles, such as those incurred by professional drivers, are associated with adverse respiratory, cardiovascular and neurologic effects, and oxidative DNA damage (a mechanism of carcinogenesis and mutagenesis) (Knibbs and Morawska 2012).

Evidence continues to accumulate on the role of oxidative stress in the health risks associated with traffic-related air pollution (WHO 2013c). PM generated from on-road vehicles has high oxidative potential, possibly due to metals arising from engine and/or brake abrasion. This is significant as these non-exhaust pollutants are not currently regulated. Some studies, but not all, have demonstrated that as traffic intensity increases, the capacity of roadside PM to generate tissue-damaging reactive oxygen species increases (de Kok et al. 2006). PM from high-traffic density sites is more toxic to animals than PM from other locations (Gerlofs-Nijland et al. 2007, Gilmour et al. 2007). Road dust and particles from tyre and break wear induce inflammation in cultured cells and pulmonary inflammation in rats (US EPA 2012a, Denier van der Gon et al. 2013). A German cohort study found that C-reactive protein, an inflammatory blood marker and predictor of cardiovascular disease, was increased to a greater extent in response to long-term exposure to traffic-related PM than exposure to total ambient air PM (Hennig et al. 2014). However, particulates collected from the exhaust stack of a motorway tunnel evoked less of an inflammatory response in cultured respiratory airway cells than ambient PM collected from metropolitan Sydney (Kumar et al. 2014b).

The Health Effects Institute review of the effects of traffic-related air pollution indicated that toxicological studies suggest that traffic exhaust emissions (including the PM component) alter cardiovascular and respiratory function and provide plausible mechanisms for the health effects of environmental exposures (HEI 2010a). When viewed together with epidemiological evidence, a stronger case could be made for a causal role for traffic-related air pollution in cardiovascular disease morbidity and mortality than for respiratory health outcomes. Various toxicological and epidemiological studies implicate traffic-related PM as likely to be causal in the associations between ambient traffic-related air pollution and cardiovascular health effects (Lippmann 2014). It is unclear which specific components of traffic-related PM are responsible for health effects. Much has been learned about the cardiovascular and respiratory effects of diesel exhaust exposure where as the

effects of petrol engine exhaust, gaseous versus particulate components of traffic emissions, and road dust, are not well understood.

Lastly, it is likely that the health effects of exposure to traffic-related air pollution will vary regionally. Not only because of differences in the health status of populations, but also due to differences in the exposure itself. The characteristics of petrol and diesel motor vehicle fuel are not the same in different regions of the world. Furthermore, the composition of traffic (*e.g.* more motorcycles in economically developing countries), driving and vehicle maintenance habits and, pollution control measures differ from country to country.

5.3.4 Summary

While none of the proxies for exposure to on-road vehicle PM meet all the criteria for an ideal surrogate for personal exposure, the weight of evidence from a considerable number of studies, employing different methods of exposure assessment in different geographic areas, strongly suggest that exposure to PM generated by traffic is detrimental to health.

Simultaneous emissions of gaseous and particulate pollutants in exhaust and, the disentanglement of traffic-related PM from other PM in complex urban environments, make the estimation of the health impacts from exposure to PM emitted by on-road vehicles a challenge. Not all of the health effects associated with living in close proximity to major roadways have been shown to be related to traffic-related PM. Diabetes, arthritis, cancer and autism have been associated with living near major roadways but there is little evidence relating these diseases specifically to exposure to traffic-related air pollution. It has been demonstrated that PM emitted from traffic is associated with increased risks of mortality, cardiovascular and respiratory morbidity and, adverse birth outcomes. Children, the elderly, people with chronic disease and people otherwise genetically susceptible are particularly at risk of the health effects of traffic-related PM.

Ultrafine PM in motor vehicle exhaust, as well as the metallic components of brake, tyre and road wear are potential sources of toxicity in on-road vehicle PM emissions. Toxicity studies provide mechanistic evidence (inflammation and oxidative stress) that PM from on-road vehicles contribute to the cardiovascular and respiratory health effects observed in epidemiology studies. There is some suggestion from source-apportionment studies that PM from on-road vehicles is more detrimental to health (particularly cardiovascular health) than is PM from other sources.

On-road vehicle emissions contribute significantly to the air that many people breathe. This is particularly so in countries such as Australia, with a majority urban population. Fuel and vehicle exhaust emission regulations have contributed to reductions in on-road vehicle PM emissions however increasing traffic density and unregulated traffic non-exhaust PM emissions mean that PM from on-road vehicles remains a serious public health concern.

- Exposure to PM from on-road vehicles is associated with increased risks of mortality, cardiovascular and respiratory morbidity and, adverse birth outcomes.
- There is some suggestion from source-apportionment studies that PM from on-road vehicles is more detrimental to health (particularly cardiovascular health) than is PM from other sources.
- Not all health effects associated with living near major roadways are necessarily associated with traffic-related air pollution, low socioeconomic status and traffic noise are possible confounding factors.
- Exposure to on-road vehicle PM exacerbates asthma symptoms in children.
- Non-exhaust PM (resuspended road dust and, wear from brakes, tyres and road surfaces) is increasingly becoming an important source of on-road vehicle emissions.
- Toxicology data provide mechanistic support for the health effects associated with exposure to traffic-related PM.
- The health impacts of on-road vehicle PM emissions in Australia have not been extensively investigated.

5.4 On-road diesel vehicle exhaust emissions

Diesel exhaust is a complex mixture of hundreds of constituents in either gas or particle form. Diesel engines produce higher levels of particles than petrol engines although particulate emissions can be drastically reduced with the addition of particulate filters (Bureau of Transport and Regional Economics 2005, Geller et al. 2006). Diesel exhaust particles from modern, optimal combustion engines are primarily $PM_{2.5}$ (including a considerable component of ultrafine particles, $PM_{0.1}$). Diesel exhaust particles have a core of elemental carbon and adsorbed organic compounds and, small amounts of sulphate, nitrate, metals and other trace elements (Wichmann 2007). Metals and other elements detected in diesel exhaust PM include: barium, calcium, chlorine, chromium, copper, iron, lead, manganese, mercury, nickel, phosphorous, sodium, silicon and zinc (US EPA 2002).

Black Carbon and $PM_{2.5}$ mass

Black carbon is a component of $PM_{2.5}$ and is defined as carbon that is measured by light absorption. Black carbon is synonymous with black smoke, elemental carbon and $PM_{2.5}$ absorbance (WHO 2012). Combustion engines, especially diesel, are one of the main sources of black carbon in urban areas.

Health effect-black carbon associations are not quantitatively the same as health effect-$PM_{2.5}$ mass associations (WHO 2012). Studies of short-term health effects suggest that black carbon is a better indicator of harmful PM from combustion sources (especially traffic) than is PM mass, but the evidence from long-term health studies is inconclusive. There is a school of thought that black carbon is (or correlates highly with) a toxic component of PM, especially for cardiovascular effects (US EPA 2012c, WHO 2012). Essentially, because black carbon is highly correlated with $PM_{2.5}$ mass there is not enough evidence to conclude that the health effects associated with exposure to black carbon differ from those associated with exposure to $PM_{2.5}$ mass. The Task Force on Health Aspects of Air Pollution, coordinated by the WHO, recommended that $PM_{2.5}$ (rather than black carbon) should continue to be used as the primary metric for evaluating the health effects of human exposure to PM (WHO 2012).

In practical terms, black carbon is a universal indicator of a variable mixture of particulate material from a variety of combustion sources, not only diesel. For this reason, studies that measured black carbon at the exclusion of PM have not been used in this report to evaluate the health effects of PM from diesel vehicle exhausts.

The large collective surface area of small particles derived from diesel exhaust engenders a large capacity for adsorption of chemical compounds either from gaseous diesel emissions or from the environment. There are some indications that these adsorbed chemicals contribute greatly to the toxicity of diesel emissions (WHO 2013c). The gaseous components of diesel exhaust include: carbon dioxide, oxygen, nitrogen, carbon monoxide, oxides of nitrogen, sulphur dioxide, and numerous hydrocarbons including PAHs. As vehicle exhaust plumes dilute in the atmosphere there

is an evolving competition among new particle formation (via nucleation), particle growth (via condensation and coagulation), and reduction of particle size and mass (via evaporation) (Canagaratna et al. 2010). Reactions of hydrocarbons in diesel exhaust with oxygen species in the atmosphere can result in particle formation directly from gases (Maricq 2007). The contribution of diesel vehicle exhaust to secondary PM in ambient air is uncertain. Some studies have suggested that diesel exhaust is a greater contributor to ambient secondary PM than emissions from petrol fuelled vehicles whereas other studies have suggested the opposite (WHO 2013c). Even subsequent to the coagulation of diesel exhaust particles in the vehicle tailpipe and the adsorption of other compounds, emitted diesel exhaust particles are generally less than 2.5 µm in diameter (D'Anna 2009).

Upon emission from diesel vehicle tailpipes, PM in diesel exhaust is dispersed, transported widely in the atmosphere and coagulates with other atmospheric particles. The atmospheric lifetime of diesel exhaust particles ranges from minutes to several days (US EPA 2002). Limited information is available about the physical and chemical transformation of diesel exhaust PM after emission from the tailpipe and it is not clear what the overall toxicological consequences of these transformations are.

Diesel exhaust PM varies significantly in chemical composition and particle size according to engine type (heavy-duty, light-duty, method of fuel injection), engine operating conditions (idle, accelerating, decelerating) and, fuel formulations (high/low sulphur fuel, petroleum-based diesel, biodiesel) (Maricq 2007). There are currently no clear conclusions on how these differences change the toxicity of diesel exhaust PM (WHO 2013c). Further complicating the picture are atmospheric conditions (temperature, solar radiation, humidity and wind) that can effect diesel exhaust particle number and size distribution (US EPA 2002). There are differences in emissions between on-road and non-road engines due to the aforementioned factors as well as the fact that non-road diesel engines are generally older technology than on-road diesel engines. This chapter deals specifically with on-road diesel emissions (non-road diesel emissions are covered in *section 5.5*).

In developed economies, concentrations of PM in on-road vehicle diesel exhaust emissions have been significantly reduced as more stringent emission regulations have advanced engine and fuel technology (Hesterberg et al. 2011, Brijesh and Sreedhara 2013). In the US, the mass of PM emitted from heavy-duty diesel trucks with post-2006 diesel engine technology is only 1% of the mass of PM emitted from pre-1988 unregulated engines (Hesterberg et al. 2011). Emissions of sulphur dioxide (a precursor to secondary PM formation) from on-road diesel engines have also decreased with the introduction of regulations to lower the sulphur content in diesel fuel (US EPA 2002). PM in modern day diesel vehicle exhaust differs not only in reduced particle mass and number compared to exhaust from older diesel technology, but also in the chemical and physical properties of the particles. These differences in PM from modern diesel vehicles (post-2006 US technology) compared to older diesel engine technology, contribute to differences in toxicity and risk associated with exposure to diesel exhaust (Hesterberg et al. 2011). However despite improvements in technology, diesel vehicles still represent a significant source of PM emissions. Of the exhaust emissions from all vehicles in London in 2009, it was determined that 91% of $PM_{2.5}$ were attributable to diesel vehicles (Moore and Newey 2012). Despite Europe setting stringent standards in diesel vehicle exhaust emission regulation, there are signs that diesel vehicles' increasing share of the vehicle market is reversing the downward trends in emissions from road transport in some cities (OECD 2014).

In Australia, national standards (Australian Design Rules) for new vehicle emissions (petrol and diesel) are the responsibility of the Australian Government and are regularly updated to harmonise with the international regulations of the United Nations Economic Commission for Europe (Department of Infrastructure and Regional Development 2014). Exhaust emissions from in-service passenger and goods diesel vehicles are managed via state and territory governments, which are required to submit annual reports that include: assessments of the need to take action to manage emissions, descriptions of actions taken and, assessment of the effectiveness of these actions (NEPC 2009).

5.4.1 Nature of the contribution of on-road diesel vehicle emissions to PM in NSW

The contribution of diesel exhaust from on-road, heavy duty commercial and light duty vehicles to total PM_{10} and $PM_{2.5}$ emissions in the Sydney Region in 2008 was 4.1% and 6.9%, respectively (NSW EPA 2012a). Heavy duty commercial diesel vehicles comprise rigid trucks, truck-trailer combinations and heavy (>5 tonne) buses. In NSW there are very few heavy duty vehicles that do not use diesel (NSW EPA 2012f). Light duty diesel vehicles comprise both passenger and commercial vehicles. Improved performance and superior fuel economy has increased the numbers of this class of vehicle on NSW roads. In 2008 diesel passenger vehicles comprised 6.3% of new passenger vehicle registrations, up from 0.8% in 2005 (NSW EPA 2012f). The proportion of all registered motor vehicles in NSW that use diesel fuel has continued to increase since 2008 (*Table 5.4.1*). Improvements in engine and fuel technology facilitated by the introduction of national standards for vehicular emissions have reduced PM emissions from on-road diesel engines by around 80-90% since 2003 (Bureau of Transport and Regional Economics 2005). However this reduction will have been partially offset by the increasing numbers of diesel vehicles on NSW roads.

Table 5.4.1 Registered diesel motor vehicles in NSW

Year	Number of diesel vehicles	Diesel vehicles as a proportion of all motor vehicles
2009	507 566	11.1%
2013	756 836	15.2%
2014	843 083	16.5%

Source: (ABS 2014c)

Although fewer in number, the contribution of heavy duty commercial vehicles to ambient air PM should not be underestimated. Air emission inventories combined with transport modelling in urban South-East Queensland has estimated that while heavy duty vehicles (mostly diesel fuelled) contributed only 6% of VKT, they contributed 54% of total particle number in the region (Keogh et al. 2009). It has been estimated that in 2008, heavy duty diesel exhaust contributed 30% and 40% to PM_{10} and $PM_{2.5}$ motor vehicle emissions, respectively, in the NSW GMR (NSW EPA 2012f).

In NSW, the majority of on-road diesel vehicle exhaust emissions occur in Sydney (*Table 5.4.2*), and therefore diesel exhaust PM emissions occur in close proximity to the majority of the population (in

2013, 64% of the population of NSW resided in Sydney (ABS 2014a)). Geographic information system mapping in the US has shown that ambient concentrations of PM from diesel exhaust are increased in highway corridors and within urban areas (McEntee and Ogneva-Himmelberger 2008). While ambient concentrations of PM from vehicle exhaust emissions decrease rapidly within a few hundred metres of roadways (due to dilution and coagulation), concentrations of secondary PM formed from gaseous exhaust emissions do not have such steep spatial gradients (Canagaratna et al. 2010). Essentially, exposure to diesel exhaust PM will be particularly high near heavily trafficked highways and roads, and will be elevated (compared to background levels) in all urban areas in NSW.

Table 5.4.2 Estimated diesel exhaust $PM_{2.5}$ emissions in 2008 in the Sydney Region and in the rest of the Greater Metropolitan Region (GMR)

	Estimated $PM_{2.5}$ emissions (tonnes/year)	
	Sydney Region	**Rest of GMR**
Heavy duty commercial vehicles	574	242
Light duty vehicles	239	60

(NSW EPA 2012f)

The NSW Air Emissions Inventory estimates emissions of PM that are emitted from diesel vehicle tailpipes (primary PM). The contribution that diesel vehicle exhaust gaseous emissions make to ambient PM concentrations via the formation of secondary PM in the atmosphere is unquantified. Source apportionment studies have not identified the specific contribution of on-road diesel vehicle emissions to ambient PM in NSW.

5.4.2 Australian evidence of health effects

Australian evidence of health effects related to exposure to diesel vehicle exhaust is limited to a few investigations of occupational exposures. Research in the Australian mining industry has found that if airborne levels of diesel exhaust PM are reduced, so is the amount of eye and upper respiratory tract irritation (Rogers and Davies 2005). Case-control studies of Australian children with brain tumours and leukaemia suggest that parental occupational exposure to diesel vehicle exhaust may increase the risk of these childhood cancers (Reid et al. 2011, Peters et al. 2013). This effect was not attributed to the PM component of diesel exhaust and no distinction was made between exposure to on-road and non-road diesel vehicle exhausts. A case-control study of Western Australian men found that occupational exposure to diesel exhaust did not increase the risk of prostate cancer (Fritschi et al. 2007). On the basis of international evidence and Australian mining industry studies, the Australian Institute of Occupational Hygienists recommended that worker exposure to diesel PM should be below 0.1 mg/m^3 (8 hour average, measured as elemental carbon) (Australian Institute of Occupational Hygienists 2013). The health effects on which this recommendation was based were primarily eye and respiratory irritation and secondarily, potential lung cancer risk.

Toxicological investigations of the health effects of exposure of research animals to diesel exhaust PM have been conducted in Australian laboratories. These studies are not specific to Australian conditions as they were conducted in controlled environments and the diesel exhaust to which animals were exposed was not specific to Australian diesel fuels and vehicles. Therefore the

evidence from these studies is grouped together below with other toxicological evidence from international studies.

5.4.3 International evidence of health effects

In 1998, the State of California considered that there was sufficient evidence to list diesel exhaust PM as a toxic air contaminant that required action to reduce public exposure and health risk (California EPA Air Resources Board 1998b). This conclusion was based on:

- studies in human volunteers in which intranasal challenge with diesel exhaust particles induced immunological and inflammatory responses;
- diesel exhaust particles being toxic to rodent and human cells and, causing mutations in bacteria and mammalian cells;
- animal studies in which short-term exposure to diesel exhaust particles induced inflammation in lung airways, compromised lung function, and increased susceptibility to infection.

Although not specifically attributed to the particle component of diesel exhaust, both short- and long-term occupational exposures to diesel exhaust have been associated with respiratory symptoms and decreased lung function (California EPA Air Resources Board 1998a). Long-term occupational exposure to diesel exhaust has also been associated with an increased lung cancer risk that was strongly suggestive of a causal relationship. This association was not attributed to the particle component of diesel exhaust, both the gaseous and particle phase of diesel exhaust contain compounds that are potentially carcinogenic (California EPA Air Resources Board 1998a).

In 2002, the US EPA concluded that:

> "*non-cancer effects in humans from long-term exposure to diesel PM are not evident…allergenic inflammatory disorders of the airways and responses typical of asthma have been demonstrated under short-term exposure scenarios to either diesel exhaust or diesel PM…environmental exposure to diesel exhaust may present a lung cancer hazard to humans…the particulate phase appears to have the greatest contribution to the carcinogenic effect*" (US EPA 2002).

Thus from these early reviews of evidence, short-term exposure to diesel exhaust PM was associated with respiratory health effects while long-term exposure to diesel exhaust PM was associated with enhanced lung cancer risk. Short-term exposure studies were conducted in healthy humans (chamber studies), occupationally-exposed workers and, research animals. Long-term exposure studies were conducted in occupationally-exposed workers, populations near major highways (where distance from roadways and concentrations of black carbon were used as proxies for exposure to diesel exhaust PM) and, research animals. Due to the difficulty in distinguishing PM derived from diesel exhaust from PM from other emission sources, no study included in these reports directly assessed the health effects of exposure of the general population to diesel exhaust particulates via measurement of ambient PM.

As a consequence of the stringent regulation of on-road diesel exhaust emissions in countries such as Australia, new technology has resulted in changed diesel exhaust emissions (Brijesh and Sreedhara 2013). The application of new technology has reduced emissions of particle mass, nitrous

oxides, volatile organic chemicals (VOCs) and PAHs in diesel exhaust (Hesterberg et al. 2009). The application of this new technology has generally had positive impacts but it may also have had some adverse consequences. For example, the addition of filter traps has, in specific cases, decreased PM mass in diesel exhaust but significantly increased the number of smaller particles (WHO 2013c). There is sufficient evidence to conclude that the health effects studies of pre-2007 diesel exhaust are likely to have little relevance in assessing the potential health risks of new technology diesel exhaust (Hesterberg et al. 2011, McClellan et al. 2012). For this reason, the remainder of this review is confined to studies and reports published since 2007.

The sophistication of source apportionment methods have not enabled an assessment of the effects of diesel exhaust PM, which is usually included within PM apportioned to total vehicle emissions (which includes emissions from petrol vehicles) (US EPA 2009). Black carbon, elemental carbon and organic carbon have each been used as markers for diesel-derived PM but other combustion sources (other fossil fuels and wood burning) are also sources of these entities. A study in the US city of Atlanta that differentiated ambient PM from diesel exhaust (based on chemical and physical characteristics and correlation patterns among PM at specific locations), found that daily hospital emergency department visits for cardiovascular diseases (but not respiratory diseases) were associated with same-day ambient $PM_{2.5}$ apportioned to diesel exhaust (Sarnat et al. 2008). That study also found that cardiovascular disease (but not respiratory disease) emergency department visits were associated to a similar extent with $PM_{2.5}$ apportioned to petrol exhaust. Thus it is questionable whether $PM_{2.5}$ from diesel vehicles in this study was having a different health impact than $PM_{2.5}$ from other vehicle exhausts. Elemental carbon in $PM_{2.5}$ has also been strongly associated with cardiovascular disease (but not respiratory disease) hospital admissions in 119 US cities (Peng et al. 2009), however as stated above, while elemental carbon is often attributed to diesel exhaust it is not specific to this source. A study in the US that utilised geographical information system mapping found that areas of elevated diesel exhaust PM were clustered with increased asthma incidence in certain towns (McEntee and Ogneva-Himmelberger 2008). In an evaluation of the effects of a ban on diesel motor vehicles in Lebanon it was found that the ban coincided with a reduction in hospital emergency department respiratory admissions for children, but only for the first year after the ban (El-Zein et al. 2007).

Controlled chamber studies of short-term exposures of humans to diesel exhaust particles have reported the following health effects:

- an increase in blood anti-oxidant capacity, possibly in response to increased systemic oxidant stress;
- inconsistent changes to heart rate;
- increased exercise-induced burden on the heart in individuals with cardiovascular disease;
- decreased responsiveness of blood vessels to mediators of vasodilation;
- decreased compliance of arteries and increased vasoconstriction;
- increased blood pressure;
- minimal changes to levels of blood markers of inflammation;
- increased clotting potential of blood and reduced capacity to remove clots that form, which could link short-term diesel exhaust exposure to heart attacks;
- mild nose and throat irritation but an absence of other respiratory symptoms;
- mild constriction of lung airways but otherwise no change in lung function;

- inflammation of the airways;
- enhanced allergic responses in atopic individuals

(Hesterberg et al. 2009, US EPA 2009, Cosselman et al. 2012, WHO 2012, WHO 2013c).

The diluted vehicle exhaust that was used in these studies also contained exhaust gases, which may have contributed to the observed health effects. Exposure of animals to diesel exhaust, from which particulates have been extracted, results in electrical changes in the heart consistent with ischaemic heart disease (Campen et al. 2005). Yet chamber studies in humans have found that extraction of particulates from diesel exhaust prior to exposure results in the diesel exhaust no longer having adverse cardiovascular effects (Lucking et al. 2011, Mills et al. 2011). Clearly exposure to diesel exhaust particulates can exert health (particularly cardiovascular) effects. Many of the human exposure studies have used diesel exhaust PM concentrations well above usual ambient traffic-related exposures. The few studies where exposures were conducted at lower levels provide equivocal findings, with several studies reporting an absence of response (Hesterberg et al. 2009). Overall, the data convincingly demonstrates effects on physiological endpoints with relevance to adverse respiratory and cardiovascular effects (Kelly and Fussell 2012). However, the health impacts of long-term exposure to ambient concentrations of diesel exhaust PM remain unknown. It is also not clear whether particles specifically from diesel engine exhaust are more potent than general ambient PM, on a mass basis (WHO 2013c).

In 2012, the International Agency for Research on Cancer (IARC) classified diesel engine exhaust as "carcinogenic to humans" (IARC 2012a). The evidence on which this conclusion was based came from:

- studies of cohorts of workers who were exposed to diesel exhaust in their occupation (railway workers, bus garage workers, bus drivers, truck and other professional drivers, miners, other professions exposed to diesel exhaust);
- case-control studies in occupationally exposed populations;
- case-control studies in the general population, where exposure to diesel exhaust was estimated from occupational exposure;
- animal studies (associations between exposure and disease in research animals does not establish that such an association exists in humans, however it does provide biological plausibility and all known human carcinogens are also carcinogenic in research animals).

This evidence is not specific to diesel exhaust from on-road vehicles. In fact the strongest evidence in the IARC report came from occupations exposed to non-road diesel exhaust (see *chapter 5.5*). Many occupational studies referred to in the IARC report showed positive correlations between lung cancer risk and the level of exposure. Possible reasons why associations between lung cancer and diesel exhaust exposure were less evident with on-road vehicles (compared to non-road diesel exhaust) are:

- newer technology and more stringent emission regulations associated with on-road vehicle emissions mean that emission intensity from on-road diesel vehicles is lower than from non-road diesel sources;
- in occupational settings it is easier to identify exposure to non-road diesel emission sources (rail locomotives, mining equipment) than it is to identify exposure to on-road diesel emissions. Exposure misclassification and confounding are more likely to be a problem with

on-road diesel vehicle exposure than non-road diesel exposure because on-road exposures do not usually occur at defined industrial sites and are usually accompanied by exposure to petrol vehicle emissions.

The dose-response relationships cited by the IARC support exposure to diesel exhaust as being causative for lung cancer. Positive exposure-lung cancer risk associations remained when results were adjusted for smoking (the strongest known risk factor for lung cancer). Because positive exposure-lung cancer risk associations were seen in different study types and in several occupations, the IARC report indicated that it is improbable that this association is a result of chance, bias or confounding. Yet, a systematic literature review conducted since publication of the IARC report concluded that neither 42 cohort studies nor 32 case-control studies indicated a clear exposure-response relationship between diesel exhaust exposure and lung cancer (Sun et al. 2014). The lack of objective exposure information was cited as the main problem in interpreting the evidence. With the exception of a few case-control studies, where a surrogate air pollution marker was used for diesel exposure (*e.g.* elemental carbon, nitrogen dioxide), exposure assessment was based on work history. Another review of the occupational epidemiological evidence concluded that the evidence was inadequate to confirm the diesel-lung cancer hypothesis and that weak exposure-response associations could be explained by bias, confounding, chance or exposure misclassification (Gamble et al. 2012). A pooled analysis of data from 11 case-control studies conducted in Europe and Canada, found that cumulative occupational diesel exhaust exposure was associated with a significant, 31% increase in lung cancer risk (Olsson et al. 2011). Potential confounders such as smoking and employment in occupations with known cancer risks were adjusted for in the data analysis.

The IARC report presented some evidence that exposure to diesel exhaust was associated with bladder cancer (IARC 2012a). The evidence was neither as extensive, nor of the same quality as that for lung cancer. Evidence relating to other cancers, including childhood cancers, was inconsistent.

As with the chamber studies, the evidence from occupational exposures relates to the whole diesel exhaust mix of gases and particles, and not specifically to the particulate component.

The IARC report did not include evidence from studies of environmental exposures because ambient air pollution comprises emissions from many sources:

> *"At present, it is very difficult to assess the specific contributions of these sources to the observed cancer risk. These (environmental) studies have not been included in the review, because they would contribute little information in addition to the studies reviewed"* (IARC 2012a).

However, evidence from occupational exposures is not directly applicable to the general population for the following reasons:

- workers in diesel-exposed industries are not representative of the general population. They have attributes, such as level of fitness, socioeconomic status and smoking rates, which are different to the general population and can impact on cancer risk. Some of these attributes can be measured and accounted for in data analysis but this is still not equivalent to conducting a population-wide study;

- workers in diesel-related industries are likely to be exposed to significantly higher levels of diesel exhaust than people in the general population. [However, this is not necessarily always the case; implementation of occupational health and safety guidelines can minimise work exposure. Also, work exposure only occurs during the time at work whereas the general population is exposed over a lifetime and potentially for more hours each week.]

Furthermore, in long-term occupational studies, diesel exposure is inferred from occupational data such as job title and length of time in the job. These measures are a poor surrogate for the true underlying exposure determined from personal or nearby fixed site monitoring. Also, it is possible that workers who develop severe disease transfer away from higher exposure jobs and will therefore not be accounted for in occupational studies. These limitations do not discredit the results from occupational studies, but they do impact external validity.

Notwithstanding the fact that risks identified in heavily exposed occupational groups could precede positive findings in the general population, the evidence in the IARC report only demonstrates that diesel exhaust is a carcinogenic hazard in occupational settings. The quantifiable risk to the general population of exposure to on-road diesel exhaust emissions is unknown. It has been estimated that lifetime environmental exposure to diesel engine exhaust results in 21 excess lung cancer deaths per 10,000 people (Vermeulen et al. 2014). This rate is approximately 200-fold greater than usually acceptable limits of lung cancer risk in the US and Europe. However, this estimate was based on three occupational cohort studies in the US that involved older (pre-1998) on-road and non-road diesel technology. Risk estimates from occupational studies do not extrapolate to environmental exposures for the reasons considered above. Furthermore, the risk estimates were based on elemental carbon, a proxy measure of diesel engine exhaust. In the environment, PM-associated elemental carbon is also derived from other combustion sources including petrol vehicle exhaust.

Biodiesel currently represents <2% of diesel use in NSW (NSW Trade & Investment 2014) and is therefore not discussed in detail here. There is conflicting evidence about the extent to which biodiesel exhaust emissions present a lower risk to human health compared to petroleum-based diesel emissions (Hemmingsen et al. 2011, WHO 2013c, Yanamala et al. 2013, Mullins et al. 2014) and more studies are needed in this area.

The toxic potential and associated health risks of diesel exhaust PM relative to other ambient PM is currently unknown. Results of an intervention study conducted in Sao Paulo, Brazil during a strike of the diesel bus fleet, found that PM collected when the buses were running was more mutagenic than PM collected when the buses were not running (Carvalho-Oliveira et al. 2005). This is despite traffic being greater when the buses were not running. The diesel exhaust technology in this study was older and dirtier than currently applies in Australia.

Exposure of experimental animals to diesel exhaust, including isolated diesel engine exhaust particles, has been shown to induce DNA damage, consistent with a carcinogenic effect (IARC 2012a). Exposing research animals to whole diesel exhaust generally increases the incidence of lung cancer (IARC 2012a). The effect of isolated diesel engine exhaust particles has been less consistent however, exposure to only the gas-phase of diesel exhaust has not resulted in an increase in the incidence of lung tumours in any animal species tested to date (IARC 2012a). The IARC report concluded that:

"*There is sufficient evidence in experimental animals for the carcinogenicity of diesel engine exhaust particulate matter*".

However not all animal studies have demonstrated carcinogenic effects. Sub-chronic (1-3 months) and chronic (28-30 months) exposure of animals to US 2007-compliant diesel exhaust has not been associated with markers of genotoxicity or lung cancer lesions (HEI 2012, HEI 2015). These exposures resulted in mild inflammation and oxidative stress in lung tissue and mild, progressive decrease in lung function but only at the highest levels of exposure. It was noted in these studies that biologic responses to new-technology diesel exhaust (*i.e.*, US 2007-compliant) were markedly less severe than observed with traditional-technology diesel exhaust (pre-2007).

Most animal studies of the toxicological effects of diesel exhaust PM have exposed animals via instillation directly into airways or used relatively high inhalation concentrations. As with other PM, exposure of animals to diesel particulates increases oxidative stress in the lung and heart (US EPA 2009). Many studies have examined the respiratory effects of diesel exhaust in animal models and several have demonstrated increases in inflammatory cells and inflammatory response proteins in the lung in response to diesel exhaust exposure (US EPA 2009). Lung cells exposed to diesel exhaust PM release various mediators of inflammation (Schwarze et al. 2013). The organic (PAH) fraction of diesel exhaust PM, and not the metal component, appears to be related to the pro-inflammatory response (Ristovski et al. 2012, Totlandsdal et al. 2013) but it is still unclear which constituents are most important for the inflammatory response. Oxidative stress appears to be central to immune-mediated inflammation resulting from the inhalation of diesel exhaust PM (Ristovski et al. 2012). The receptor for advanced glycation end-products (RAGE) found on the surface of many cell types, and induced during times of oxidant stress, has been implicated in lung inflammation caused by diesel exhaust PM (Reynolds et al. 2011, Barton et al. 2014). Highly elevated doses of diesel exhaust PM leads to chronic lung inflammation in animals, however at lower doses (that are still higher than typical ambient levels), there are few adverse effects (Hesterberg et al. 2009).

As with PM from other sources, it is thought that oxidative stress underpins the health effects arising from exposure to diesel exhaust PM. Within hours of asthmatic subjects being exposed to diesel exhaust, there is a systemic oxidative stress response (Yamamoto et al. 2013). It is possible that diesel exhaust PM may have enhanced effects in individuals with diseases such as diabetes and obesity that are associated with oxidative stress. Systemic effects of diesel exhaust PM were found to be aggravated in an animal model of diabetes (Nemmar et al. 2013b) and inflammation was increased in obese animals exposed to diesel exhaust PM compared to non-obese animals (Moon et al. 2014).

Exposure of research animals to diesel exhaust PM can inhibit bacterial clearance from lungs and enhance the progression of influenza and other viral infections (US EPA 2009, Noah et al. 2012). Diesel exhaust PM may weaken defences against pathogens and enhance susceptibility to infection by increasing proteins in the lung that can act as receptors for bacteria and viruses (US EPA 2009). Diesel exhaust PM has been shown to be an adjuvant to allergens in some (Alberg et al. 2011), but not all (Harkema et al. 2009), animal models of allergic airway disease. Diesel exhaust PM enhances the 'allergenicity' of pollen and house dust mite in mice and results in an atopic response characteristic of asthma (Chehregani and Kouhkan 2008, Takahashi et al. 2010, Acciani et al. 2013). PAHs in diesel exhaust PM may be responsible for diesel exhaust PM promoting the development of

allergic disease (Lubitz et al. 2010). Diesel exhaust PM can directly influence the cell signalling proteins secreted by immune cells and thereby predispose immune defences toward an allergic response (Chan et al. 2006). However diesel particles have not evoked enhanced inflammatory responses in already allergic subjects (Riedl et al. 2012, WHO 2013c). There is much uncertainty associated with extrapolating allergic responses in animals to humans as there is considerable variability in allergic responses across species (Hesterberg et al. 2009). Significant uncertainties remain on the potential mechanisms of diesel exhaust PM-induced exacerbation of allergic and immune responses.

Few animal studies have supported a role for exposure to diesel exhaust PM with respect to cardiovascular effects. Often these studies involved an exposure route (*e.g.* intravenous injection) or high concentration that has limited relevance to inhalation of ambient traffic-related pollution (Nemmar and Inuwa 2008, Kelly and Fussell 2012, Bai and van Eeden 2013). However some animal studies have used inhalation of relevant concentrations of diesel exhaust emissions. Exposure of rats to diesel exhaust PM for three hours resulted in an increase in cardiac arrhythmia in a rat model of chronic heart failure but not in control "healthy" rats (Anselme et al. 2007). When rats were exposed to diesel exhaust PM for one day a week for 16 weeks, markers of vascular impairment were elevated in the aorta (Kodavanti et al. 2011). Diesel exhaust PM may exert cardiovascular effects via oxidative stress-induced reductions in bioavailable nitrous oxide, which is an important mediator of vascular tone and blood pressure control (Miller et al. 2009, Wauters et al. 2013). Instillation of diesel exhaust PM into the lungs of mice results in an increase in atherosclerosis in an animal model of the disease (Miller et al. 2013, Poss et al. 2013). Animal studies have found associations between diesel exhaust PM exposure and an increase in the activation of blood clotting mechanisms, systemic inflammation and, impairments in vasodilation (Hesterberg et al. 2009). Thus, animal studies provide some evidence of the biological plausibility of diesel exhaust-induced cardiovascular health responses in human chamber studies. However, other studies have only observed increases in markers of cardiovascular inflammation in female rats (not males) and only with exposure for 24 months (not exposure ≤12 months) (HEI 2012, HEI 2015).

Diesel exhaust exposure in pregnant mice has resulted in decreased body weights in offspring and, abnormal reproductive effects in primarily male offspring (US EPA 2009, Ema et al. 2013). Intranasal application of diesel exhaust PM in pregnant mice appears to increase asthma susceptibility in offspring (Manners et al. 2014). Exposure to diesel exhaust PM during pregnancy has also been shown to affect the central nervous and immune systems in offspring (Ema et al. 2013). Chronic exposure to diesel exhaust *in utero* and early life increases susceptibility to heart failure in adult mice (Weldy et al. 2013, Weldy et al. 2014). There is no evidence of reproductive or inherited health responses at diesel exhaust levels in the range typical of ambient environments.

As PM ages in the atmosphere, exposure to ultraviolet radiation in sunlight results in photochemical transformations. It is not clear what the overall toxicological consequences of these transformations are because some compounds in the particles are altered to more toxic forms while others are made less toxic (US EPA 2002). Photochemical transformations can enhance the oxidative capacity of particles (WHO 2013c). Oxygenated organic aerosols are strongly correlated to the oxidative potential of diesel PM (Stevanovic et al. 2013) and, the oxygenated organic aerosol component of PM is expected to be greater in secondary PM. Exposure of animals to simulated, atmospherically-aged diesel emissions generally increases the markers of inflammation and oxidative stress

compared to non-aged emissions (Zielinska et al. 2010). Exposing diesel exhaust particles to ambient concentrations of ozone increases the lung inflammation and injury in animals when the particles are instilled in the trachea (Zielinska et al. 2010). Thus, there is indirect evidence that diesel exhaust particles may impact health to a greater extent as they age in the atmosphere.

5.4.4 Summary

The weight of evidence for health and toxicity effects associated with exposure to diesel exhaust PM is greater than for any other single PM emission source. This does not necessarily imply that diesel exhaust PM is more hazardous to health than other source-specific PM, but rather that diesel emissions have probably been the subject of more health and toxicity-effects studies than other PM emission sources. Although there is limited evidence that exposure to diesel exhaust PM is more detrimental to health than exposure to PM in general, a range of adverse health effects of exposure to diesel exhaust have been demonstrated in both humans and animals. These effects are likely to involve inflammation and oxidative stress. Cardiovascular effects have been particularly evident in controlled exposure studies in humans. The contribution of the gaseous and particulate components of diesel exhaust to these health effects is unknown. On-road diesel exhaust PM is potentially carcinogenic, as proclaimed by the IARC, although carcinogenic risks have been based on occupational exposure studies and primarily from non-road sources. The carcinogenic risk from usual environmental exposure to on-road diesel emissions is unknown.

New engine technology that results in more complete combustion in modern on-road vehicles, along with changes in fuel composition and control technologies (particulate filter traps), are likely to have reduced environmental exposures to diesel exhaust PM. However these technological changes may not necessarily have reduced the toxicity of emitted PM. Nevertheless, in some urban areas diesel exhaust particles can be a substantial component of the total PM to which people are exposed and therefore, diesel exhaust particles may disproportionately impact health. Health-effects studies that are relevant to the latest diesel technology are required, as are environmental particle-speciation/source apportionment studies that definitively determine the particles in ambient air that are derived from diesel exhaust. The health hazards associated with road-side concentrations of diesel exhaust PM are largely unknown.

- Controlled (chamber study) exposures to diesel vehicle exhaust has adverse cardiovascular and respiratory effects (particularly cardiovascular) in humans and, cardiovascular, respiratory, reproductive, developmental, cancer and allergy augmentation effects in animals.

- Occupational epidemiology studies have shown that exposure to diesel vehicle exhaust (primarily from non-road diesel sources) is associated with an increase in lung cancer risk.

- Animal studies have demonstrated that the *particulate* component of diesel vehicle exhaust has adverse health effects, including increases in the incidence of lung cancer.

- The biological responses to diesel vehicle PM involve oxidative stress and lung inflammation.

- Studies of the health effects of the general population to environmental exposures to on-road diesel exhaust PM emissions have not been conducted in Australia, due to the difficulty in separating out the effects of on-road diesel exhausts PM emissions from general traffic PM emissions. However the health risks are unlikely to be negligible.

- Further mechanistic and chamber studies of diesel exhaust exposures, using the latest diesel engines and fuel, will help to elucidate the associated health effects.

5.5 Non-road diesel exhaust emissions

Sources of non-road diesel exhaust emissions that make significant contributions to ambient PM include: railway locomotives, shipping and, stationary diesel engines and non-road vehicles in the mining, industrial, construction and agricultural sectors. In Australia, it has been estimated that greater than 87% of annual PM emissions from non-road diesel engines occur in the mining, construction and agricultural sectors (ENVIRON Australia 2010).

As with on-road vehicle diesel emissions, PM emitted from non-road diesel engines contain many chemical compounds. More than 70 organic compounds have been identified in PM emitted from a diesel generator burning low-sulphur diesel fuel (Liang et al. 2005). The PM emitted in that generator's exhaust contained more PAHs than did the diesel fuel itself, a result of combustion-generated products. The characteristics of PM in non-road diesel exhaust depend upon engine technology, engine rating (power), engine operating conditions and fuel formulation (Moldanová et al. 2009, Park et al. 2012, Sippula et al. 2014, Zhang and Balasubramanian 2014). For example, diesel exhaust particles emitted from ships are very small (<1µm) (Diesch et al. 2013) and disperse widely (González et al. 2011).

Worldwide, emission standards for non-road diesel exhaust have been implemented later than for on-road diesel vehicles. PM emission standards for on-road heavy-duty diesel engines were first implemented by the US EPA for engines manufactured in 1988, whereas for non-road equipment and railway locomotive engines, PM emission standards were promulgated in 1996 and 2000, respectively (Hesterberg et al. 2011). Regulations for non-road diesel exhausts have also been introduced in Europe (1997), Canada (2005), Japan (2006), India (2006), China (2007) and Brazil (2011), but not in Australia (ENVIRON Australia 2010). US EPA standards for non-road diesel engines that are yet to be fully phased in will result in the widespread use of diesel particulate filters and reductions in PM emissions of approximately 99% compared to pre-control engines (US EPA 2012c).

The implementation of emission standards for non-road diesel emissions in other countries has had some beneficial environmental effects in Australia as the diesel engines used in Australia are not manufactured locally. They are imported into Australia, mainly from countries that have emissions control standards (67% of non-road diesel engines and equipment is imported from Japan, the US and China) (NSW EPA 2014c). However, an assessment of new non-road diesel engines sold in Australia found that emission limits lagged behind comparable non-road diesel engines sold in the US and Europe. Only 5% of non-road diesel engines sold in Australia in 2008 were reported by industry as meeting the US 2008 emission standards (ENVIRON Australia 2010).

The different (or non-existent) regulations for non-road diesel emissions compared to on-road diesel emissions, along with different engine operating conditions, engine age (usually older engines in non-road applications) and, fuel composition, mean that diesel exhaust emissions are different for non-road diesel engines and on-road diesel vehicles. In NSW, with the exception of some underground mines, all land based non-road diesel equipment uses Automotive Diesel Oil (ADO) which meets the Australian Standards. Some underground mines are permitted to use diesel with a higher sulphur component (section 13 exemption); this diesel produces lower PM concentrations. Unlike on-road vehicles, a wide variety of engine sizes, power ratings and operating conditions apply to non-road diesel engines. Thus, the emissions from different types of non-road diesel engines

could be expected to vary considerably. The vast majority of diesel PM characterisation work has occurred in on-road vehicle exhaust emissions and little is known about how diesel PM emissions from non-road applications differ from those of on-road vehicles (Maricq 2007).

Whilst in port, ships contribute substantial amounts of PM emissions to the environment (Cullinane and Cullinane 2013). There are substantial emissions associated with manoeuvring ships in ports and even whilst stationary, when ships run engines to power ancillary activities. Shore power, also referred to as *cold ironing*, involves switching off auxiliary engines while at berth and supplying the ships with electricity from shore (Holmes 2011). As well as reducing air pollution emissions, shore power has the added benefit of protecting adjacent premises from the noise pollution from generators. Significant reductions in emissions from shipping are also to be gained from moving to low-sulphur marine diesel fuel (Holmes 2011) and implementing post-combustion technologies such as particulate filters (Cullinane and Cullinane 2013).

In Hong Kong, at a site adjacent to a cargo port, ship PM emissions were estimated to contribute 7.6 $\mu g/m^3$ to ambient $PM_{2.5}$ concentrations (Yau et al. 2013). In the US, ambient concentrations of $PM_{2.5}$ in suburbs next to railyards and railways have been estimated to be increased by 1.7-6.8 $\mu g/m^3$ (Galvis et al. 2013, Jaffe et al. 2014). If these examples were to apply in Australia, the contribution of $PM_{2.5}$ from these sources of non-road diesel exhaust emissions would add considerably to the ambient concentrations of $PM_{2.5}$ in areas surrounding these activities.

Overall, PM emissions are greater from non-road diesel engines than from on-road diesel vehicle engines. This is, in part, a result of the non-road diesel engines that are in-service being older than on-road diesel vehicle engines, where equipment turnover is greater. In Australia, non-road diesel engines consume about 70% as much diesel fuel as on-road vehicles, but non-road diesel engines are estimated to emit more particles due to greater emission intensities (NSW EPA 2014c). Heavy duty non-road diesel engines in Australia are estimated to have $PM_{2.5}$ emission intensities that are greater than that of on-road diesel vehicle engines (*Table 5.5.1*) (NSW EPA 2014c). This means that these engines emit more $PM_{2.5}$ per volume of diesel fuel combusted compared to on-road diesel vehicle engines.

In Australia, there are currently no national emission standards for non-road diesel engines. The NSW EPA is pursuing the introduction of such standards (NSW EPA 2014a) and The Senate Committee on the *Impacts on health of air quality in Australia* recommended the implementation of a national emissions standard for small, non-road engines equivalent to the current US EPA standards (The Senate 2013). Emissions from in-service non-road diesel engines remain largely unregulated, with the exception of occupational health and safety regulations for underground mining equipment. However, from 2011 to 2014, the NSW EPA ran a voluntary *NSW Clean Machine Program* that provided subsidies to retrofit older diesel engines with emission reduction devices (NSW EPA 2014c). This lack of regulation is in stark contrast to on-road diesel vehicle engine emissions that are subject to standards and, for which guidelines for a variety of programs in NSW and nationally exist to help reduce emissions (ENVIRON Australia 2010, NSW EPA 2014a). ADO (the main diesel fuel used in the non-road diesel sector (NSW EPA 2014c)), is regulated by national fuel quality standards that first came into force in 2002 (Federal Register of Legislative Instruments 2009). Progressive limitations on diesel fuel content imposed by these regulations, including

threshold concentrations on ash and sulphur, will have had limited impact on reducing PM emissions in the non-road diesel sector.

Table 5.5.1 $PM_{2.5}$ emission intensity of non-road and on-road diesel vehicles and equipment based on the NSW EPA 2008 Air Emissions Inventory

Type	Source of diesel emissions	$PM_{2.5}$ emission intensity (g/L diesel fuel)
Non-road	Coal mining vehicles and equipment	2.73
	Other industrial vehicles and equipment (non-coal mining)	2.86
	Aircraft (ground operations)	0.60
	Commercial boats (excluding shipping/large marine engines)	0.75
	Commercial non-road vehicles and equipment	3.26
	Recreational boats	1.56
On-road	Light and heavy on-road vehicles	0.48*

These data were obtained from a NSW EPA report on potential actions to reduce non-road diesel engine emissions in Australia. The report excluded emissions from railway locomotives and shipping (large marine engines) on the basis that these sources were typically not included in international non-road diesel engine regulations at the time of reporting (2014). Source: (NSW EPA 2014c).
* These figures are as reported in the NSW EPA report. A personal communication from the NSW EPA advised that the emission intensity for on-road vehicles is 1.0 g $PM_{2.5}$/L diesel fuel, not 0.48, but still less than most non-road sources.

Significant growth in non-road diesel equipment use in Australia is projected for the next two decades, primarily in mining (NSW EPA 2014c). Significant estimated increases in emissions and associated health costs accompany these projections (NSW EPA 2014c).

5.5.1 Nature of the contribution of non-road diesel exhaust emissions to PM in NSW

The NSW Air Emissions Inventory for the GMR does not differentiate non-road emission sources on the basis of fuel type, although some sources use primarily diesel fuel. The five largest non-road uses of diesel fuel in the inventory are shown in *Table 5.5.2*.

Together, the five largest non-road consumers of diesel fuel make significant contributions to PM emissions in Sydney and the GMR (*Table 5.5.3*). While on-road vehicles consume more diesel fuel in total than non-road sources, on-road vehicles have lower total PM emissions because they are regulated to meet emission standards and consequently have lower emission intensities (NSW EPA 2014a). Within the emissions inventory, the source categories, *industrial vehicles and equipment*

and *shipping*, are the major sources of PM from diesel exhaust-emitting activities in the GMR and Sydney region, respectively. Non-road vehicles and equipment make up the fourth largest anthropogenic source of $PM_{2.5}$ emissions and the largest unregulated source in the GMR (NSW EPA 2014a).

Table 5.5.2 Estimated annual diesel fuel consumption by the five largest non-road consumers of diesel fuel in the Greater Metropolitan Region (GMR) in 2008

Source type	Annual diesel fuel consumption (*kL/year*)
Industrial vehicles and equipment	737,337
Locomotives (line haul and passenger)	128,836
Commercial boats	120,180
Aircraft ground operations	23,858
Shipping	18,589*

*Fuels such as marine gas oil and residual oil not included.
Source: (NSW EPA 2012e)

Table 5.5.3 Combined contribution of the five largest non-road consumers of diesel fuel to total (anthropogenic and non-anthropogenic) PM emissions in the GMR and Sydney region according to the 2008 NSW Air Emissions Inventory

Area	PM	Combined contribution of PM emissions from the five largest non-road consumers of diesel fuel*	Major source (among five) of PM emissions
		(*% of total PM emission*)	
GMR	**PM_{10}**	2.7	1.7 (Industrial vehicles & equipment)
	$PM_{2.5}$	8.3	5.2 (Industrial vehicles & equipment)
Sydney region (within the GMR)	**PM_{10}**	4.4	2.6 (Shipping)
	$PM_{2.5}$	7.2	4.2 (Shipping)

*The five largest non-road consumers of diesel fuel were: industrial vehicles & equipment, railway locomotives, commercial boats, aircraft ground operations and, shipping. These PM emissions do not represent solely diesel engine exhaust emissions, as while each of these five emission sources uses primarily diesel fuel, other fuel types are also used.
Source: (NSW EPA 2012a)

In the GMR, PM emissions from diesel equipment at industrial premises is primarily attributed to coal mining equipment (NSW EPA 2014a). In a submission to The Senate Committee on the *Impacts on health of air quality in Australia*, the NSW EPA reported that in the Upper Hunter region non-road

diesel equipment is responsible for 13.2% of $PM_{2.5}$ emissions (The Senate 2013). Other industrial activities contributing to non-road diesel emissions in the GMR are metalliferous mines, quarries, landfill or waste operations, ports and, construction and infrastructure projects (NSW EPA 2014a).

Cargo shipping at Port Botany, Port Kembla and Newcastle Port and, cruise shipping in Sydney Harbour, contribute to PM emissions in and around Sydney. Ship emissions inventory data shows that the greatest auxiliary engine energy production is for container vessels at Port Botany, followed by cruise vessels in Sydney Harbour (Holmes 2011). In Sydney Harbour more than half of ship fuel consumption occurs while ships are stationary (Goldsworthy 2014). Most existing berths in NSW ports do not have sufficient electricity infrastructure to provide shore power to ships, although recent planning approvals have required provisions to be made so that shore power can be implemented in the future (Holmes 2011).

The overall significance of non-road diesel engine emissions in NSW is likely to be underappreciated from the NSW Emissions Inventory since the GMR does not cover a significant proportion of mining, agriculture and forestry in NSW (NSW EPA 2014c).

5.5.2 Australian evidence of health effects

Australian evidence of health effects related to exposure to diesel exhaust emissions are limited to a few studies of occupational exposure that are discussed in this report in *Section 5.4.2*.

5.5.3 International evidence of health effects

Investigations of the health effects of exposure to non-road diesel exhaust emissions are almost exclusively from studies in occupational settings, where high exposure levels have been reported for underground mining and tunnel construction (Pronk et al. 2009). In studies examining the health effects of lifetime, cumulative exposure to diesel exhaust, those occupations where high exposure occurs in confined spaces report associations between chronic exposure and health effects (Jarvholm and Reuterwall 2012). This is born out in exposure-response relationships for lung cancer risk, which have been seen in underground miners but not in surface mine workers (Gamble et al. 2012). That is not to say that exposure to non-road diesel exhaust emissions outside of an occupational setting cannot have detrimental health effects. However in an environmental setting it is difficult to differentiate exposure to PM from non-road diesel exhaust emissions from other sources of PM, especially on-road diesel vehicles.

In the single largest study of mortality associated with quantified exposure to non-road diesel exhaust (over 12,000 mine workers from eight US, non-metal mines), diesel exhaust exposure was positively (adversely) associated with lung cancer (Attfield et al. 2012). Exposure was quantified from work histories and measurements of PM at mine sites. As well as observing a positive exposure-response function, lung cancer mortality was higher in the miners than would be expected in a general population. Mortality in the miners from other cancers, heart disease and COPD was not greater than expected in a general population. The cohort study did not take smoking rates into consideration in the analysis, however a nested case-control study (conducted within the cohort) that adjusted results for smoking also found that exposure to diesel exhaust was associated with lung cancer mortality (Silverman et al. 2012). A recent review of the mine worker data, by an expert

panel assembled by the Health Effects Institute, concluded that associations between lung cancer and diesel exhaust exposure in both the cohort and case-control studies were essentially robust to alternative modelling approaches (HEI Diesel Epidemiology Panel 2015). A study of Swedish construction workers found that lung cancer risk was not increased in operators of heavy construction equipment whereas both lung cancer and prostate cancer risk was increased in truck drivers within the construction industry (Jarvholm and Silverman 2003). These results suggest that working with non-road diesel equipment is less of a cancer risk than driving on-road diesel trucks. However the study did not provide any quantitative assessment of exposure to diesel exhaust. A study of German miners found only moderate increases in lung cancer risk with cumulative diesel exhaust exposure and less lung cancer mortality than would be expected in the general population (explained by the study researchers as a "healthy worker effect" and selection bias, as only the healthiest people remained in the historical cohort) (Neumeyer-Gromen et al. 2009). An exposure-response relationship has been demonstrated between diesel exhaust exposure and lung cancer mortality in railway workers in the US (Laden et al. 2006a). The exposure-response relationship was only evident in workers hired after 1945, the year that diesel locomotives were introduced. Diesel exhaust-exposed railway workers (engineers and conductors) hired before 1945 (and therefore not exposed to diesel locomotive emissions for a portion of their working life) had a 30% increased risk of lung cancer mortality compared to "non-diesel exhaust exposed" railway workers (clerks and signal maintainers). Diesel exhaust-exposed workers hired after 1945 had a 77% increased risk of lung cancer mortality compared to their non-exposed counterparts. A secondary analysis of data from over 55,000 railway workers showed that while locomotive workers had a greater lung cancer risk than either shop workers or clerks, the risk of lung cancer for locomotive workers decreased the longer they were employed in this job (HEI 1999).

Pooled analyses of the results of occupational health studies suggest that working in an occupation with diesel exhaust exposure is associated with approximately 33-47% increased risk of lung cancer (Bhatia et al. 1998, Lipsett and Campleman 1999). Those studies included occupations associated with both non-road and on-road sources of diesel exhaust however when the data were stratified by the type of occupation (*e.g.* railway workers, truck drivers, bus workers), both on-road and non-road sources of diesel exhaust were positively associated with lung cancer. In an evaluation of the carcinogenic risk of diesel engine exhausts by the IARC, the strongest evidence of exposure to diesel exhaust being associated with lung cancer came from occupational exposures to sources of non-road diesel (railway workers and miners) (Benbrahim-Tallaa et al. 2012, IARC 2012a). In general, occupational studies where exhaust exposures were predominantly due to diesel engine emissions consistently showed positive associations between exposure and lung cancer risk. While much of the evidence of a lung cancer effect from diesel exhaust exposures does not differentiate between the PM and gaseous components of diesel exhaust, it appears that the particulate phase contributes to the carcinogenic effect (US EPA 2002). The limitations of translating results from occupational to environmental (non-occupational) settings are discussed in *Section 5.4.3*.

Given the increasing concern surrounding occupational lung cancer risk, studies have concentrated on this health outcome. There is little evidence relating occupational exposure to non-road diesel exhaust with other health outcomes. Two studies have reported on the effects on airway inflammation. Non-smoking, underground, Swedish iron ore miners at low risk of silicosis, in a mine using diesel engines, demonstrated persistent airway inflammation (Adelroth et al. 2006). However the air pollution in the mine was complex and airway inflammation might also be attributable to

dust unrelated to the diesel exhaust. An investigation of acute exposure to diesel exhaust in an underground mine via the use of diesel powered and pneumatic equipment on separate days gave inconclusive results concerning the impact of diesel engine exhaust on airway inflammation (Burgess et al. 2007).

The health effects of environmental exposure to non-road diesel exhaust PM is difficult to ascertain as in most settings on-road diesel emissions are ubiquitous and cannot be differentiated from non-road diesel exhaust. An energy crisis in the US required the use of diesel-powered generators to supply electricity to residents of Tacoma, Washington during the winter and spring of 2001. This intervention enabled a comparison of the health impacts of PM when additional diesel generators were, and were not, in use (Mar et al. 2010). Ambient concentrations of $PM_{2.5}$ in Tacoma were not significantly affected by the use of the additional diesel generators. A small positive association between ambient levels of $PM_{2.5}$ and hospital emergency department visits for asthma existed in the city of Tacoma however this association was not observed during the period when the additional diesel generators were in use. Thus there was no evidence from this study that $PM_{2.5}$ specifically from diesel generators were associated with adverse health effects.

An assessment of the health impacts of port-related goods movement throughout the state of California estimated that the majority of deaths associated with this activity were the result of exposure to diesel exhaust emissions PM (California Air Resources Board 2006). It was concluded that the major air pollution source category for mortality effects was trucks (on-road vehicles) moving port cargo, however as regulations reduce diesel emissions from on-road vehicles it is expected that emissions from shipping and railway locomotives will contribute increasingly greater impacts on health near ports (Kuwayama et al. 2013). It has been estimated that globally, shipping-related PM emissions are responsible for approximately 60,000 cardiopulmonary and lung cancer deaths annually (Corbett et al. 2007).

There is some evidence that the operating conditions of non-road diesel engines affect the toxicity potential of emitted PM. PM collected in an underground mine during the operation of an engine at low power (light-load) promoted mutagenesis in bacterial cells whereas PM collected when the same engine was operated at high power (heavy-load) was not associated with mutagenic activity (Shi et al. 2010). Similarly, diesel exhaust from a generator operated at lighter loads caused greater effects on heart rate and rhythm in experimental animals than did heavy-load diesel exhaust (McDonald et al. 2011). However, exposure to the heavy-load diesel exhaust caused more lung inflammation and greater susceptibility to viral infection.

5.5.4 Summary

Few studies have specifically examined the health effects of exposure to non-road diesel exhaust emissions separately from on-road diesel emissions. Since the emission standards that apply to on-road diesel vehicles do not apply to non-road diesel, it would be expected that the health effects of inhalation of non-road diesel exhaust will be as great, or greater, than that of on-road diesel vehicles. Comparative studies of PM from non-road and on-road diesel emissions sources have not been conducted. Most occupational health studies of diesel exhaust exposure relate to non-road emission sources. In occupational settings it has been shown that exposure to non-road diesel exhaust emissions is associated with an increased lung cancer risk. It appears that PM in diesel

exhaust contributes to this effect. Modelling studies suggest that diesel exhaust emissions from shipping have health impacts, however the evidence base is small and further studies in this area are required.

- Occupational exposure to non-road diesel exhaust emissions has been associated with increased lung cancer risk.
- It is difficult to differentiate non-road from on-road diesel exhaust emissions in environmental settings and therefore few studies have examined the health effects of exposure to non-road diesel exhaust PM in environmental settings.
- The comparative health impacts of non-road and on-road diesel PM exhaust emissions are unknown, however due to the lack of emission standards for non-road diesel engines in Australia, $PM_{2.5}$ from non-road diesel sources in NSW is greater than for on-road diesel vehicles.

5.6 Solid fuel (wood) domestic heating

The combustion of carbon-based fuel, including liquid fossil fuel (petrol and diesel) that powers transportation and, solid fuel (wood, coal and biomass) that is used for industrial power generation and domestic heating and cooking, results in PM emissions containing trace elements and organic compounds (Morawska and Zhang 2002). The conditions under which solid fuel combustion occurs (especially in small domestic heaters and cookers) are usually far from optimal. This results in incomplete combustion and the emission of hundreds of different chemical compounds.

It is estimated that more than half of the population in developing countries rely on solid fuel for their household energy needs (United Nations Development Programme and WHO 2009). In the developing economies of Africa and Asia, a very high proportion of PM combustion emissions are derived from solid fuels used for domestic heating and cooking, many of which have sub-optimal combustion (*e.g.* animal dung, crop residue, charcoal, wood, coal) (Grahame et al. 2014). These largely unregulated emissions occur in spaces of human occupancy and are responsible for premature deaths and nearly 5% of the global burden of disease (WHO 2014). In developing countries, household solid fuel use has been associated with low birth weight, pneumonia and acute respiratory infections in children, COPD, chronic bronchitis, cardiovascular disease and, lung cancer (United Nations Development Programme and WHO 2009, Po et al. 2011, Rogalsky et al. 2014). Interventions to reduce indoor exposure to wood smoke via the provision of cooking stoves with chimneys to vent emissions have resulted in decreases in respiratory symptoms, blood pressure and the incidence of low birth weights (McCracken et al. 2007, Romieu et al. 2009, Thompson et al. 2011). As well as affecting health directly, household air pollution from inefficient stoves contributes to the health burden of ambient air pollution in developing countries (Lim et al. 2012).

In developed economies the proportion of PM emissions derived from domestic cooking and heating is significantly less than in developing countries because solid fuels are used less intensively (Grahame et al. 2014). However, domestic cooking and heating can still indirectly generate significant PM emissions in developed economies via coal-powered electricity generation. For instance, in countries with cold winters, the use of solid fuels to heat homes still increases local ambient PM air pollution. It has been shown that household heating with coal, biomass or wood increases ambient levels of PM in European and North American communities (Glasius et al. 2006, Su et al. 2011, Molnar and Sallsten 2013, Saffari et al. 2013, Sari and Bayram 2014, Sarigiannis et al. 2014). In winter, ambient PM can increase up to two-fold in response to solid fuel household heating combined with: topographical features (valley or sheltered area), low wind and a temperature inversion (increasing temperature with altitude), which traps pollution close to the ground (Grange et al. 2013, Trompetter et al. 2013, Sarigiannis et al. 2014). In a rural community in New Zealand, $PM_{2.5}$ from household wood combustion was the major contributor to elevated ambient PM in winter (Ancelet et al. 2013). Programs to reduce household wood burning in the United States have resulted in marked reductions in the levels of wintertime ambient $PM_{2.5}$ (Ward et al. 2010).

In NSW, wood is the preferred choice (over 90%) for domestic solid fuel heaters (NSW EPA 1999). Therefore the remainder of this review will focus specifically on PM emissions from wood combustion and the health effects associated with exposure to PM from wood-fired heaters.

Components of PM emitted from wood combustion that are of health concern include carcinogenic and irritant hydrocarbons (*e.g.* PAHs, benzene), oxygenated organics (*e.g.* aldehydes, phenols) and chlorinated organics (*e.g.* dioxins) (Bari et al. 2011, Masiol et al. 2012). Wood smoke particles are generally smaller than 1 µm (Naeher et al. 2007). The characteristics of emitted PM are dependent upon the type of wood burnt and the intensity of combustion, which is related to the way the heater is used and the specific type of heating appliance (Kocbach Bolling et al. 2009, Syc et al. 2011, Tapanainen et al. 2012). An Australian study showed that the levels and types of PAHs emitted differed according to the types of woods burned for domestic heating purposes (Zou 2003). The chemical and physical characteristics of particles emitted are also influenced by the combustion conditions, which differ among heaters (Lamberg et al. 2011). Changing from an old, less efficient, combustion appliance to a modern appliance can have a greater impact on the toxic properties of emissions than any reduction in the mass of emitted PM might indicate.

A study found that on high-pollution winter days, children in Christchurch, New Zealand were exposed to PAHs emitted from wood-fired heaters (Cavanagh et al. 2007). The health significance of these acute exposures is unknown. It is important to note that there are other sources (*e.g.* road vehicle exhaust emissions) of exposure to these potentially harmful compounds (Delhomme and Millet 2012, Masiol et al. 2012).

Modern, cleaner, solid fuel appliances with flues for extracting emissions from the home are available in developed economies. In homes in Ireland and Scotland that burn wood in flued appliances, indoor air quality is generally good (Semple et al. 2012). Children and women in developed countries are less exposed than in developing countries because they spend more time away from home at school and work. Despite significant reductions in household exposure compared to developing countries, health effects have been associated with wood combustion in homes in developed countries. The use of wood as a fuel in one's own home has been associated with lung cancer (Lissowska et al. 2005, Hosgood et al. 2010), respiratory symptoms in children (Honicky et al. 1985, Bothwell et al. 2003), COPD (Orozco-Levi et al. 2006) and, cardiovascular inflammation (Allen et al. 2011) in both Europe and North America. Indoor exposure of emissions from wood-fired heaters clearly impacts on health in developed countries, although the impact is far less than experienced in developing countries.

As this report is about ambient (outdoor) PM, this review will focus on health effects related to exposure to ambient PM derived from domestic wood-fired heaters, and will not cover the effects of domestic fuel combustion on indoor air quality. Emission intensities for domestic solid fuel appliances in developing countries are not applicable to the Australian situation. Therefore, only epidemiological studies conducted in countries with developed economies like Australia are considered here. This evidence is synthesised with results from animal toxicological studies and controlled human exposure studies.

5.6.1 Nature of the contribution of wood fired emissions to PM in NSW

In the 2008 NSW Air Emissions Inventory, wood-fired domestic space heating was the largest emitter of PM in the Sydney region (27.7% and 46.5% of total PM_{10} and $PM_{2.5}$ emissions, respectively) (NSW EPA 2012a). Wood-fired heating also made large contributions to PM emissions in the GMR (6.2%

and 18.8% of total PM_{10} and $PM_{2.5}$ emissions, respectively). The estimations in the 2008 NSW Air Emissions Inventory were based on a survey of 801 households in the GMR that provided information on the percentage of homes with wood-fired heaters, heater usage, heater type, whether the heater was marked as compliant with the Australian Standard (legislation requires wood heaters sold in NSW to meet emission limits) and, wood usage. The survey estimated that there were 250,000 wood-fired domestic space heaters in operation in the GMR (NSW EPA 2012c). From survey responses collected by the Australian Bureau of Statistics (ABS) from 12,841 households, it was estimated that in 2011, 5% of Sydney households (83,300) and 21% of households elsewhere in NSW (230,300) used wood as their main source of energy for heating (ABS 2011). From 2008 to 2011 the proportion of NSW households using wood as the main source of energy increased (ABS 2011).

Exposure to ambient PM from wood-fired heaters is influenced by:

- the amount of PM from wood combustion that is emitted from chimneys, which is influenced by flue and heater design and, operational practices;
- the local topography; and,
- atmospheric conditions (*i.e.*, temperature inversions and winds).

Like emissions from motor vehicles, emissions from wood-fired heating occur predominantly near to where people live. The proportion of households with wood-fired heating varies considerably by local area (from 5% to 19% by local government area in the GMR) (NSW EPA 2012c). Wood-fired domestic heaters are a seasonal source of emissions. It is likely that under certain conditions (cold weather, temperature inversion, light winds, dense housing in a valley) population exposure to PM from wood-fired heating could be considerable. For example, the city of Armidale on the northern tablelands of NSW, has approximately 50% of homes using wood-fired heaters and, is located in a shallow valley, which creates surface inversion conditions on some winter nights (Hine et al. 2007). It has been reported that 85% of wintertime ambient particulates in Armidale originate from wood-fired heaters and that in the winter months in the three years to 2003, ambient PM exceeded 50 $\mu g/m^3$ on an average of 40 days (Hine et al. 2007). It has been estimated that in Armidale, annual exposure to ambient $PM_{2.5}$ pollution from wood smoke alone was more than double the exposure to $PM_{2.5}$ in Sydney from all sources (Robinson et al. 2007). A source apportionment study of ambient PM in the Upper Hunter Valley towns of Singleton and Muswellbrook found that in winter, 38% and 62% of ambient $PM_{2.5}$ mass was derived from wood smoke in each town, respectively (Hibberd et al. 2013). A source apportionment study in the Sydney suburb of Liverpool, attributed 45% of wintertime $PM_{2.5}$ mass collected at a single monitoring site to domestic wood-fired heating (Cohen et al. 2011). This compares to a contribution of 28% for vehicle emissions in winter in Liverpool.

The perceived risks associated with wood smoke emissions from domestic heating are lower than other emission sources as a result of the social acceptability of this activity and the notion that wood smoke, being a natural substance, must be benign to humans (Hine et al. 2007, Naeher et al. 2007, Reeve et al. 2013). However, there is nothing inherent in the combustion of wood to suggest that inhalation of emitted particles should be less of a health risk than inhalation of PM from other combustion sources.

5.6.2 Australian evidence of health effects

The only Australian studies that have examined the health effects of exposure to ambient air pollution from domestic wood-fired heating are from Tasmania. Two studies were conducted in Launceston, a city in which the use of wood to heat households has historically been high, 66% of households in 1992 (CSIRO 2005). The high use of wood-fired heaters in Launceston combined with topographical and atmospheric conditions that limit dispersion of air pollution, resulted in regular exceedances of the NEPM 24-hour average PM_{10} standard in winter months during the 1990's (Keywood et al. 2000, CSIRO 2005). Air quality in Launceston has improved markedly since 2000, partly in response to a reduction in wood-fired heater use attributable to community education campaigns and a wood-fired heater replacement programme implemented from 2001 to 2004 (CSIRO 2005, Johnston et al. 2013a).

In a retrospective analysis of mortality before and after the interventions to reduce wood-fired heater use in Launceston, it was shown that the improvement in wintertime air quality (including reductions in ambient PM_{10}) coincided with significant reductions in annual mortality for all-cause, cardiovascular and respiratory mortality in men but not women (Johnston et al. 2013a). In wintertime, reductions in cardiovascular and respiratory mortality, for men and women combined, were of borderline statistical significance. There were no significant changes in mortality over the same time period in the city of Hobart, which was used as a control city.

A study comparing self-reported respiratory symptoms in survey respondents of Launceston and Hobart found that in 2004 there was no significant difference in the prevalence of respiratory symptoms between residents of the two cities (Bennett et al. 2010). There were also no significant differences in respiratory symptoms in subgroup analyses of participants from the two cities with pre-existing chronic respiratory disease, and those who used wood-fired heaters in their home. Despite improvements in Launceston air quality, in 2004, ambient PM_{10} concentrations were still higher in Launceston compared to Hobart (Bennett et al. 2010). Either, PM from wood smoke was not having an effect on the respiratory symptoms of Launceston residents, the respiratory health effects of wood smoke emissions were overshadowed by other determinants (confounders) of respiratory health, non-differential misclassification of exposure biased the results towards the null or, selection bias in survey response masked any health effect.

A third study from Tasmania examined the association between subjective self-assessed exposure to ambient wood smoke and the presence and severity of asthma in survey respondents from across the state (Bui et al. 2013). Asthma severity, but not prevalence, was modestly associated with exposure to ambient wood smoke. This association was observed for both users and non-users of home wood-fired heating. The study also found that the frequency of heavy vehicle traffic near to the home was associated with asthma severity (a stronger association than was observed for wood smoke).

Together, these studies provide limited evidence that exposure to ambient PM from wood smoke is responsible for adverse health effects.

5.6.3 International evidence of health effects

The majority of studies of the health effects of domestic wood burning have compared the health of residents of households with wood-fired heaters with the health of individuals in the same community that heat their homes by other means. These studies are primarily addressing the impact of indoor air pollution only. Few studies have examined the impact of ambient air wood smoke on the health of whole communities in areas where wood combustion heating of homes is commonplace. One such study determined the prevalence of respiratory symptoms by questionnaire in two areas of Seattle, Washington, where wood smoke had significantly different effects on ambient air quality because of topographic differences between the two locations (Browning et al. 1990). For the two populations as a whole, there were no differences in respiratory symptoms (cold, cough, congestion and wheezing) however there were more respiratory symptoms in the high wood smoke area in children aged 1-5 years. This study supports other findings from Seattle and elsewhere in the US that young children are particularly susceptible to the adverse health effects of wood smoke. Other health outcomes in children associated with exposure to ambient PM from wood-fired heating include increased hospital emergency department visits and hospitalisations for asthma, increases in asthma symptoms, increases in acute respiratory infections and, decreased lung function (Zelikoff et al. 2002, Boman et al. 2003, Naeher et al. 2007). An intervention in a rural community in the US that reduced the use of wood-fired heaters and subsequently wintertime ambient $PM_{2.5}$ resulted in a reduction in reported wheeze and respiratory infections in children (Noonan et al. 2012).

Time-series studies conducted in areas of high wood-fired heater use during high wintertime ambient PM levels in the US and Christchurch, New Zealand, have shown that days of high ambient PM levels are associated with increases in mortality, total hospital emergency department visits, asthma hospital emergency department visits and, respiratory hospital admissions for all age groups in subsequent days (Schwartz et al. 1993, Lipsett et al. 1997, McGowan et al. 2002, Naeher et al. 2007). Increases in cardiovascular disease hospital admissions were also significantly associated with daily increases in PM_{10} in Christchurch, but to a lesser degree than for respiratory conditions (McGowan et al. 2002). Increases in daily ambient PM_{10} during winter months in Christchurch have also been associated with increases in medication use in susceptible sub-groups such as people with COPD (Town 2001). The greater impact in these studies of wood smoke exposure on respiratory health effects over cardiovascular effects is supported by data from Seattle where $PM_{2.5}$ concentrations were associated with asthma aggravation but not cardiovascular events such as myocardial infarction or sudden cardiac arrest (Naeher et al. 2007).

In Temuco, Chile, one of the most highly wood smoke-polluted cities in the world, ambient PM_{10} was associated with daily total, respiratory and cardiovascular mortality (Sanhueza et al. 2009). However the ambient concentration of PM_{10} (often >100 $\mu g/m^3$ in winter) and the degree to which wood combustion contributed to this, were much higher than experienced in NSW.

A source apportionment study in Copenhagen found an association between ambient PM_{10} attributed to biomass and hospital admissions in the elderly (≥65 years) for respiratory disease, but not cardiovascular disease (Andersen et al. 2007). However as local biomass combustion, mainly from wood, was considered limited, it was surmised that a significant biomass contribution came from long-range atmospheric transport. A source apportionment study from Atlanta, US, found that

same-day $PM_{2.5}$ apportioned to wood smoke was associated with hospital emergency department visits for cardiovascular disease but not respiratory disease (Sarnat et al. 2008). Most studies suggest that exposure to wood smoke particles has a greater impact on respiratory health however the contrary data from Atlanta indicates the need for more studies.

None of these studies collected personal exposure data. Ambient air PM concentrations resulting from domestic wood-fired heating are often spatially heterogeneous, complicating exposure assessment. Furthermore, personal exposure to ambient air during cold winters, when people spend more time inside, will be highly dependent upon personal activities and movements to and from school, work and home. Not all studies have demonstrated associations between health effects and the use of wood-fired heaters. However, the consistency of respiratory health effects (and less so cardiovascular effects) for both indoor and outdoor exposure to wood-fired heater emissions suggests that domestic wood burning is likely to be causative for respiratory health effects. A review of evidence of the adverse health effects of ambient air pollution in relation to residential wood combustion in modern society concluded that the adverse impacts of wood smoke are not less than for other sources of ambient PM (Boman et al. 2003).

Despite that fact that wood smoke contains compounds such as PAHs that have been shown in occupational epidemiology studies to be associated with an increased risk of lung cancer (Armstrong et al. 2004), the evidence that exposure to wood smoke increases cancer risk is weak. Nonetheless, there is an observation of chronic inhalation of wood smoke increasing the incidence of lung cancer in mice (but not rats) (Liang et al. 1988) and, wood smoke emission extracts are mutagenic to isolated cells (Lim and Seow 2012). The majority of evidence relating exposure to wood smoke with lung cancer comes from studies of indoor exposure in developing countries, although lung cancer has also been associated with indoor exposures in Japan, Europe and North America (Sobue 1990, Lissowska et al. 2005, Hosgood et al. 2010, Sloan et al. 2012). The IARC has concluded that *indoor* emissions from household combustion of biomass fuel (primarily wood) are probably carcinogenic to humans (IARC 2010). To date, there is no evidence that exposure to *ambient* air pollution from domestic wood combustion is associated with an increased risk of cancer.

Many constituents of wood smoke can produce acute and/or chronic physiological and structural effects in exposed animals and humans. From this it may be inferred that exposure to wood smoke will elicit a biological response, and in the majority of toxicological studies this is indeed the case (*Table 5.6.1*). The evidence from toxicological studies provides biological plausibility for the epidemiological evidence suggesting that exposure to wood smoke affects human respiratory health.

Controlled exposure of humans to wood smoke particulates have resulted in increases in a blood marker of acute inflammation and alterations in the balance of blood coagulation factors (Barregard et al. 2006). However, neither lung function nor airway inflammation, were affected by acute exposure to wood smoke particles (Sehlstedt et al. 2010, Riddervold et al. 2012).

Toxicological studies suggest that short-term inhalation of wood smoke compromises lung immune defence mechanisms that are important for maintaining host resistance against respiratory infections (Zelikoff et al. 2002). The exposure of animals to wood smoke compromises the ability of host animals to clear bacteria from their lungs (Zelikoff et al. 2002). This effect is not observed in wood smoke from which particles have been removed (Naeher et al. 2007), suggesting that wood smoke particles are responsible for the compromised immune defence.

Table 5.6.1 Biological responses in animals exposed to wood smoke

Responses consistent with exacerbations of respiratory disease
• epithelial cell injury in respiratory airways • increases in the number of lung inflammatory cells and biochemical mediators of inflammation • increased oxidative stress in lungs
Responses consistent with reductions in lung function
• slowed respiration (a reflex resulting from nerve stimulation by wood smoke gases) • increased airway resistance • increased airway reactivity
Responses consistent with increases in respiratory infection
• inhibited clearance of bacteria instilled in lung airways

Sources: (Zelikoff et al. 2002, Naeher et al. 2007)

The effects of inhaled wood smoke in toxicity studies are most dramatic after acute, high-dose exposure. When animals were exposed to wood smoke daily for up to six months at concentrations more relevant to environmental exposures the effect on lung inflammation, cell toxicity, bacterial clearance and carcinogenic potential were mild (Seagrave et al. 2005, Reed et al. 2006).

When cell cultures were exposed to wood smoke particles responses included: increased production of mediators of inflammation, DNA damage, increases in free radicals, oxidative stress response, increases in adhesion molecules and suppression of an immune response (Leonard et al. 2000, Danielsen et al. 2011, Forchhammer et al. 2012, Hawley and Volckens 2013, Migliaccio et al. 2013). These effects are consistent with the health effects observed in animals and humans.

In a crude assessment of the relative health effects of diesel exhaust, petrol exhaust, wood smoke and coal combustion emissions, rats and mice were exposed to these emission sources under the same exposure regime and a variety of biological responses measured (Mauderly et al. 2014). All four exposures caused statistically significant effects. An overall ranking of toxicity was not plausible because of the wide range of effects. However overall, wood smoke elicited the fewest biological responses. Quantifying the hazard of wood smoke particles is complicated by the fact that, to a large extent, the toxicity of emitted particles depends on the type of wood burned, the type of heating appliance and, the combustion conditions (Zou 2003, Kocbach Bolling et al. 2009, Tapanainen et al. 2012, Cassee et al. 2013). A simple risk assessment for wood smoke is not possible since the toxicity of emitted PM can vary significantly. What is clear is that incomplete combustion leads to the emission of hazardous substances and therefore it is prudent to minimise exposure to such emissions.

5.6.4 Summary

The health effects of exposure to ambient PM derived from domestic wood-fired heating has not been extensively studied. The majority of evidence comes from two locations, Christchurch, New Zealand and Seattle, US. The epidemiological and toxicological evidence supports an association between exposure to wood smoke particles and detrimental respiratory health effects, particularly in children. Although the evidence base is limited, the consistency of effects suggests that exposure to wood smoke particles in ambient air is causative for respiratory health effects. However, there have been insufficient studies to conclude on the relative health impact of exposure to ambient PM from wood-fired heating compared to ambient PM in general.

Particles in wood smoke contain hundreds of chemical compounds with toxic properties. The health effects observed in poorly ventilated housing in developing countries demonstrate the potential harms from exposure to these compounds. The concentration of wood smoke particulates in ambient air under usual environmental conditions may not be sufficient to warrant labelling these compounds as hazardous in most circumstances. However the multitude of potential health effects of exposure to wood smoke particles in ambient air have not been rigorously investigated in epidemiological studies.

- Exposure to ambient PM derived from domestic wood-fired heating is associated with detrimental respiratory health effects, particularly in children.
- The health effects of outdoor exposure to PM from domestic wood-fired heating has not been extensively studied and the relative health impact of exposure to this PM compared to ambient PM in general is uncertain.
- The variations in the toxicity of wood smoke depending on the type of wood burned and the degree of combustion make the assessment of the environmental health impact of exposure difficult.
- The large number of potentially hazardous compounds in wood smoke suggests that there are health effects of exposure that remain to be determined.

5.7 Bushfires and hazard reduction burning

Bushfire activity is strongly influenced by four factors – climate, fuels, ignition agents and human activity. There is consensus that global climate change is contributing to the increasing threat of bushfires, albeit with large regional variability (Flannigan et al. 2005, Cramer et al. 2014). Bushfires (elsewhere known as forest fires or wildfires) are forecast to occur more frequently in the future and the fires are likely to be more intense in Australia, California, Southern Europe and, other fire-prone regions of the world (Fried et al. 2008, Clarke et al. 2011, De Sario et al. 2013, Liu et al. 2015). Higher temperatures, longer and more frequent droughts and, the increasing incidence and intensity of storms with accompanying lightning strikes are a dangerous confluence of climate change effects that will increase the potential for ignition and spread of bushfires. As a consequence, exposure to air pollution from bushfires is anticipated to increase (The Interagency Working Group on Climate Change and Health 2010). A rise in the threat of high impact bushfires will increase the requirement to conduct regular hazard reduction burns in order to minimise available fuel, adding to the air pollution burden.

Large wildfires regularly occur in central South America and Africa, often lit deliberately to clear land, but the health impacts in these regions have been little studied. During 1997-2004, Africa and South America together contributed more than 82% to global annual fire emissions (Koble et al. 2008). Other regions particularly impacted by large wildfires are Australia, South-West US, Southern and Eastern Europe, China and, South-East Asia. The overall contribution of bushfires to global atmospheric concentrations of pollutants varies from year to year, depending upon the number and intensity of fires. At a regional scale, the contribution of bushfires to overall ambient air pollution concentrations can be significant. For example, during August 2003, the contribution of bushfire emissions in Southern Europe to ambient $PM_{2.5}$ concentrations appeared to be comparable to that of all anthropogenic emissions (Koble et al. 2008).

Bushfire smoke contains hundreds of air contaminants in both the particle and gaseous phases (Lemieux et al. 2004, Alves et al. 2011, Garcia-Hurtado et al. 2014). Bushfires and prescribed burns result in many of the same emissions as does domestic wood combustion with differences dependent on the fuel (type and condition of the biomass) and burning conditions (*e.g.* oxygen availability for combustion, the high intensity of the fire, and other atmospheric and environmental conditions) (Lewtas 2007, Reisen and Brown 2009). On a global scale, bushfire emissions represent an important source of atmospheric carbon dioxide, carbon monoxide, methane, nitrous oxides, VOCs and PM (Aleksandropoulou et al. 2013). More than 90% of the mass of bushfire particles is organic matter (Vicente et al. 2013). Contaminants in the particle phase that may cause adverse health effects include nitrogen- and sulphur-based compounds, aldehydes, volatile and semi-volatile organic compounds, PAHs, dioxins, organic acids and free radicals (Reisen and Brown 2006). These contaminants (not necessarily in bushfire smoke) have been linked to skin, eye, nose and throat irritation, coughing and wheezing, drowsiness and, cancer (Youssouf et al. 2014b).

The heat of large-scale bushfires results in powerful convection currents taking the air above fires to high altitudes from where air pollution plumes can be carried by wind over very long distances. Particles may be transported up to 3000 kilometres from the fire source (Tomshin and Solovyev 2014). In the South-West US, fires have been causally linked to increased hospital admissions 300-500 kilometres away (Moeltner et al. 2013).

Bushfires generate aerosol particles of varying sizes but, 80%-90% of PM mass produced by bushfires is $PM_{2.5}$ (Vicente et al. 2013, Youssouf et al. 2014a). $PM_{2.5}$ particles are generated directly during the combustion process and also formed secondarily via condensation and the reaction of emitted gases (De Sario et al. 2013). Particles generated by bushfires and by urban sources are similar in size distribution but may be substantially different in chemical constituents. This, together with the presence of different co-pollutants in the two environments, may lead to different health impacts.

Bushfires as a PM emission source are distinct from many other sources in that emission events are sporadic, unpredictable, and often very intense. Exposure to emissions usually occurs over periods of days or weeks. Dust storms and volcanic eruptions are other emission sources that share these characteristics. These determinants limit the types of epidemiological investigations that can be conducted.

Of the many sources of airborne particulates in Australia, bushfires are a source of particular relevance, and hence Australia provides a good setting in which to investigate the health impacts. Approximately half of the published studies on the health impacts of bushfire smoke have been conducted in Australia and California (Liu et al. 2015). The majority of investigations are time-series studies, where the frequency of health outcomes (*e.g.* hospitalisations, symptoms, mortality, and medication use) during and/or immediately after a bushfire event, are compared with the frequency when bushfires were absent. These natural, *ad hoc* experiments do not provide the same quality of evidence as cohort studies, where suspected confounders of exposure-effect associations (*e.g.* pre-existing disease, socio-economic status) can more easily be accounted for. If an impact on population health is observed at the time of a bushfire it cannot be totally discounted that the effect is attributable to some other variable that also changes over time such as a concurrent heatwave.

The principal advantage of investigations of the health effects of exposure to bushfire emissions (compared to other air pollution exposures) is that the exposure is often extreme. Smoke haze in Malaysia from 1997 Indonesian forest fires resulted in 24-hour average PM_{10} levels that peaked at 930 $\mu g/m^3$, 15-times baseline levels (Reisen and Brown 2006). In Europe, California and Australia, ambient PM_{10} and $PM_{2.5}$ concentrations are often over 500 $\mu g/m^3$ and 100 $\mu g/m^3$ respectively during bushfire events (Liu et al. 2015). It is reasonable to conclude that people near or downwind of a bushfire are exposed to bushfire smoke (although actual personal exposure is rarely quantified). In the absence of bushfires, exposure to emissions from this source is nil. Thus, exposure misclassification, which is a major source of error in epidemiological investigations of the effects of outdoor air pollution, is less of a concern in bushfire studies. However, no single method of estimating exposure to bushfire smoke (satellite measurement, ground-based air pollution monitoring, routine data on time and place of fires, questionnaires) is without limitation (Youssouf et al. 2014b). When three different methods were used to assess smoke exposure during a Canadian summer, the associations of exposure with respiratory and cardiovascular health outcomes differed depending on the assessment method used (Henderson et al. 2011). What is important is not that these differences exist, but to recognise the limitations of each method, and to accumulate evidence using different methods in well conducted studies. When similar health outcomes are consistently demonstrated in a variety of different types of epidemiological study, in different populations and, varying bushfire scenarios, causality can more confidently be established.

The intensity of emissions from bushfires poses a difficulty in interpreting study results. With the possible exception of severe dust storms, the level of ambient air PM exposure during a bushfire may be the highest that a population will experience. While it is probable that the health impacts of bushfire PM are attributable to the peak concentrations ambient PM mass, it is also possible that health effects are associated with the toxicity of these specific combustion particles. From a public health perspective, it could be argued that this point is irrelevant. If bushfires increase ambient air PM to hazardous levels it is prudent to minimise exposure.

5.7.1 Nature of the contribution of bushfire smoke to PM in NSW

The NSW Air Emissions Inventory estimated that bushfires and hazard reduction burns combined contributed 2.8% and 7.6% to PM_{10} and $PM_{2.5}$ emissions respectively in the GMR in 2008 (NSW EPA 2012a). However emissions from these sources are sporadic and likely to vary considerably from year to year. Additionally, these relatively small proportions to overall PM are not indicative of the peak PM concentrations that can occur during bushfires. From 1996 to 2007, 46 days were identified where Sydney experienced extreme ambient PM concentrations (PM_{10} or $PM_{2.5}$ greater than the 99th percentile) as a result of bushfires or hazard reduction burns (Johnston et al. 2014). The highest ambient PM levels recorded in NSW are attributable to dust storms and bushfires. Bushfires in 1994 and 2001-03 were major contributors to the extremely high concentrations of particle pollution (exceedances of the NEPM standard for PM_{10}) recorded in the GMR for those years (NSW EPA 2012g). Severe bushfires can cause ambient PM to exceed Australian standards for several weeks (NSW Department of Environment Climate Change and Water 2010). One such smoke event in Albury, NSW in 2003, lasted for 38 days, with a maximum daily-PM_{10} level of 415 µg/m^3 and 21 days on which air quality was defined as *hazardous* or *very poor* (Kolbe and Gilchrist 2009). In Sydney during the period 1994-2007, on those days for which a cause for high ambient PM could be identified, 94% of high-PM days were attributed to bushfires or hazard reduction burns (Johnston et al. 2011b). Thus, smoke from landscape burning is responsible for the majority of extreme ambient PM levels in NSW.

In Australia, between 1973 and 2010, fire-prone weather events (a combination of high surface air temperature, low rainfall, low relative humidity and high wind speed) have become more common, particularly in the southeast of the continent (Clarke et al. 2013). Climate models consistently predict that the future bushfire risk for Southeast Australia will increase (Clarke et al. 2011) and climate change has already impacted on bushfire frequency and intensity in NSW (Hughes 2014). As the bushfire risk increases there is an increased need for hazard reduction burns. These burns add to the air pollution burden in NSW. In NSW during 2012-13, the largest ever hazard reduction burn program was undertaken, comprising a total of 330 burns (NSW Department of Environment and Heritage 2014).

In summary, because bushfires and hazard reduction burns occur sporadically it is difficult to quantify the annual exposure to emissions from these sources compared to other sources of particulates in NSW. However, some of the highest ambient PM levels in NSW are a consequence of these events, and the number and intensity of these events is expected to increase due to climate change.

5.7.2 Australian evidence of health effects

Several Australian studies have compared health outcomes on bushfire and non-bushfire days (*Table 5.7.1*). It has been shown that the positive associations between daily ambient PM_{10} concentrations and daily hospital admissions for respiratory conditions in Sydney (Morgan et al. 2010) and Brisbane (Chen et al. 2006) were stronger during bushfires than when bushfires were not present. Bushfires had no effect on the association between daily PM_{10} levels and daily cardiovascular hospital admissions in Sydney (Morgan et al. 2010). In other studies, extreme ambient PM_{10} and $PM_{2.5}$ concentrations during Sydney bushfire events were associated with increases in mortality (Johnston et al. 2011a), presentations to hospital emergency departments (Johnston et al. 2014) and, hospital admissions for respiratory conditions (Martin et al. 2013). In these studies, high-PM bushfire days were not consistently associated with cardiovascular outcomes. Although some possible confounders of the measured associations were taken into account in the data analyses (*e.g.* temperature, and in some studies, ozone), it is possible that the health outcomes observed resulted from other aspects of bushfires such as anxiety associated with the event and, gaseous pollutants, rather than PM emissions. However, the associations observed between bushfire days and increased respiratory outcomes are consistent with large epidemiological studies of the effects of exposure to PM.

Two other studies found that the Sydney bushfires of January, 1994 had no effect on the number of hospital emergency presentations for asthma (Smith et al. 1996) or children's lung function (Jalaludin et al. 2000). Neither the total suspended particles in ambient air (Smith et al. 1996) nor PM_{10} (Jalaludin et al. 2000) during these bushfires were associated with those outcomes.

Several studies have examined associations between daily health outcomes and PM levels in Darwin during the dry season (April-November), when the surrounding savannah grasslands are extensively burned and the fires are the predominant source of PM. Ambient PM_{10} concentrations in Darwin during the dry season have been shown to be significantly associated with asthma symptoms, asthma medication use and, daily hospital admissions for respiratory conditions (Johnston et al. 2002, Johnston et al. 2006, Johnston et al. 2007, Hanigan et al. 2008, Crabbe 2012). PM_{10} concentrations were not consistently associated with hospital admissions for cardiovascular diseases. In Victoria, ambient PM_{10} concentrations were associated with daily hospital emergency department presentations for respiratory conditions during the severe bushfire season of 2002-2003 (Tham et al. 2009). Although those studies were conducted during periods of bushfire activity, health outcomes were not related specifically to individual bushfire events. Nevertheless, the results are consistent with other Australian evidence that exposure to PM from bushfires is associated with adverse respiratory, but not cardiovascular, outcomes.

Table 5.7.1 Australian evidence that PM emissions from bushfires have adverse health impacts

Study	Population	Exposure metric	Health outcomes
Effect of bushfires on the ambient PM_{10}-health outcome association			
(Morgan et al. 2010)	Sydney	Ambient PM_{10} >99th percentile, accompanied by bushfire event (1994-2002)	Associations between PM_{10} and daily hospital admissions for respiratory disease, COPD (> 65 years) and asthma (15-64 years) were stronger on bushfire smoke days. Bushfire smoke days had no effect on associations between PM_{10} and mortality or cardiovascular hospital admissions.
(Chen et al. 2006)	Brisbane	Bushfires >1 hectare (1997-2000)	Association between PM_{10} and daily hospital admissions for respiratory disease marginally stronger on bushfire days.
Effect of high-PM bushfire days on health outcomes			
(Johnston et al. 2014)	Sydney	Ambient PM_{10} or $PM_{2.5}$ >99th percentile, accompanied by bushfire event (1996-2007)	Bushfire smoke days associated with increased hospital emergency department presentations: +3% (non-trauma) +7% (respiratory) +23% (asthma) +12% (COPD). Inconsistent effects on cardiovascular presentations. No associations with children's emergency department presentations.
(Martin et al. 2013)	Sydney, Newcastle and Wollongong	Ambient PM_{10} or $PM_{2.5}$ >99th percentile, accompanied by bushfire event (1994-2007)	Bushfire smoke days associated with increased hospital admissions in Sydney: +6% (respiratory) +13% (COPD) +12% (asthma). Non-significant increases in hospital admissions in Newcastle and Wollongong. No associations with cardiovascular admissions.
(Johnston et al. 2011a)	Sydney	Ambient PM_{10} >99th percentile, accompanied by bushfire event (1994-2007)	Bushfire smoke days associated with a 5% increase in non-accidental mortality.
Effect of the Sydney, January 1994, bushfires on health outcomes			
(Jalaludin et al. 2000)	32 children with history of wheeze	Ambient PM_{10} during bushfires	Daily PM_{10} during bushfires was not associated with poor lung function in children.
(Smith et al. 1996)	Western Sydney	Total suspended particles in ambient air during bushfires	Daily total suspended particles during bushfires were not associated with hospital emergency department presentations for asthma.

5.7.3 International evidence of health effects

A review of the health impacts of bushfire smoke concluded that the majority of epidemiological studies (61 in total) have found that exposure is associated with an increased risk of respiratory morbidity (Liu et al. 2015). Respiratory diseases were also the health outcome most studied (in over

90% of studies). There was insufficient evidence to conclude that bushfire smoke is consistently associated with cardiovascular morbidity. Almost all studies reported that ambient levels of PM increased dramatically during bushfire events and, most studies used ambient PM concentration to indicate smoke exposure. The review concluded that children, the elderly, and those with underlying chronic diseases, appear to be most susceptible to the health effects of bushfire smoke.

Few studies have assessed associations between exposure to bushfire smoke and all-cause, non-accidental mortality. The relatively brief period of exposure during bushfire events makes it difficult to determine whether any difference in the number of deaths during those times is significantly different from the usual day-to-day variations in deaths. A study from Athens, Greece found an association between bushfire events during 1998-2004 and non-accidental mortality (Analitis et al. 2012). Sub-analysis of the data attributed nearly all of the increase in mortality to respiratory-related deaths. Another study found that in a Malaysian valley that was exposed to the smoke of Indonesian forest fires during 2000-2007, days of forest fire smoke haze (defined as PM_{10} >100 $\mu g/m^3$) were associated with a 19% increase in respiratory, but not total non-accidental, mortality (Sahani et al. 2014). Studies in Finland, the US and Singapore have similarly failed to show that bushfire smoke significantly increases total daily deaths (Emmanuel 2000, Vedal and Dutton 2006, Hanninen et al. 2009). It has been suggested that the global annual mortality from landscape fire smoke is over 300,000, with the majority in Africa and South-East Asia (Johnston et al. 2012), however there is little epidemiological evidence that non-occupational exposure to bushfire smoke significantly increases mortality.

During bushfires, increased hospitalisations and emergency department visits for respiratory diseases (asthma, COPD, upper respiratory tract illnesses, respiratory infections, rhinitis) have been observed in all bushfire-prone regions of the world where health-effects investigations have been conducted (Dennekamp and Abramson 2011, Henderson and Johnston 2012, De Sario et al. 2013). Many of the studies reported positive associations between ambient PM concentrations during bushfire events and adverse respiratory health outcomes.

In Singapore, during the 1997 Indonesian forest fires, a 100 $\mu g/m^3$ increase in PM_{10} was associated with increases in: upper respiratory tract illnesses (12%), asthma (19%) and, rhinitis (26%) (Emmanuel 2000). Although the associations were reported for PM_{10}, 94% of particles in the smoke haze were $PM_{2.5}$. Increases in respiratory symptoms and decreases in lung function during bushfires have been observed in both healthy subjects and, in asthmatic and COPD patients (De Sario et al. 2013). In Canada, $PM_{2.5}$ emissions from wildfires were positively associated with the dispensing of medication used to relieve the symptoms of asthma and COPD (Elliott et al. 2013). Also in Canada, PM_{10} principally (but not exclusively) from wildfires during a severe fire season in 2003, was associated with increased respiratory hospital admissions, but not cardiovascular admissions (Henderson et al. 2011). A 10 $\mu g/m^3$ increase in PM_{10} during the 2003 fire season was associated with a 6% increase in physician visits for asthma (Henderson et al. 2011). Similarly, during catastrophic fires in southern California in 2003, ambient $PM_{2.5}$ was associated with increased respiratory hospital admissions, especially asthma (an increase in $PM_{2.5}$ during fires of 70 $\mu g/m^3$ was associated with a 34% increase in asthma admissions), but not cardiovascular admissions (Delfino et al. 2009). In the Californian study, wildfire-related $PM_{2.5}$-respiratory admission associations were strongest in people over 64 years of age or less than 5 years of age. In Denver, Colorado, spikes in $PM_{2.5}$ and PM_{10} due to wildfires were associated with increased respiratory symptoms among 21

Denver residents with COPD, although it could not be determined whether these people were especially susceptible to the effects of wildfire PM as no comparison was made with people without COPD (Sutherland et al. 2005). In general, the evidence suggests that people vulnerable to the health effects of air pollution, whether through age or chronic respiratory disease, are likely to be especially affected by bushfire-related PM emissions.

Of interest, an analysis of the respiratory symptoms of non-asthmatic teenagers during the 2003 Californian fires showed that symptoms were greater in those individuals with smaller lung airways (Mirabelli et al. 2009). Relatively small lung airways are considered a marker for susceptibility to future compromised respiratory function. Thus the adverse health effects associated with fire smoke may not necessarily be restricted to individuals with pre-existing respiratory diseases.

Wood smoke contains toxic pollutants such as PAHs that have shown to be carcinogenic in other exposure settings (*e.g.* occupational) (Armstrong et al. 2004). At sufficient concentration and duration of exposure these toxins may increase cancer risk. However, the long-term cancer risk from brief exposures to bushfire smoke is unknown. Short-term, although extreme, elevated exposures to carcinogens in bushfire smoke are likely to result in small total exposures relative to total, lifetime exposures to carcinogens from other combustion sources such as diesel. Epidemiological studies have shown that urban firefighters exposed to smoke over an entire working lifetime have about a three-fold increased relative risk of developing lung cancer (Hansen 1990). Exposures by urban firefighters will include smoke from the combustion of many different types of fuel. However, based on this estimation we can infer that the cancer risk due to environmental exposure to bushfire smoke is likely to be very low.

Being pregnant during wildfire events has been associated with lower birth weights (Holstius et al. 2012). However evidence for this, and evidence relating bushfire smoke to other health outcomes not already addressed in this *section*, is very limited (Liu et al. 2015).

There is inconsistent evidence about the relative toxicity of PM from bushfires compared to urban ambient PM. PM collected from bushfires has been shown to elicit a greater inflammatory response in mouse lungs (Wegesser et al. 2009) but a lesser inflammatory response in human lung airway cells (Nakayama Wong et al. 2011) compared to ambient PM collected in the absence of bushfires. Coarse PM from landscape fires was shown to produce a greater inflammatory response in mouse lungs than fine or ultrafine PM from landscape fires (Wegesser et al. 2010, Kim et al. 2014). However ultrafine PM from landscape fires elicited greater cardiac responses in mice (Kim et al. 2014). Bushfire particulates elicited a greater oxidative stress response in human lung cells compared to ambient PM collected in the absence of bushfires (Nakayama Wong et al. 2011). An oxidative stress response is observed in mouse lungs within 1-hour of instillation of coarse PM from bushfires (Williams et al. 2013). There does not appear to be any correlation between the PAH content of bushfire PM and its toxicity (Wegesser et al. 2010). There is some evidence that heat-labile organic compounds in bushfire PM are the toxic component of bushfire particulates and that most, if not all, of the toxic response is the result of oxidative stress (Franzi et al. 2011).

As bushfires commonly occur on days of extreme heat and very high temperatures have health impacts (Bouchama 2004), most studies of the health effects of bushfire smoke have adjusted for the effects of high air temperature. It is possible that the health impacts of PM may be greater on high temperature days and that this interactive effect makes bushfire-related PM appear more

harmful (Shaposhnikov et al. 2014). However, the observation that exposure to bushfire smoke is more strongly associated with respiratory health effects than cardiovascular health effects suggests that the health outcomes observed during bushfire events are not primarily a response to heat stress. If heat stress were responsible for the observed outcomes, cardiovascular outcomes would be expected to be as prominent (if not more so) as respiratory health effects (Basu 2009).

5.7.4 Summary

Epidemiological studies, including considerable Australian evidence, consistently show that the very high ambient PM emissions during bushfires are associated with increases in respiratory morbidity, especially increases in symptoms related to asthma and COPD. The evidence relating bushfire PM with mortality or cardiovascular morbidity is inconsistent. However, the number of studies investigating these health outcomes is small and further investigations are required before a conclusion can be drawn.

It is not possible at this time to deduce whether increased respiratory symptoms are a consequence of the very high ambient PM levels that occur during bushfires and/or, whether PM from bushfires are specifically more toxic to lungs than PM from other sources. There is limited evidence from animal studies to indicate that PM from bushfires may be more toxic to lungs than other PM. Induction of an oxidative stress response appears to be the principal *modus operandi* of bushfire PM toxicity.

- There is strong evidence from Australia (and elsewhere) that exposure to PM from bushfires is responsible for an increase in respiratory morbidity.
- Hospital admissions and presentations to hospital emergency departments for asthma and COPD are increased at times of high ambient PM levels during bushfire events.
- There is inconsistent evidence that exposure to bushfire PM is associated with mortality or cardiovascular morbidity.
- Bushfires have been responsible for some of the highest levels of ambient PM reported in Australia.
- There is limited evidence that PM emitted during bushfires is more toxic than PM from other sources.

5.8 Crustal dust

Crustal dust is a product of wind erosion. While wind erosion may occur anywhere, it is most prevalent in arid and semi-arid climates. The global source areas for wind erosion and crustal dust generation are the deserts and semi-arid plains of Asia, Africa, North America and Australia. Areas within source regions that provide ideal conditions for dust generation include dry lake beds, open deserts, fallow farm fields and other disturbed lands, unpaved roads, quarries and, building sites (Van Pelt and Zobeck 2007). All crustal dusts are not equal in their compositional characteristics, with composition heavily dependent on the aforementioned source areas and particle transformation during atmospheric transport (Chow et al. 2003, Huang et al. 2010). The mineral and chemical composition of crustal dust particles also depends on the geographic location. Sulphates and nitrates derived from alkaline soils are characteristic of Asian dusts (Ichinose et al. 2008), Saharan dusts are composed principally of oxides and carbonates (Vanderstraeten et al. 2008), whereas Australian dusts are low in carbon and high in crustal metals, particularly iron (Radhi et al. 2011, Gunawardena et al. 2013). It is possible that compositional differences of dust derived from different land surfaces will result in different toxicological profiles for the dust particles. The relative health impacts of different crustal dust particles is unknown as comparative investigations of the health effects of dust derived from different locations have not been conducted.

Primarily because they are the largest source of airborne crustal dust, nearly all health effect investigations of crustal dust relate to dust storm particles (sometimes referred to as desert storms). The particles within dust storms are undoubtedly a mixture of PM from various sources (including anthropogenic) however the largest sources are uncultivated arid and semi-arid lands and, fallow agricultural fields. The amount of dust storm activity is dependent upon anthropogenic modification of land surfaces, natural climate variability and, changes in climate brought about by global warming. Denuding of land surfaces through deforestation for agriculture and the removal of surface water for human use has undoubtedly increased dust emissions. Increasing aridity through anthropogenic activities and global warming may increase overall dust emissions in the future (United Nations Environment Programme 2006). However future dust storm activity is uncertain (Goudie 2009).

Turbulent winds raise large quantities of dust from arid lands. The dust can be transported thousands of kilometres. Dust storms originating in the deserts of Mongolia, northern China, and Kazakhstan seasonally affect much of eastern Asia (Watanabe et al. 2011), with the dust traversing the Pacific Ocean to North America (Han et al. 2008) and, some dust particles even transported one full circuit around the globe (Uno et al. 2009). The further dust travels, the more the dust cloud is dispersed and the less likely that health impacts of exposure will be evident. Nevertheless, the impact of dust storms can be observed many hundreds of kilometres downwind from the emission source. Human health effects are not only observed in arid, low populated areas, but also in major urban centres such as Athens, Madrid, Beijing, Shanghai, Seoul, Taipei, Tokyo, Sydney, Brisbane and Melbourne (Goudie 2014). These cities are downwind and sufficiently close to large sources of crustal dust (Sahara Desert in North Africa, central Asian deserts, deserts of north-west and central Australia) that are impacted from time-to-time by strong winds. These winds can result in ambient PM loadings in the urban centres that far exceed levels that impact health.

Although a large proportion of particles in dust storms may be very large particles, PM_{10} are present in significant quantities (O'Hara et al. 2006). Once aloft, larger particles settle out over relatively

short distances in a continuous fractionation process whereas smaller particles (typically <10 µm) travel great distances in the atmosphere, 100-3,000 metres above the Earth's surface (Toepfer et al. 2012). Dust storms, like bushfires, are events that can result in extreme levels of ambient PM not normally encountered in either urban or non-urban settings. An unusually large dust storm in September 2009 resulted in a maximum PM_{10} concentration in Sydney that was greater than 11,000 µg/m^3 (Merrifield et al. 2013). More commonly, PM_{10} maximums during dust storms are in the order of 200-3000 µg/m^3 (Goudie 2014), still many times greater than usual ambient concentrations. Crustal dust is commonly described as 'coarse' but this description is not accurate. $PM_{2.5}$ particles are also present in significant quantities in dust storms. High concentrations of $PM_{2.5}$ during dust storms (42-1638 µg/m^3) have been observed in cities in Southern Europe, Eastern Asia, Eastern Australia and elsewhere (Goudie 2014). The range of particle sizes in a dust storm mean that inhaled dust has the potential to elicit a variety of health effects based on site of deposition. Coarse particles are more likely to be deposited in the upper airways and the larger airways of the lower respiratory tract and thereby cause upper and lower respiratory symptoms and illnesses (Sandstrom and Forsberg 2008). Particles smaller than 2.5 µm penetrate into the gas exchanging region of the lungs and ultrafine particles (<0.1 µm) may pass into the bloodstream and even into individual cells, with consequences for cardiovascular health (Martinelli et al. 2013).

Generally the duration of elevated ambient PM from dust storms at any one location is less than a day. Acute, but extreme, exposure to dust particles is likely to affect individuals who are most susceptible such as the elderly, those with pre-existing cardio-pulmonary disease and, the very young.

Bacteria, fungi, pollen spores and, influenza virus are associated with crustal dusts in Australia and elsewhere (Polymenakou et al. 2008, Chen et al. 2010a, Lim et al. 2011, Toepfer et al. 2012, Goudie 2014). Mineral particles offer a measure of protection to microbes during travel and there is some evidence that attachment of viruses to dust particles enhances virus survival, and may enhance long-range host-to-host transport (Chen et al. 2010a). A large variety of bacterial and fungal pathogens have been isolated from desert dust and it has been proposed that this biological component of dust could be important in the distribution of microorganisms and associated diseases such as human respiratory infections (Griffin 2007). There is some evidence that the microbial component of particles may contribute to the lung inflammatory potential of Asian dust storms (Ichinose et al. 2008). A variety of other trace elements and compounds found in crustal dust could also impact health (*Table 5.8.1*).

Some of the components in *Table 5.8.1* can be of anthropogenic origin. If dust storms move across industrialised, agricultural or polluted land areas they can pick up and transport anthropogenic material (Van Pelt and Zobeck 2007, Kim et al. 2012c). The health risks associated with the minute traces of toxic material in crustal dust are unknown. Specific bacterial and fungal infections in humans have been temporally associated with dust storms: coccidiomycosis (a fungal infection) in the US, meningococcal meningitis in Africa and, conjunctivitis in Africa and Asia (Yang 2006, Goudie 2014, Sprigg et al. 2014). With the exception of coccidiomycosis, where the fungal pathogen is in the air during dust events, it is unclear whether infections associated with dust storms are a result of pathogens travelling on dust particles or, whether dust particles physically damage the nasal mucosa, thereby easing the invasion of pathogens from other sources. The vast majority of evidence

of health effects associated with dust storms relate to respiratory and cardiovascular disease. This evidence is reviewed here.

Table 5.8.1 Trace components of crustal dusts that may affect health

Biological pathogens
bacteria and endotoxins (bacterial components that give an inflammatory response)
fungi
pollen
viruses
protein
lipid
Trace elements
metals (*e.g.* lead, mercury, arsenic, cadmium, iron)
radioactive elements
Trace compounds
silicates (*e.g.* asbestos and other fibrous compounds)
alkali salts
PAHs
dioxins
pesticides and herbicides

Sources: (Cook et al. 2005, Erel et al. 2006, Polymenakou et al. 2008, Sandstrom and Forsberg 2008, Leski et al. 2011, Goudie 2014)

5.8.1 Nature of the contribution of crustal dust to PM in NSW

The 2008 NSW Air Emissions Inventory reported that crustal dust made a small contribution to PM emissions in the GMR (3.6% and 2.0% of total PM_{10} and $PM_{2.5}$, respectively) (NSW EPA 2012a). These contributions are low, however the significance of dust storms to PM exposure in NSW is that for these brief periods the ambient concentration of PM can be the highest that the population is ever exposed to.

The deserts and semi-arid lands of central Australia are the largest source of crustal aerosols in the Southern Hemisphere (Radhi et al. 2011). Australian dust storms require a specific sequence of events to occur: intense flood events followed by prolonged drought conditions, and then the onset of strong winds (generally around September-November) leading to significant erosion of alluvial dust (Jayaratne et al. 2011). Such a sequence of events occurs in central Australia from time-to-time and with prevailing westerly winds the suspended dust is transported to the east coast of Australia. This chain of events occurred on 23 September 2009, when the measured 24-hour mean level of PM_{10} and $PM_{2.5}$ in Sydney were over 11,000 µg/m^{3}and 1,600 µg/m^3, respectively (Merrifield et al. 2013). A second day of extreme PM concentrations occurred three days later (24-hour mean PM_{10} of 783 µg/m^3, 24-hour mean $PM_{2.5}$ of 110 µg/m^3). Chemical analysis of dust particles in Sydney indicated that the dust came from both desert and agricultural lands (Aryal et al. 2012). This dust storm was estimated to be the most severe in Australia for 70 years (Jayaratne et al. 2011). Dust storms that lead to elevated particulate pollution across much of NSW are rare events. From 1994

to 2007 there have been six dust storm events that have resulted in daily PM_{10} levels in Sydney exceeding the 99th percentile of the distribution of 24-hour mean PM_{10} (Johnston et al. 2011a). During these six dust storms, the 24-hour mean PM_{10} in Sydney ranged from, approximately 50 µg/m^3 to 200 µg/m^3, levels that are considered detrimental to health.

Dust storms and bushfires result in the highest ambient PM levels in NSW (Department of Sustainability 2010). While a dust storm may last for several days, as it moves with the wind the concentration of ambient PM will only remain elevated for a matter of hours at any one location. For example, a dust storm in October 2002 that resulted in a maximum concentration of PM_{10} in Sydney of 266 µg/m^3 only elevated PM_{10} levels for a period of 6 hours (Chan et al. 2005). The storm had a larger impact in Brisbane, recording a maximum PM_{10} concentration of 841 µg/m^3 and elevated PM_{10} levels for 16 hours.

Severe dust storms in NSW are brief, intense and large. The dust plumes associated with the 2002 and 2009 dust storms were more than 2,000 kilometres long (McTainsh et al. 2005, Jayaratne et al. 2011) and the storm traversed NSW from west to east. The vast majority of the population of NSW at that time is likely to have had some exposure to these dust storms.

5.8.2 Australian evidence of health effects

The episodic nature of large dust storms in Australia makes the study of associated health effects challenging. Nevertheless, during a large dust storm many people are likely to inhale a higher than usual concentration of PM, either from being outdoors at the time of the dust plume passing or from outdoor air entering indoor spaces.

Analysis of daily mortality data indicated that six dust storms from 1994 to 2007, which increased daily ambient PM_{10} in Sydney to above the 99th percentile (corresponding closely with the NEPM 24-hour mean PM_{10} standard of 50 µg/m^3), were associated with a 16% increase in non-accidental mortality three days after the dust events (Johnston et al. 2011a). Two of the six dust events also coincided with days of bushfire smoke however the study found that bushfire smoke days had a minimal effect on mortality. The statistically significant increases in mortality with so few dust events provide strong evidence of the effect on mortality of dust storms. The number of deaths associated with these six dust storm events was too small to identify significant increases in specific causes of mortality. However, as more than half of the non-accidental mortality recorded in Sydney 1994-2007 was either cardiovascular or respiratory in nature, these conditions featured among deaths associated with dust storm events.

The 2009 Australian dust storm offered the opportunity to examine the health impacts of the largest dust storm in Australia in recent times. The storm was associated with a 4% increase in presentations to hospital emergency departments across Sydney (Merrifield et al. 2013). There was a 20% increase in respiratory presentations, a 23% increase in asthma presentations and, no significant change in cardiovascular presentations. Children and the elderly particularly featured in emergency department presentations at the time of the dust storm. Hospital admissions for asthma increased by 14% but there was no increase in respiratory or cardiovascular admissions (Merrifield et al. 2013). A single hospital in Brisbane reported a 39% increase in emergency department

admissions the day after the 2009 dust storm, but no increases in emergency admissions two days after the storm (Barnett et al. 2012).

Another Brisbane study examined the daily symptoms, medication usage and lung function of 76 people with asthma during 11 dust events occurring during 1992-1994. Dust events were identified by a measure of the colour of total suspended particles, visibility data and, particle sizing of rural and urban dust particles (Rutherford et al. 1999). Some of the days defined as dust event days in this study had relatively moderate increases in ambient PM_{10} concentrations (<40 $\mu g/m^3$). The health impact of each dust event was assessed individually. A number of the dust events were significantly associated with increases in asthma symptoms, but the relationship was not consistent across all dust events. The dust events that had a greater impact on people with asthma tended to have the highest ambient particulate levels and, a higher proportion of total suspended particles being PM_{10}.

These mortality and hospital data point to significant dust storm events in Australia impacting on health. Further studies are required to determine which sub-groups of the population are most vulnerable to the effects of dust storms and which health effects are most strongly associated with exposure to crustal dust.

5.8.3 International evidence of health effects

The number of studies addressing the health effects associated with exposure to desert dust have significantly increased (from a low base) in the past decade, with the majority of studies examining the health of Asian (major dust source central Asian arid lands) and Southern European (major dust source the Sahara desert of North Africa) populations (de Longueville et al. 2013). These populations have been a focus because seasonal winds regularly deposit large quantities of desert dust in Eastern Asian and Southern European cities. In addition to studies of dust storm events, several source apportionment studies have investigated the health effects of PM from crustal sources that are not specifically linked to dust storms. The results of these studies are also summarised here.

Dust storm events have been associated with increases in all-cause mortality, although this is not a consistent observation (de Longueville et al. 2013). Asian dust storms have been associated with increases in all-cause daily mortality in Taipei and Korean metropolitan centres (Chan and Ng 2011, Lee et al. 2013). However other studies in Eastern Asian cities have found no such association (Kwon 2002, Lee et al. 2014). Associations between dust storm events and increases in daily mortality are more consistently seen in the elderly (Kwon 2002, Chan and Ng 2011, Lee et al. 2013).

Observed associations between ambient PM concentrations and mortality during dust storms have been inconsistent. In Seoul, South Korea, ambient $PM_{2.5}$ levels during dust storm events (when $PM_{2.5}$ was >65 $\mu g/m^3$) were significantly associated with daily mortality but no association existed for non-dust storm days (Kim et al. 2012a). Similarly in Barcelona, Spain, concentrations of coarse PM ($PM_{10-2.5}$) (but not $PM_{2.5}$) were associated with daily mortality on Saharan dust days but not on non-dust days (Perez et al. 2008). Another study from Spain found that on Saharan dust days, PM_{10} (but not $PM_{2.5}$ or coarse PM) was associated with mortality, whereas on non-dust days, $PM_{2.5}$ (but not PM_{10} or coarse PM) was associated with mortality (Jimenez et al. 2010). In contrast, studies from Seoul and Athens, Greece, have found that PM_{10} was more strongly associated with mortality on days without

dust storm events (Lee et al. 2007, Samoli et al. 2011). A review of the impact of Saharan dust storms on mortality in European cities concluded that concentrations of $PM_{2.5}$ during dust events does not impact on mortality, and that the evidence related to coarse PM is inconsistent (Karanasiou et al. 2012). Most studies have found that PM_{10} or coarse PM is more strongly associated with mortality during desert dust episodes than at other times (WHO 2013c).

A source apportionment study, using data from the Harvard Six Cities Study in the US, found $PM_{2.5}$ from crustal sources was not associated with daily mortality, whereas $PM_{2.5}$ from mobile and coal combustion sources were associated with mortality (Laden et al. 2000). This suggests that crustal $PM_{2.5}$ may have less of an impact on mortality than $PM_{2.5}$ from these other sources.

Most investigations of the association between dust storm events (Saharan and Asian) and cardiovascular disease mortality suggest that exposure to high levels of crustal dust increases cardiovascular mortality (de Longueville et al. 2013). Asian dust storm events have been associated with increases in daily cardiovascular mortality in some (Chan and Ng 2011, Lee et al. 2013), but not all studies (Chen et al. 2004, Lee et al. 2014). In Italy, the association between coarse PM and cardiovascular mortality was much stronger on dust days than dust-free days (Mallone et al. 2011). Furthermore, in source apportionment studies conducted in Washington DC, US, and Santiago, Chile, PM apportioned to crustal dust has been associated with cardiovascular mortality (US EPA 2009).

Asian dust storms have generally not been associated with statistically significant increases in daily mortality from respiratory diseases (Kwon 2002, Chen et al. 2004, Chan and Ng 2011, Lee et al. 2013). In Italy however, Saharan dust storms have been associated with significantly increased daily respiratory mortality in the elderly (≥ 75 years) (Zauli Sajani et al. 2011).

With respect to morbidity effects, there is less evidence associating exposure to crustal dust with cardiovascular disease than there is for respiratory disease. Associations between increased ambient PM concentrations during Asian dust storms and daily hospital admissions for cardiovascular diseases are inconsistent (de Longueville et al. 2013). Neither admissions for stroke (Yang et al. 2005, Kang et al. 2013), ischaemic heart disease (Bell et al. 2008, Tam et al. 2012b) nor, congestive heart failure (Yang et al. 2009) have been strongly associated with Asian dust storm events. The US EPA has also noted that dust storm events resulting in high concentrations of airborne crustal particulates have inconsistently been linked to increases in cardiovascular disease hospitalisations and emergency department presentations (US EPA 2009). It was observed that over a 10-year period in Cyprus, high PM_{10} dust storm days were significantly associated with all-cause, but not cardiovascular, hospital admissions (Middleton et al. 2008).

Notwithstanding the above, there is some evidence from source apportionment studies that PM from crustal sources (both $PM_{2.5}$ and coarse PM) are associated with cardiovascular health effects (Stanek et al. 2011). PM apportioned to crustal dust has been associated with cardiovascular hospital admissions in Copenhagen and, heart rhythm changes in Helsinki, Los Angeles and Boston (US EPA 2009).

There is considerable evidence linking exposure to crustal dust with respiratory illness. Asian dust storms have been associated with exacerbations of asthma requiring medical treatment, in both adults (Park et al. 2005, Bell et al. 2008, Lee and Lee 2014, Wang et al. 2014a) and children (Yoo et al. 2008, Kanatani et al. 2010). Pollen may augment the effect of Asian desert dust on asthma

symptoms in adults, but it also appears that dust particles alone can aggravate asthma symptoms (Watanabe et al. 2011). In Taipei, dust storms have been associated with increases of 3-5% in medical centre visits for respiratory conditions in children (Chien et al. 2012). Exposure to PM_{10} during dust storms has been associated with an increase in hospitalisations for COPD in Hong Kong (Tam et al. 2012a), Taipei (Chiu et al. 2008) and Israel (Vodonos et al. 2014). Source apportionment studies have associated PM from crustal sources with increased respiratory symptoms in children with asthma and, decreased lung function in adults with asthma (US EPA 2009). Overall, crustal dust exposure is more consistently associated with hospitalisations for asthma and COPD than it is for all respiratory hospitalisations (de Longueville et al. 2013).

The incidence of pneumonia, an inflammatory condition of the lung, has been associated with populations exposed to dust storms in the US, Russia and the Middle East (Griffin 2007). Pneumonia is predominantly caused by viral or bacterial infections. It is not known how dust storms may increase the rate of pneumonia but a potential immune-modulatory effect or less likely viruses and/or bacteria on desert dust could be responsible for the associations observed between dust storms and pneumonia. Increased ambient PM_{10} during Asian dust storm events has been associated with increased hospital admissions for pneumonia in one study (Cheng et al. 2008) but not others (Bell et al. 2008, Tam et al. 2012a). The positive association was observed in Taipei, where a 4-5% increase in daily hospital admissions for pneumonia was seen 1-2 days after high PM_{10} dust events (Cheng et al. 2008). It may be that viruses or bacteria on desert dust particles were responsible for the development of pneumonia symptoms within 1-2 days of exposure, but it is also possible that inhalation of dust particles suppressed the normal immune response to allow commensal bacteria to cause disease.

It is possible that health effects result from exposure to specific components of crustal dusts, however uncertainties in exposure assessment usually fail to convincingly attribute health effects to specific constituents of crustal dust. Iron-specific dusts (relevant to Australian conditions) have been linked with respiratory symptoms and lung cancer mortality in occupational settings (Cook et al. 2005). There have been reports of dusts from salt lakes (also relevant to Australia as large dust storms in eastern Australia have originated from the region of Lake Eyre) being associated with cough, wheeze and, eye and nose irritation (Cook et al. 2005). Inhaled material from soils of central Europe, Finland, Turkey, and Canada, identified as containing a number of fibrous minerals, have been linked to asymptomatic lung lesions (plural plaques) in neighbouring agricultural communities (Cook et al. 2005). A study of school children in Seoul found that during dust storms, ambient concentrations of PM were not significantly associated with lung function however the concentration of metals bound to particles was significantly associated with a decrease in lung function (Hong et al. 2010). There is no other evidence of disease being caused by inhalation of specific minerals found in crustal dust, except where exposure to the toxic component has been concentrated because of anthropogenic activity (*e.g*. asbestos mining).

A few toxicological studies have investigated the effect of crustal dust exposure on immune responses and markers of inflammation. Exposure of hypertensive rats for 4.5-6 hours to concentrated ambient particles collected from an Asian dust storm, resulted in markers of inflammation being increased in the blood and lungs in a dose-response manner (Lei et al. 2004). Instillations of PM_{10} collected from a Middle Eastern desert into the lungs of normotensive rats produced only mild inflammatory responses (Wilfong et al. 2011). Desert dusts from the US have

been shown to induce an oxidative stress response and the release of inflammatory molecules in respiratory cells and, provoke inflammatory injury in the lower respiratory tract of animals, responses that were comparable to other PM (Ghio et al. 2014). Dust particles are sensed by cells lining respiratory airways, which initiates specific immune responses (Esmaeil et al. 2014). It has been demonstrated that Asian dust storm and non-dust storm $PM_{2.5}$ collected from the same area cause comparable dose-response DNA damage in animal lungs and human immune cells (Wei and Meng 2006, Meng and Zhang 2007).

Together, these toxicological studies suggest that crustal dust particles are not benign, and have similar toxic properties to other ambient PM.

5.8.4 Summary

The health impacts of exposure to crustal dust currently remain under-evaluated and under-reported. The unpredictable nature of dust storms means that health investigations are usually conducted retrospectively. This is a limitation as measurements of potential confounders and health outcomes can only be obtained from routinely collected health data.

Severe dust storms impact on the health of vulnerable populations. That is, people with existing chronic disease, the elderly, and the very young. Evidence from Australia and elsewhere shows associations between dust storm events and increased mortality. Although dust storms are associated with mortality, the effect of dust storms on ambient PM-mortality associations is variable. Cardiovascular mortality is more consistently associated with extreme dust events than is respiratory mortality, although the evidence base is small.

In contrast to mortality, respiratory morbidity (especially asthma and COPD) is more strongly associated with high ambient PM concentrations during dust storm events than is cardiovascular morbidity.

Ambient concentrations of PM during severe dust storms can be extremely high for a relatively brief period of time, and the health effects observed in vulnerable populations during these events likely reflect these acute exposures to extreme concentrations of ambient PM. It is not clear whether exposure to lower levels of crustal dust elicits any health effects, although source apportionment studies suggest that exposure to crustal dust, other than during dust storms, is associated with cardiovascular and respiratory health effects.

The toxicity of crustal dust PM has not been extensively investigated. Crustal dust particulates exhibit similar toxic properties to PM from other sources. There are many components of crustal dust (*e.g*. microbes, metals) with the potential to cause health effects however it is not known whether specific components of crustal dust exert specific health effects.

- The concentration of PM during severe dust storms are some of the highest levels of ambient PM observed in Australia.
- A significant proportion of particles in dust storms are <2.5 µm and both fine and coarse crustal dust PM have been associated with adverse health effects.
- Severe dust storms are associated with increased daily mortality in Australia and elsewhere, however dust storms have inconsistent effects on PM exposure-mortality associations.
- Severe dust storms are associated with an increase in respiratory morbidity in Australia and elsewhere.
- Extreme PM levels during dust storms particularly impact the elderly, the very young and, people with chronic disease.
- Source apportionment studies suggest that exposure to crustal dust (other than during dust storms) is associated with cardiovascular and respiratory health effects.
- The few studies to have investigated the toxicity of crustal dust particles have shown that these particles exhibit similar toxic properties to PM from other sources.

5.9 Sea salt

The major source of sea salt PM air pollution is the breaking of waves on the ocean's surface and the bursting of seawater bubbles due to air entrapment (Foltescu et al. 2005).

Sea salt PM consists of both inorganic sea salt and organic matter. Sea salt particles are predominantly composed of the salt ions, chloride and sodium. However a number of other trace elements and compounds are also found in sea salt PM (*Table 5.9.1*).

Table 5.9.1 Composition of sea salt PM

Chemical Component	Approximate percentage by weight
Chlorine	55.1
Sodium	30.6
Sulphate	7.7
Magnesium	3.6
Calcium	1.2
Potassium	1.1
Bicarbonate	0.4
Bromide	0.2

Source: (US EPA 2004)

Sea salt particles are predominantly concentrated in the coarse mode, with a median diameter of around 7 µm, although particles can be smaller than 1 µm (US EPA 2004, European Environment Agency 2012).

Sea salt can significantly impact air quality in coastal areas (Tsyro et al. 2011). Sea salt particles play an important role in atmospheric chemistry. The chloride and bromide ions in sea salt particles can be displaced by nitric and sulphuric acids in the atmosphere (US EPA 2004, White 2008). As a result of these chemical reactions, the nitrogen and sulphur content of ambient PM can vary substantially between marine and inland environments (US EPA 2004). Some studies of sea salt PM distinguish between *fresh* sea salt particles and atmospherically *aged* sea salt particles (Hasheminassab et al. 2014, Pun et al. 2015). Aged sea salt is characterised by nitrates and sulphates and, negligible chloride ions (Hasheminassab et al. 2014). Atmospheric ageing alters sea salt PM properties, including increasing particle mass, as nitrate and sulphate are heavier than chloride ions (White 2008).

Sea salt is a major contributor to PM emissions globally however estimations of the total contribution vary widely (Foltescu et al. 2005, White 2008, US EPA 2009, Tsyro et al. 2011, Viana et al. 2014). Sea salt PM mass concentration has great temporal and spatial variability that is highly dependent on weather and climate (Foltescu et al. 2005). Sea salt particles contribute substantially to ambient PM_{10} and $PM_{2.5}$ in remote marine areas, and can also be a significant contributor to ambient PM over land masses (Foltescu et al. 2005, White 2008, Tsyro et al. 2011). Data indicate that in some coastal regions the contribution of sea salt to PM_{10} can be as much as 80% of the annual average particle mass (European Environment Agency 2012).

An ambient PM modelling study has reported that average concentrations of sea salt PM across 89 European land-based sites ranged from 0.3-13 $\mu g/m^3$ (Manders et al. 2010). Concentrations along the Atlantic and North Sea Coasts were around 5 $\mu g/m^3$ and at inland locations at a distance of about 300 kilometres from the coast, concentrations were approximately 2-5 $\mu g/m^3$.

The WHO report, *Health Risks of Particulate Matter from Long-range Transboundary Air Pollution*, estimated that the usual contribution of sea salt in European coastal areas was 2-4 $\mu g/m^3$ for PM_{10} and 0.2-0.8 $\mu g/m^3$ for $PM_{2.5}$ (WHO 2006b). Higher levels of sea salt PM_{10} (7-11 $\mu g/m^3$) were seen in some European coastal areas. Sea salt PM_{10} had the highest spatial variability. Generally, the contribution of sea salt to ambient PM_{10} mass ranged from 5-15% at coastal locations but was as high as 88% and 56% on the Portuguese and Irish coasts, respectively. A US study reported greater contributions of aged sea salt particles(2-27%), compared with fresh sea salt particles (1-13%), to total ambient $PM_{2.5}$ mass at eight sites in California (Hasheminassab et al. 2014).

5.9.1 Nature of the contribution of sea salt to PM in NSW

The 2008 NSW Air Emissions Inventory estimated that in the GMR, sea salt contributed 23% and 10.5% to total ambient PM_{10} and $PM_{2.5}$, respectively. In the Sydney region, sea salt contributed 15% and 3.8% to total ambient PM_{10} and $PM_{2.5}$, respectively. In the GMR, sea salt was the second largest source of PM behind coal mining while in the Sydney region, sea salt was the second largest source of PM behind solid fuel domestic heating.

The CSIRO *Upper Hunter Valley Particle Characterisation Study* estimated that in the Upper Hunter Valley towns of Singleton and Muswellbrook, (industry) aged sea salt was the second and third highest contributor to total $PM_{2.5}$, at 18% and 13%, respectively (Hibberd et al. 2013). Fresh sea salt was estimated to contribute 8% and 3% to Singleton and Muswellbrook $PM_{2.5}$, respectively. The contributions of the industry aged sea salt and fresh sea salt varied with the seasons and local weather. Aged sea salt had a larger contribution in summer and spring when there was a greater capacity for photochemical reactions during the warmer months. The contribution of aged sea salt and fresh sea salt to $PM_{2.5}$ in the Upper Hunter was found to be similar to contributions estimated for the western Sydney suburbs of Richmond (Cohen et al. 2012) and Liverpool (Cohen et al. 2011), with similar seasonal patterns occurring. Another source characterisation study collected $PM_{2.5}$ from seven sites: Liverpool, Mascot, Lucas Heights and Richmond, in or near Sydney; Muswellbrook and Mayfield, in the Hunter region; and Warrawong, in the Illawarra region (Crawford and Cohen 2013). Not surprisingly, the study found that the lowest estimated contributions of sea salt to ambient PM were at those sites furthest inland, Richmond (6.7%), Muswellbrook (6%) and Liverpool (8.7%). At the more coastal sites the contribution of sea salt to ambient PM ranged from 12.7% to 15.6%. Sea salt PM concentrations were also highest during summer, likely due to the prevailing on-shore sea breezes.

5.9.2 Australian evidence of health effects

This review found no Australian studies of the health effects associated with exposure to sea salt PM.

5.9.3 International evidence of health effects

In the past decade, a small number of source apportionment studies have been published that have included sea salt PM in the investigation of the health effects of source-specific PM. Positive associations with ambient concentrations of sea salt $PM_{2.5}$ have been reported for respiratory disease-related hospital admissions (Bell et al. 2014, Pun et al. 2015), cardiovascular mortality (Mar et al. 2006, Ito et al. 2011), and all-cause mortality (Mar et al. 2006). However, the majority of studies have not found significant associations between ambient concentrations of sea salt PM and adverse health effects, including: cardiovascular disease-related hospital admissions (Andersen et al. 2007, Bell et al. 2014, Kioumourtzoglou et al. 2014), respiratory admissions (Andersen et al. 2007), asthma admissions (Andersen et al. 2007), cardiovascular mortality (Ito et al. 2006, Ostro et al. 2011, Zhou et al. 2011), respiratory mortality (Zhou et al. 2011) and, all-cause mortality (Ito et al. 2006, Ostro et al. 2011, Zhou et al. 2011). These studies are discussed in further detail below.

Bell *et al.* reported that while sea salt $PM_{2.5}$ only contributed 1.7% to total ambient $PM_{2.5}$ in the north eastern US, it was associated with a small, but significant, increase in respiratory disease-related hospital admissions (Bell et al. 2014). In an earlier particle characterisation and health effects study, Mar *et al*. found consistently significant estimates of mortality effects for sea salt $PM_{2.5}$ for 5-day lagged data (Mar et al. 2006). However, the WHO's *REVIHAAP Report* suggests that this may be a chance finding given the long lag period (WHO 2013c).

One of the only studies to investigate the health effects of both source-apportioned $PM_{2.5}$ and PM_{10} was conducted in Barcelona (Ostro et al. 2011). The study identified eight different source factors, including aged sea salt, which was the second least contributor to total PM mass from all sources. The aged sea salt PM was found to contain not only chlorine and sodium, but also elements such as aluminium, calcium, potassium, manganese, iron and selenium. The study did not find any association between either sea salt $PM_{2.5}$ or PM_{10} and, either cardiovascular or all-cause mortality. However, the study reported significant associations between mortality and most of the other source-specific $PM_{2.5}$.

In a source apportionment study of $PM_{2.5}$ in Seattle and Detroit, sea salt $PM_{2.5}$ was not associated with all-cause, respiratory or cardiovascular mortality, in either city (Zhou et al. 2011). However, $PM_{2.5}$ apportioned to other, anthropogenic, sources were shown to be associated with mortality. Similar results were seen in a source apportionment study conducted in Washington DC (Ito et al. 2006).

A study of source apportioned PM_{10} and adverse health outcomes conducted in Copenhagen, Denmark found that sea salt PM_{10} had minimal effect (Andersen et al. 2007). The authors suggested that sea salt PM_{10} in Copenhagen was a proxy for clean air, with higher sea salt PM_{10} concentrations indicating lower pollutant concentrations, thus explaining the absence of health effects when sea salt PM_{10} concentrations were high.

Two studies of source apportionment of PM_{10} and morbidity conducted in Hong Kong have been published recently (Pun et al. 2014, Pun et al. 2015). Eight PM sources were identified in the studies: vehicular exhaust, soil/road dust, regional combustion, residual oil, fresh sea salt, aged sea salt, secondary nitrate and secondary sulphate (Pun et al. 2014, Pun et al. 2015). Fresh and aged sea salt contributed 3.7% and 12.8%, respectively, to the total ambient PM mass (Pun et al. 2014). Aged,

(but not fresh) sea salt PM_{10}, was significantly associated with a small increased risk of respiratory hospitalisations (Pun et al. 2015). However, vehicle exhaust, regional combustion, secondary sulphates and secondary nitrates PM_{10}, had larger effects on respiratory admissions than did aged sea salt. In models that incorporated two air pollutants (rather than a single pollutant), only vehicular exhaust PM_{10} and aged sea salt PM_{10} remained significantly associated with increased respiratory admissions.

A panel study of respiratory health in children with asthma living in New Haven, Connecticut, US, reported no associations between exposure to total, sea salt, sulphur, biomass combustion, or oil combustion $PM_{2.5}$ and adverse respiratory symptoms (Gent et al. 2009). Exposures to road dust $PM_{2.5}$ were significantly associated with increased wheeze, persistent cough, shortness of breath, and inhaler use, whilst vehicular $PM_{2.5}$ was associated with wheeze, shortness of breath and chest tightness.

A recent study in Boston, US, found no association between exposure to sea salt $PM_{2.5}$ and cardiovascular disease hospital admissions (Kioumourtzoglou et al. 2014).

Most of the studies reviewed here report that different PM sources exerted effects for different time lag structures, suggesting that there may be varying mechanisms of effect for PM from different sources (Mar et al. 2006). Further research is needed to confirm this.

Only four clinical studies related to sea salt PM have been reported in the literature. A Finnish study of non-smoking adults aged 50 years and over with coronary artery disease investigated source-specific $PM_{2.5}$ exposure and associations with a cardiac marker of ischaemic heart disease (Lanki et al. 2006). The study identified five main sources of $PM_{2.5}$ being local traffic, long-range transported PM, crustal dust, oil combustion and sea salt, with sea salt contributing only 0.9% of the total ambient $PM_{2.5}$ mass. Both long-range transported PM and traffic $PM_{2.5}$ were significantly associated with the cardiac biomarkers. Sea salt $PM_{2.5}$ had an association that was of borderline significance.

Another European study examined the association between $PM_{2.5}$ composition and beta-blocker use in people with cardiovascular disease and, heart rate variability (de Hartog et al. 2009). The study found no association between sea salt $PM_{2.5}$ and measures of heart rate variability. A second study of the same study cohort reported no association between a marker of lung epithelial injury and exposure to sea salt PM (Jacquemin et al. 2009).

Mills *et al*. conducted a randomised blind cross-over trial in healthy volunteers and cardiovascular disease patients and reported that exposures to concentrated $PM_{2.5}$ for 2 hours resulted in increased levels of a marker of inflammation in exhaled breath, and in cardiovascular patients, a small increase in circulating platelets (Mills et al. 2008). However, exposure to $PM_{2.5}$ did not result in any significant change in systemic inflammation markers or vascular function as measured by heart rate, blood pressure and forearm blood flow. Interestingly, the authors partly attributed the lack of effect to the fact that the vast majority of the concentrated $PM_{2.5}$ consisted of sea salt.

A toxicological study using PM samples collected from six European countries, studied the effects of fine and coarse PM on inflammatory and cytotoxic responses in macrophage cells (Jalava et al. 2008). Macrophages are one of a number of human cell types that help to clear particles from the lungs. The study found that exposure of macrophages to the sea salt component of the fine and

coarse particles, was positively correlated with the inflammatory and cytotoxic responses, however the correlations were not statistically significant. As part of the same study, the fine and coarse PM collected from the six European countries were instilled into the trachea of mice. Both the fine and coarse PM caused pulmonary inflammatory responses (Happo et al. 2010). While formal source apportionment was not conducted, the components of coarse PM most strongly correlated with the inflammatory response were tracers of soil (potassium, magnesium, copper, manganese and iron) and sea salt (sodium, chloride and nitrate).

5.9.4 Summary

Few studies have investigated the health effects of exposure to sea salt PM, with mixed results. Some source apportionment studies have demonstrated associations between sea salt PM and, mortality or respiratory morbidity outcomes however the majority of studies have found no association. Clinical and toxicological studies are yet to identify any clear mechanism by which inhalation of sea salt particles in particular may exert biological effects.

- A few source apportionment studies have demonstrated associations between ambient sea salt PM and, mortality or respiratory morbidity outcomes however the results of such investigations have been inconsistent.
- There is insufficient data to draw conclusions on the health effects specific to the inhalation of either aged or fresh sea salt PM.

5.10 Biogenic sources (PM derived from volatile organic compounds)

Ambient PM from biological sources include: pollens, mould spores, biological toxins, organic carbon of direct biological origin and, organic carbon resulting from reactions with volatile organic compounds (VOCs) (WHO 2006a). Most of these sources are outside the scope of this review. Here we have reviewed the evidence of health effects associated with exposure to PM derived from VOCs of biological origin.

VOCs from vegetation are one of the major natural contributors to air pollution, reacting with other compounds to form highly oxidised secondary organic aerosols (SOAs). SOAs occur when VOCs are oxidised to form organic carbon compounds with lower volatility which enables them to partition between the gaseous and particulate phases (Seinfeld and Pankow 2003, WHO 2006a, Hogrefe et al. 2011). These processes are heavily influenced by temperature and to a lesser extent humidity. SOAs are a major source of secondary PM, and hence important to air quality. However, there is currently insufficient data to recommend a guideline value for ambient organic carbon concentration, despite organic carbon contributing significantly to total ambient $PM_{2.5}$ mass (WHO 2013c). SOAs can also be derived from a variety of anthropogenic sources of VOCs including sources of combustion. Biogenic VOCs, particularly isoprene, also contribute to air quality through ozone production (WHO 2006a, Hogrefe et al. 2011).

On a global scale, vegetative emissions of VOCs are thought to be the major contributor to SOAs, although anthropogenic sources of VOCs can dominate in urban areas (Seinfeld and Pankow 2003, US EPA 2004, WHO 2006a, McNeill 2015). A number of different models have been used to estimate biogenic sources of SOAs (Hogrefe et al. 2011). However, chemistry modelling is currently unable to fully match observations of quantity, extent of oxidation or, spatial distribution, and mechanistic processes are not well understood (McNeill 2015). The US EPA identified that estimates of biogenic hydrocarbon emissions are uncertain because of heterogeneity in atmospheric reactions and the ageing of particles and, a lack of understanding of the processes related to partitioning of semi-volatile compounds between the gas and solid phase (US EPA 2004). Hogrefe *et al*. indicated that this uncertainty impacts on the estimates of ambient $PM_{2.5}$ from this source (Hogrefe et al. 2011). These sentiments are echoed by the *Committee on the Medical Effects of Air Pollutants* (COMEAP), which indicated that high resolution spatially modelled ambient $PM_{2.5}$ concentrations from non-anthropogenic sources, other than sea salt, are difficult to estimate (COMEAP 2010). Much progress has been made in determining the importance of biogenic and anthropogenic sources in SOA formation, however it is an area that is not well understood (US EPA 2004).

5.10.1 Nature of the contribution of biogenic PM to PM in NSW

This review did not identify any estimations of the contribution of biogenic VOCs to ambient PM concentrations in NSW.

5.10.2 Australian evidence of health effects of biogenic PM

This review did not identify any Australian studies of health effects associated with inhalation of PM derived from emissions of biogenic VOCs.

5.10.3 International evidence of health effects of biogenic PM

The health effects of PM derived from biogenic VOCs have not been investigated. The WHO has stated that the potential health effects of VOC-derived biogenic PM are "basically unknown" (WHO 2006a).

The health effects associated with exposure to gaseous and particulate products of terpenes (one type of biogenic VOCs) have been examined however terpenes are also used in household cleaning and other consumer products, and exposure studies have involved indoor exposures (Rohr 2013). Rohr states, "literature on the health effects of SOAs is relatively scarce, and research on the effects of biogenic SOA even scarcer" (Rohr 2013). Panel studies have examined the effects of exposure to tracers of SOAs however these entities can be derived from a multitude of sources besides biogenics. Rohr suggests that for future epidemiological studies, chemical markers of biogenic and anthropogenic SOAs could be used, although cost implications could be limiting. Toxicological studies suggest that SOAs can have inflammatory and irritant effects (Rohr 2013). However the relevance of these results to exposure to PM derived from biogenic VOCs is tenuous.

5.10.4 Summary

It is thought that biogenic emissions of VOCs may contribute substantially to secondary organic aerosols in the atmosphere, however, the health effects of PM from this source have not yet been investigated.

- The contribution of biogenic VOCs to ambient PM concentrations in NSW is unknown.
- The health effects of PM derived from biogenic VOCs have not been investigated in Australia or elsewhere and so the effects are unknown.

5.11 Health effects of source-specific PM relevant to NSW –Summary

Evidence from many studies indicates that the source of PM emissions is an important determinant of the health effects of ambient PM exposure. There is more evidence of health effects associated with exposure to PM from some emission sources compared to others. The majority of solid evidence of adverse health outcomes associated with exposure to source-specific PM comes from studies of the effects of exposure to PM from the following emission sources.

- Coal-fired power stations;
- On-road vehicles;
- Diesel exhaust emissions;
- Bushfires;
- Crustal dust (especially during dust storms).

However, the amount of evidence does not necessarily reflect the relative harmfulness of PM from these different sources, as the sources with the majority of health effects evidence (*i.e.*, coal-fired power stations, on-road vehicles and diesel exhaust emissions) are also the sources that have been most extensively studied. Thus it is not entirely clear that PM from these sources is more harmful than PM from less well-studied sources, or that there is a clear 'hierarchy' of harmfulness for PM from different emission sources. Despite these limitations, it is possible to draw some conclusions regarding the strength of evidence of health effects related to exposure to source-specific PM.

- **There is strong evidence that exposure to ambient PM from combustion sources (*i.e.*, coal-fired power stations, on-road vehicles, diesel exhaust) has adverse effects on health.**
- **The high concentrations of ambient PM during bushfires and dust storms are clearly associated with adverse health effects, particularly in vulnerable individuals (*i.e.*, the elderly, the very young and, people with chronic respiratory and cardiovascular disease). There is weaker evidence that ambient crustal dust, in the absence of a dust storm, has impacts on health.**
- **Methodological limitations of studies of the environmental health effects of coal mining activities mean that there is limited evidence that exposure to coal dust particles, independently of other coal mining pollutants, is responsible for adverse health outcomes.**
- **The health effects of ambient exposure to smoke from domestic wood-fired heating has not been extensively studied however this exposure has been associated with impacts on respiratory health, particularly in children.**
- **The evidence of health effects associated with exposure to either sea salt or PM derived from VOCs of biogenic origin is limited due to a paucity of studies.**
- **Current evidence suggests that PM from any source could potentially impact on health, although for some specific PM sources, health data is very limited.**
- **In general, the magnitude of health effect for source specific PM does not, at present, appear to be greater than the effects of exposure to total PM mass.**

A significant contributing factor to the limited evidence of health effects attributable to exposure to source-specific PM is the measurement of exposure. Population and personal outdoor exposure to source-specific PM is a relatively new area of investigation. It is technically challenging to quantify exposure to PM from specific outdoor emission sources because the number of different sources of PM is large and the concentrations of PM in ambient air are constantly changing. The techniques to measure ambient source-specific PM and the methods used to quantify exposure to these particles have advanced considerably in recent years. These advancements aim to provide more accurate determinations of exposure. It is likely that future studies will provide stronger evidence of specific health effects associated with exposure to PM from specific emission sources.

6. Evidence of the health impacts of different size fractions of PM

PM emissions are regulated in various regions of the world on the basis of ambient air concentrations of PM with median aerodynamic diameters of <10 µm (PM_{10}) or <2.5 µm ($PM_{2.5}$). For this reason, most studies investigating the health effects of PM have also used these size cut-offs. The widely accepted view about PM size and the health effects is summarised by the following:

> the smaller the particle size, the further the particle can penetrate the ever smaller branches of the respiratory airways, and inhalable particles >2.5 µm deposit primarily in the larger airways of the lung and affect respiratory health, whereas smaller particles penetrate to the alveoli and terminal bronchioles of the lung (where inhaled gases exchange with gases in the blood), where they initiate health effects of the cardiovascular system and other organs (Sandstrom and Forsberg 2008, Kelly and Fussell 2012, Nemmar et al. 2013a).

There is no physiological basis for the 2.5 µm cut-off (Cao 2013). Lung deposition is greater for smaller particles although it is not a linear relationship (*Figure 6.1*) (Chow 1995). From 2.5 µm to 10 µm there is a decrease in lung deposition, with almost no particles >10 µm depositing in the lung.

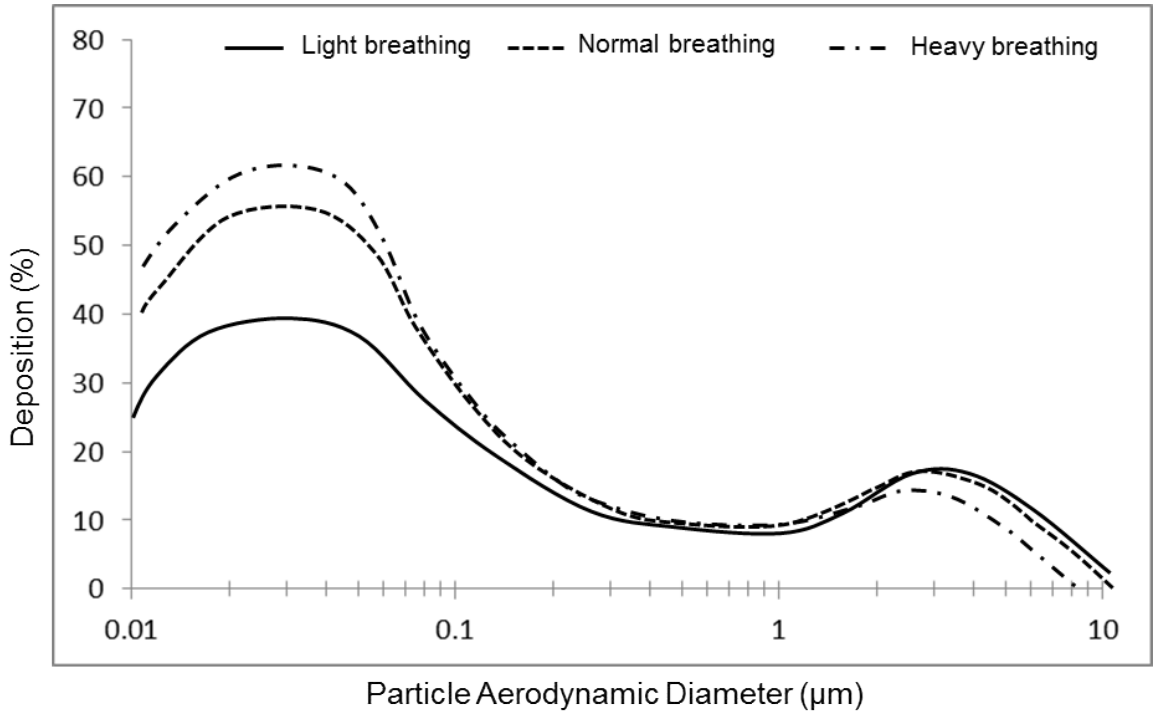

Figure 6.1 Lung deposition of PM by particle size (adapted from (Chow 1995))

The PM_{10} size fraction (that is, all particles <10 µm in diameter) also includes particles that are <2.5 µm in diameter. Thus PM_{10} and $PM_{2.5}$ both contain particles that are <2.5 µm. This overlap obviously attenuates observed differences in health effect associations with these two PM fractions. A WHO review concluded that analyses based on PM_{10} are generally unable to support statements on the relative importance of $PM_{2.5}$ and coarse PM ($PM_{10-2.5}$) (WHO 2004). To avoid the attenuation of the differences in the health effects between PM_{10} and $PM_{2.5}$ because of overlap between the two size fractions, a more useful comparator to $PM_{2.5}$ is coarse PM. However, coarse PM is seldom measured separately and reported on.

A further complication in comparing the health effects of PM_{10} and $PM_{2.5}$ occurs when the same increase in particle mass is used as the index of effect for each particle fraction. In six cities in the US, a 10 µg/m^3 increase in $PM_{2.5}$ was associated with a 1.5% increase in daily mortality compared to a 0.8% increase in daily mortality for a 10 µg/m^3 increase in PM_{10} (Schwartz et al. 1996). Both effects were statistically significant. The greater increase in mortality with $PM_{2.5}$ may reflect the fact that the increment in PM for which health effects were assessed, is measured in mass concentration (µg/m^3) and any increase in mass concentration will result in a larger increase in particle number concentration (PNC) for the smaller ($PM_{2.5}$) particle fraction than for the larger (PM_{10}) particle fraction. If PNC is relevant to the health effect, then measurement of increments in particle mass concentration would represent a biased index of effect. Hence, some continuing uncertainty about the most relevant index of PM exposure for measuring health effects (particle mass versus particle number) complicates the interpretation of the relative impact of specific size fractions.

6.1 Health effects associated with the coarse PM fraction ($PM_{10-2.5}$)

The US EPA, in their 2009 *Integrated Science Assessment for Particulate Matter* concluded that the evidence from epidemiological studies, along with the more limited evidence from controlled human exposure and toxicological studies is suggestive of a causal relationship between short-term exposures to coarse PM and mortality, cardiovascular and respiratory effects (US EPA 2009).

Across 47 US cities, a 10 µg/m^3 increase in coarse PM (estimated from the difference between PM_{10} and $PM_{2.5}$) was associated with small, but statistically significant, increases in all-cause, cardiovascular, stroke and respiratory mortality (Zanobetti and Schwartz 2009). The largest effect (1.16% increase) was for respiratory mortality. When the results were adjusted to take ambient $PM_{2.5}$ levels into account, the results for coarse PM changed very little. A 10 µg/m^3 increase in $PM_{2.5}$ increased cardiovascular mortality twice as much as a 10 µg/m^3 increase in coarse PM, although as mentioned previously, equivalent mass increases in $PM_{2.5}$ and coarse PM will result in a larger increase in PNC for $PM_{2.5}$. For all-cause, stroke and respiratory mortality the effects of $PM_{2.5}$ and coarse PM were similar. Associations between coarse PM and cardiovascular mortality have also been observed in single city studies in Phoenix and Vancouver (US EPA 2009). A daily time-series analysis of mortality in Mexico City found that on a mass basis, increases in coarse PM were more strongly associated with increases in daily mortality (4.0%) compared to either PM_{10} (1.8%) or $PM_{2.5}$ (1.5%) (Castillejos et al. 2000). The effect of coarse PM on daily mortality was stronger for respiratory diseases. When both coarse PM and $PM_{2.5}$ were included in the statistical model together, the effect of coarse PM on mortality was retained (4.0% increase), while the association with $PM_{2.5}$ was eliminated (non-significant 0.18% change).

Other investigations have also reported associations between coarse PM and health effects, and sometimes these associations were stronger than for $PM_{2.5}$. A review of epidemiological evidence published in 2005 concluded that short-term exposure to coarse PM had a stronger or as strong an association with daily hospital admissions for respiratory disease, COPD and asthma as did exposure to $PM_{2.5}$ (Brunekreef and Forsberg 2005). There was also support for an association between coarse PM and cardiovascular admissions. However, there was little evidence of health effects from long-term exposure to coarse PM. In a study of six cities in France, daily hospital admissions for respiratory diseases and respiratory infections were more strongly associated with coarse PM than $PM_{2.5}$ (Host et al. 2008). However, a study examining emergency hospital admissions across 108 US counties found no significant association between respiratory hospital admissions and coarse PM (Peng et al. 2008). That study, however, found that coarse PM was significantly associated with cardiovascular admissions, although $PM_{2.5}$ was more strongly associated than was coarse PM. Coarse PM has been associated with first hospitalisations for respiratory disease in children <3 years of age in Vancouver, Canada (Yang et al. 2004). In Toronto, Canada, daily hospital admissions for asthma in children (6-12 years) were associated with coarse PM but not $PM_{2.5}$ (Lin et al. 2002). The US EPA has concluded that the strongest evidence of coarse PM adversely impacting on respiratory health has been observed among children (US EPA 2009). A more recent analysis of ten European birth cohorts within the *European Study of Cohorts for Air Pollution Effects* (ESCAPE) *Project* found that pneumonia during early life was associated with exposure (estimated using land use regression models) to coarse PM and PM_{10}, but not $PM_{2.5}$ (MacIntyre et al. 2014b).

Studies reporting on the health effects of exposure to PM from dust storms have observed mixed results with respect to the relative impact of coarse PM versus $PM_{2.5}$. In the US, increases in ambient concentrations of coarse PM as a consequence of dust storms after crop harvesting were not associated with an increase in mortality (Schwartz et al. 1999). The study authors concluded that this supports the view that coarse PM has a relatively minor health impact. However, it should be noted that the findings of this study cannot necessarily be generalised beyond the specific source of coarse PM that was investigated, that is, dust storms after crop harvesting. Some studies that have examined the effect of air pollution from dust storms on mortality have stratified data into dust and dust-free days. The effect of coarse PM on dust storm-free days has been found to be similar to the effect of coarse PM in general. Studies from Barcelona (Perez et al. 2008) and Rome (Mallone et al. 2011) found that coarse PM on dust storm-free days was positively associated with mortality, whereas a study from Madrid (Jimenez et al. 2010) did not find such an association. In the two studies from Spain, $PM_{2.5}$ was more strongly associated with mortality than was coarse PM, whereas the Rome study, found coarse PM was more strongly associated with mortality than $PM_{2.5}$.

In summary, exposure to coarse PM exerts health impacts, evidenced by increases in mortality and, both respiratory and cardiovascular hospitalisations, although this has not been observed in all settings. Coarse PM appears to have an impact on respiratory health that is at least equal to that of $PM_{2.5}$, particularly in children. The evidence relating impacts on cardiovascular health to exposure to coarse PM is presently less strong.

6.2 Health effects associated with $PM_{2.5}$

Older studies focussed on the health effect associations with PM_{10}, however, in the past two decades $PM_{2.5}$ has increasingly been studied and there is now considerable evidence linking exposure

to $PM_{2.5}$ with adverse health effects. The majority of large population studies have examined the association between mortality and short-term (daily) or long-term (months/years) exposure to $PM_{2.5}$ (*Table 6.2.1*). Short-term exposure to $PM_{2.5}$ has been associated with increases in all-cause, respiratory and, cardiovascular mortality. The data is less consistent for long-term exposures, possibly because the spatial and temporal variations of ambient pollution concentrations and the movements of individuals over a long period mean that average annual concentrations do not provide an accurate measure of exposure. However, studies of mortality and annual ambient $PM_{2.5}$ concentrations generally report an increase in mortality risk with increasing exposure (although not all estimates have been statistically significant) (COMEAP 2009). There is more evidence of associations between long-term exposure to $PM_{2.5}$ and all-cause and cardiovascular mortality than there is for respiratory mortality (US EPA 2012a).

Short-term exposure to $PM_{2.5}$ has been consistently positively associated with daily hospital emergency presentations and admissions for all respiratory diseases, COPD, asthma and respiratory infections (US EPA 2012a, WHO 2013b). Among children with asthma, exposure to $PM_{2.5}$ has been associated with respiratory symptoms and lower lung function. However, $PM_{2.5}$ has not consistently been associated with hospital admissions or presentations to emergency departments for asthma in children (US EPA 2009). The US EPA concluded that a causal relationship is <u>likely</u> between short-term exposure to $PM_{2.5}$ and respiratory effects (US EPA 2009).

Short-term exposure to $PM_{2.5}$ has also been consistently positively associated with hospital emergency presentations and admissions for ischaemic heart disease and congestive heart failure (US EPA 2009). The US EPA concluded that a causal relationship <u>exists</u> between short-term exposures to $PM_{2.5}$ and cardiovascular effects (US EPA 2009). Studies from the US and Canada published since publication of the *US EPA, 2009 Integrated Scientific Assessment for particulate matter* have also found consistent positive associations between short-term $PM_{2.5}$ exposure and cardiovascular-related hospital admissions and emergency department presentations (US EPA 2012a, Lippmann 2014).

Long-term exposure to $PM_{2.5}$ has been associated with similar cardiovascular and respiratory health effects as for short-term exposure (US EPA 2009). Compared to time-series studies examining the effects of short-term exposure, there have been fewer studies that have examined the association between average annual concentrations of $PM_{2.5}$ and measures of cardiovascular morbidity such as hospitalisations. Consequently concluding statements regarding the cardiovascular health effects of long-term exposure to $PM_{2.5}$ are not as strong as those for short-term exposure. The US EPA concluded that epidemiological and toxicological evidence is sufficient to <u>infer</u> a causal relationship between long-term $PM_{2.5}$ exposures and cardiovascular effects (US EPA 2009). Recent evidence from 11 cohorts within the European ESCAPE Study, in which land-use regression models were used to estimate individual exposures, is suggestive of associations between long-term exposure to $PM_{2.5}$ (and PM_{10}) and the incidence of first-time coronary and stroke events (Cesaroni et al. 2014, Stafoggia et al. 2014). The associations in these studies were of borderline, statistical significance.

Table 6.2.1 Effect estimates from recent large, multi-city studies of the mortality effects of exposure to $PM_{2.5}$

Study and population	Increase in $PM_{2.5}$	Increase in mortality associated with an increase in $PM_{2.5}$	
Short-term exposure			
(Dai et al. 2014) 75 US cities	10 µg/m^3	All-cause Respiratory Cardiovascular Myocardial infarction Stroke	1.18% 1.71% 1.03% 1.22% 1.76%
(Samoli et al. 2013) 12 European cities	10 µg/m^3	All-cause Respiratory Cardiovascular	0.55% 1.91% 0.86%
(Zanobetti and Schwartz 2009) 112 US cities	10 µg/m^3	All-cause Respiratory Cardiovascular Myocardial infarction Stroke	0.98% 1.68% 0.85% 1.18% 1.78%
(Franklin et al. 2007) 27 US communities	10 µg/m^3	All-cause Respiratory Stroke	1.21% 1.78% 1.03%
Long-term exposure			
(Beelen et al. 2014a) 13 European countries	5 µg/m^3	All-cause	7%
(Beelen et al. 2014b) 13 European countries	5 µg/m^3	Cardiovascular Myocardial infarction Stroke	No increase* No increase No increase
(Dimakopoulou et al. 2014) 11 European countries	5 µg/m^3	Respiratory	No increase
(Raaschou-Nielsen et al. 2013) 9 European countries	5 µg/m^3	Lung cancer	No increase
(Lepeule et al. 2012) 6 US cities	10 µg/m^3	All-cause COPD Cardiovascular Lung cancer	14% No increase 26% 37%
(Hart et al. 2011) 50,000 males in the US trucking industry	4 µg/m^3	All-cause Respiratory Cardiovascular Lung cancer	3.9% No increase No increase No increase
(Puett et al. 2009) 100,000 female nurses in the US	10 µg/m^3	All-cause Coronary heart disease	26% 102%

* *No increase*: no statistically significant increase in mortality

Paradoxically, while the evidence linking long-term exposure to $PM_{2.5}$ with mortality is stronger for cardiovascular than respiratory mortality, the evidence related to morbidity is stronger for respiratory than cardiovascular effects. The epidemiological evidence reviewed in the *US EPA 2009 Integrated Science Assessment for Particulate Matter* demonstrates associations between long-term exposure to $PM_{2.5}$ and decrements in lung function growth in children, lung function in adults, increased respiratory symptoms, and asthma development (US EPA 2009). Recent studies provide

additional evidence for these associations t (US EPA 2012a). Birth cohort studies have reported significant associations between long-term exposure to $PM_{2.5}$ and respiratory infections and asthma in children (as well as birth outcomes including low birth weight and preterm birth) (US EPA 2012a, WHO 2013c). The *US EPA 2009 Integrated Science Assessment for Particulate Matter* concluded that collectively, the evidence is sufficient to conclude that the relationship between long-term $PM_{2.5}$ exposure and respiratory effects is likely to be causal (US EPA 2009).

As measures to improve air quality in Europe and the US have brought about lower ambient pollution concentrations, evidence has emerged that further lowering of $PM_{2.5}$ levels are likely to bring about improvements in health (WHO 2013c). In Australia, the current NEPM advisory reporting standard for annual average $PM_{2.5}$ is 8 $\mu g/m^3$. A follow-up of the Harvard Six Cities Study in the US (with data from 1974-2009) found that decreases of annual average $PM_{2.5}$ down to 8 $\mu g/m^3$ (the lowest observed concentration) were associated with decreases in all-cause, cardiovascular and lung cancer mortality (Lepeule et al. 2012). Evidence from 12 southern Californian communities suggests that lung development in children (10-18-years of age) is negatively impacted by long-term exposure to $PM_{2.5}$ down to concentrations of approximately 5.0 $\mu g/m^3$ (Gauderman et al. 2004).

A study of over two million subjects used both ground-based and satellite monitoring to assign estimates of annual $PM_{2.5}$ exposure across Canada (Crouse et al. 2012). Average annual $PM_{2.5}$ concentration estimates away from population centres were <8 $\mu g/m^3$. The average annual exposure for all subjects across Canada was estimated at 8.7 $\mu g/m^3$ (substantially lower than estimates from previous studies), and the minimum average annual $PM_{2.5}$ exposure was 1.9 $\mu g/m^3$. Associations between $PM_{2.5}$ and all-cause and cardiovascular mortality risk were linear down to average annual $PM_{2.5}$ concentrations <2 $\mu g/m^3$. This suggests that improvements in health outcomes are likely to be seen with improvements in long-term air quality down to very low (essentially background) levels of ambient $PM_{2.5}$. However, there was less confidence associated with estimates of effect for $PM_{2.5}$ concentrations that are <5 $\mu g/m^3$ and it is possible that decreasing accuracy of effect estimates at low concentrations of $PM_{2.5}$ may mask an effect threshold.

Lowering annual ambient $PM_{2.5}$ levels is also likely to lead to improvements in health outcomes related to short-term exposures. Recent evidence linking increases in average daily $PM_{2.5}$ with increases in respiratory- and cardiovascular-related emergency hospital presentations and admissions included studies where the average 24-hour $PM_{2.5}$ concentrations were 6.1-6.7 $\mu g/m^3$ (US EPA 2012a). In an assessment of mortality, the estimated dose-response relationship between $PM_{2.5}$ levels and daily deaths in six US cities essentially had no minimum $PM_{2.5}$ threshold and was at least linear down to 2 $\mu g/m^3$ $PM_{2.5}$ (Schwartz et al. 2002). A threshold of effect has also not been identified for ambient PM_{10} levels and daily all-cause and cardiorespiratory mortality in the 20 largest US cities (Daniels et al. 2000), suggesting that any improvement in ambient PM_{10} will result in a decrease in these mortalities.

Existing studies do not provide evidence of a threshold of effect for ambient $PM_{2.5}$, suggesting that improvements in $PM_{2.5}$ will result in further improvements in population health outcomes. The few studies that have been able to observe ambient $PM_{2.5}$ concentrations below the current NEPM advisory reporting standard (annual average 8 $\mu g/m^3$) suggest that improvements in population health outcomes can be achieved at $PM_{2.5}$ concentrations lower than 8 $\mu g/m^3$.

The continued demonstration of adverse health effects in recent decades, even as concentrations of ambient PM have declined in some cities, does not support evidence of a threshold. However, there is less certainty associated with concentration-response relationships at very low concentrations of PM air pollution and therefore it is possible that a threshold exists but that the precision of current methods does not enable the threshold to be identified.

Some PM emissions are unavoidable (*e.g*. sea spray, crustal dust from natural landscapes) and therefore ambient PM can be reduced only so much through human endeavours. However, evidence suggests that further reductions in anthropogenic PM emissions in NSW are likely to have health benefits.

6.3 Health effects associated with ultrafine ($PM_{0.1}$) particles

Ultrafine particles ($PM_{0.1}$) are less than 0.1 µm in diameter. Within both the PM_{10} and $PM_{2.5}$ fractions, $PM_{0.1}$ contributes little to the total mass but is the dominant contributor to PNC. Combustion is the main source of $PM_{0.1}$. Specifically, the major sources of $PM_{0.1}$ emissions are: on-road vehicle exhausts, non-road transport exhausts (mostly off-road diesel), residential and commercial heating and, industrial combustion processes (HEI 2013). In the atmosphere, coagulation (particle collision and adherence) favours emitted $PM_{0.1}$ growing to >0.1 µm within hours of emission and, ambient concentrations of $PM_{0.1}$ rapidly decrease within a few hundred metres of emission sources (HEI 2013).

The properties of $PM_{0.1}$ that are cause for health concern are three-fold:

1. The very small size enables these particles to pass from the lungs into the blood circulation and potentially cause cardiovascular and other systemic health effects;
2. These particles account for the majority of particles in ambient air and $PM_{0.1}$ enter the lungs with higher efficiency and are cleared more slowly than larger particles;
3. The high surface area per unit of mass of $PM_{0.1}$ is thought to cause higher toxicity because, compared with larger particles, $PM_{0.1}$ have larger amounts of adsorbed or condensed toxic air pollutants (oxidant gases, organic compounds, transition metals) per unit mass.

Estimating human exposure to $PM_{0.1}$ in epidemiological studies is challenging because routine monitoring for $PM_{0.1}$ does not exist and there are steep spatial gradients of $PM_{0.1}$ concentrations in urban settings. It is possible that $PM_{0.1}$ contribute to elevated health risks associated with living in close proximity to roads (WHO 2013c), but further research is needed in this area. In the absence of extensive monitoring of $PM_{0.1}$, mathematical modelling methods have been used to estimate exposure (HEI 2013). Short-term $PM_{0.1}$ exposure has been inconsistently associated with various short-term (daily) respiratory and cardiovascular health effects (*Table 6.3.1*).

Table 6.3.1 Health effect associations with short-term exposure to $PM_{0.1}$

Study	Health effect	Association
	All-cause mortality	
(Breitner et al. 2009)	All-cause mortality	✓
	Respiratory health	
(Halonen et al. 2009) subjects ≥ 65 years	Respiratory mortality	×
	Asthma/COPD hospital admissions	×
	Pneumonia hospital admissions	✓
(Leitte et al. 2011)	Respiratory presentations to ED	✓
(Peel et al. 2005)	Respiratory presentations to ED	×
	Asthma presentations to ED	✓
(Halonen et al. 2008)	Asthma/COPD presentations to ED (adults)	×
	Asthma presentations to ED (children)	✓
(Sinclair and Tolsma 2004)	Asthma presentations to ED	✓
	Respiratory infection presentations to ED	✓
(Klot et al. 2002)	Asthma symptoms	✓
	Asthma medication use	✓
(Penttinen et al. 2001)	Decreased lung function in asthma patients	✓
(Osunsanya et al. 2001)	COPD symptoms	×
	Decreased lung function in COPD patients	×
	Cardiovascular health	
(Breitner et al. 2011)	Cardiovascular mortality	✓
	Ischaemic heart disease mortality	✓
(Halonen et al. 2009) subjects ≥ 65 years	Cardiovascular mortality	✓
	Coronary heart disease/stroke hospital admissions	×
	Arrhythmia hospital admissions	✓
(Stolzel et al. 2007)	Cardiovascular mortality	✓
(Kettunen et al. 2007) subjects ≥ 65 years	Stroke mortality	×
(Andersen et al. 2010)	Stroke hospital admissions	×
(Metzger et al. 2004)	Cardiovascular presentations to ED	×
(de Hartog 2003)	Cardiorespiratory symptoms in cardiovascular patients	×
(Weichenthal et al. 2011)	Heart rate variability	✓
(Rich et al. 2012)	Heart rate variability in cardiovascular patients	✓
	Blood pressure change in cardiovascular patients	✓
(Schneider et al. 2010)	Heart rate variability in cardiovascular patients	×
(Barclay et al. 2009)	Heart rate variability in cardiovascular patients	×
(Lanki et al. 2008)	Heart rate variability in cardiovascular patients	×
(Timonen et al. 2006)	Heart rate variability in cardiovascular patients	✓
(Henneberger et al. 2005)	Heart rate variability in cardiovascular patients	✓
(Ibald-Mulli et al. 2004)	Heart rate variability in cardiovascular patients	×
	Blood pressure change in cardiovascular patients	×
(Pekkanen et al. 2002)	Heart rate variability in cardiovascular patients	✓

(✓) statistically significant association observed, (×) statistically significant association not observed
ED, hospital emergency department
Studies that used total ambient particle number concentration (which includes particles >0.1 µm) as a surrogate measure for $PM_{0.1}$ concentration have not been included in this table.

There is considerable evidence that exposure to $PM_{0.1}$ is associated with exacerbation of asthma symptoms (Klot et al. 2002, Sinclair and Tolsma 2004, Peel et al. 2005, Halonen et al. 2008). Animal studies have shown that inhalation of $PM_{0.1}$ can induce respiratory airway inflammation (HEI 2013) and in controlled exposures to $PM_{0.1}$ in humans it has been shown that lung deposition of particles is higher in people with asthma than in people without asthma (WHO 2013c). Inflammation can take several days to develop and one review reports that the cumulative effects of $PM_{0.1}$ over five days are stronger than same-day effects (Morawska et al. 2004). Respiratory inflammation caused by $PM_{0.1}$ may result in biological changes that enhance the transfer of $PM_{0.1}$ beyond the lung to the cardiovascular system (Nemmar et al. 2013a).

Exposure to $PM_{0.1}$ has been associated with cardiovascular mortality (Stolzel et al. 2007, Halonen et al. 2009, Breitner et al. 2011), which may be related to effects on heart rate variability and blood pressure in people with established cardiovascular disease (Rich et al. 2012). It is considered likely that components in $PM_{0.1}$, if not the whole particle, reach the cardiovascular system (Delfino et al. 2005). High $PM_{0.1}$ exposure may promote systemic inflammation through oxidative stress responses to surface components of $PM_{0.1}$ particles. It has been observed that changes in vascular reactivity that occur in response to exposure to $PM_{0.1}$ from diesel exhaust, are not induced when humans are exposed to $PM_{0.1}$ without components adsorbed to the surface (clean carbon particles) (Mills et al. 2011). There is further evidence from animal studies that (semi) VOCs and metals on $PM_{0.1}$ are responsible for inducing inflammatory responses and, synergistic interactions between $PM_{0.1}$ and transition metals lead to greater health effects (WHO 2013c). Systemic inflammation promotes cardiovascular disease (Ross 1999) which can in turn precipitate an acute response (*e.g.* heart attack or stroke). Thus biological responses to inhaled $PM_{0.1}$ could explain events (mortality, hospital admissions) in people with cardiovascular disease.

Changes in concentrations of blood markers of inflammation and clotting in patients with cardiovascular disease have been related to exposure to $PM_{0.1}$, although $PM_{2.5}$ and PM_{10} have also been associated with changes in these biomarkers (Ruckerl et al. 2006). It could be argued that it is the particles <0.1 µm in diameter in $PM_{2.5}$ and PM_{10} that are responsible for these biological responses, however exposure to particles >0.1 µm in diameter have also been associated with biomarkers of inflammation and coagulation (Ruckerl et al. 2006). While exposure to $PM_{0.1}$ alone cannot account entirely for the adverse effects of $PM_{2.5}$ (HEI 2013), the mechanisms of toxicity of $PM_{0.1}$ (oxidative stress, inflammation, protein and DNA damage, breakdown of cellular defences) are the same as for larger particles (WHO 2013c).

While few human controlled exposure studies have evaluated specifically the effects of $PM_{0.1}$, many studies have exposed subjects to PM from sources such as wood combustion, urban traffic and diesel exhaust, which have high concentrations of $PM_{0.1}$. These studies have reported increases in markers of systemic oxidative stress, inflammation and blood coagulation (US EPA 2009). Controlled exposure to concentrated $PM_{0.1}$ has resulted in decreased lung function however increased respiratory symptoms have not been reported (US EPA 2009).

The health impact of long-term exposure to $PM_{0.1}$ has not been studied. This is a consequence of limited monitoring of $PM_{0.1}$ and insufficient spatial resolution to consistently identify contrasts in exposure (Sioutas et al. 2005). Cross-sectional studies that have assessed the prevalence of chronic disease in different locations with different concentrations of ambient $PM_{0.1}$ have not been able to

attribute exposure to $PM_{0.1}$ to either disease onset or disease progression (HEI 2013). Thus the long-term health effects of exposure to $PM_{0.1}$ are unknown.

Although there is considerable evidence that $PM_{0.1}$ can contribute to short-term health effects, there is insufficient evidence to determine concentration-effect functions. Also, the effects of $PM_{0.1}$ cannot be directly compared to the effects of larger particles since owing to its small particle mass $PM_{0.1}$ is measured in units of particle number concentration rather than particle mass concentration (which is used to quantify the larger PM fractions).

- Increases in mortality and, respiratory and cardiovascular hospitalisations have been associated with exposure to coarse PM ($PM_{10-2.5}$), although the evidence base is less than for $PM_{2.5}$ and $PM_{10.}$
- The impact of coarse PM on respiratory health appears to be at least equivalent to that of $PM_{2.5.}$
- Short-term exposure to $PM_{2.5}$ has consistently been positively associated with mortality and, respiratory and cardiovascular health effects.
- Long-term exposure to $PM_{2.5}$ has been positively associated with mortality and, respiratory and cardiovascular health effects,, however the evidence to date is more variable than for short-term effects.
- There is a small amount of evidence that exposure to $PM_{2.5}$ levels <8 $\mu g/m^3$ are detrimental to health and, decreasing ambient $PM_{2.5}$ to as low as possible is likely to have health benefits.
- Short term exposure to ultrafine particles ($PM_{0.1}$) has been associated with respiratory morbidity (particularly asthma), cardiovascular mortality and changes in cardiovascular function.
- Health effects evidence related to $PM_{0.1}$ is more limited than for other particle fractions and further monitoring of $PM_{0.1}$ is required to provide additional evidence on which to make public health recommendations.
- $PM_{0.1}$ has higher lung penetration than larger particles however the biological mechanisms behind the health effects associated with $PM_{0.1}$ are likely to be the same as for other particle fractions.
- The biological effects of $PM_{0.1}$ are consistent with observed associations with cardiovascular outcomes.
- It is presently not possible to associate $PM_{0.1}$ from specific sources with specific health effects however exposure to $PM_{0.1}$ emissions should be minimised.

7. Evidence of the effect of PM composition on health impacts

There is general consensus that the composition of PM contributes to the potential for inhaled particles to impact health. Given that PM contains elements and compounds with known toxic properties it would be somewhat surprising if the composition of PM had no effect on the capacity of individual particles to impact health. Indeed, the toxicity of elements and compounds that are part of PM may be as important as the mass concentration of ambient PM in determining the health consequences of exposure. What is important is not whether toxic chemicals exist in and on PM (they do), but whether the usual concentration of these chemicals in ambient PM are sufficiently high to affect the health of healthy and vulnerable individuals. This is very difficult to demonstrate. Not only are ambient particles composed of hundreds of different chemicals in various concentrations, but certain elements and compounds from the same source often occur together, making it difficult to determine which component(s) is of most concern. Grouping commonly associated components of PM into factors (*factor analysis*) or, apportioning certain ambient PM to a particular source (*source apportionment*) and then investigating relationships between health outcomes and these constituent groups or source apportioned PM are methods used to address this complexity (Mar et al. 2006, Kim et al. 2007). However these methods are subject to significant limitations (Grahame and Hidy 2007) and on their own do not provide conclusive evidence of health effects attributable to specific PM components. Individual constituents may be associated with health effects but it has been recognised that by simply associating PM constituents with health endpoints, constituents that are more strongly correlated with PM mass may appear more closely related to adverse health outcomes than other constituents, without being inherently more toxic (Mostofsky et al. 2012). Evidence obtained via these means needs to be considered together with controlled human exposure studies and animal toxicological studies in order to provide strong evidence linking PM composition with health impacts.

Consideration of which components of PM are more strongly associated with adverse health effects is a relatively recent development. As such, the methods to address this issue are still evolving and the amount of evidence linking PM composition with health impacts is relatively small and limited. In this chapter, evidence related to common components of ambient PM (trace metals, organic molecules, sulphates and nitrates) is summarised.

7.1 Trace metals

Metals in PM are often cited as the component most likely to exert health effects. Metals are found in many anthropogenic emission sources including vehicle exhausts and, non-exhaust vehicle emissions from mechanical abrasion of brakes and tyres. Metals are also components of crustal dust (Schlesinger 2007, Gunawardena et al. 2013). Metals known as transition metals (because their chemistry allows them to exist in many forms or oxidative states) have been investigated for possible links with health effects as these metals can react with biological tissues and generate cellular-damaging oxidative stress. Transition metals include: vanadium, chromium, iron, nickel,

copper and zinc. Other metals in PM with the potential to elicit health effects include arsenic and lead.

Evidence linking metals in PM to health outcomes has come from particle speciation studies and factor analysis. Analyses in such studies have found statistically significant associations between specific metals and health impacts however it has not been possible to attribute the health impacts solely to any one component of the PM. In California, metals in $PM_{2.5}$, particularly those from combustion emissions, have been linked to increases in daily mortality (Ostro et al. 2007). Copper, zinc, iron, manganese, titanium and vanadium in mobile source-related emissions were all associated with increases in all-cause and cardiovascular mortality. Lead was associated with all-cause but not cardiovascular mortality. Because of high correlations between various metals and other components of PM it was not possible to conclude which of these metals, if any, were responsible for changes in daily mortality. However several patterns in the data emerged. All-cause mortality was associated most strongly with copper and $PM_{2.5}$ mass. Cardiovascular mortality was associated most strongly with zinc, potassium, elemental carbon and $PM_{2.5}$ mass. However, another study (in which personal exposures were estimated) did not find an association between cardiovascular mortality and either zinc, copper, iron, nickel, silicon or vanadium for either $PM_{2.5}$ or PM_{10} (Wang et al. 2014b). In the study of Ostro *et al.*, respiratory mortality was associated most strongly with the metals copper and titanium (more strongly than was $PM_{2.5}$ mass).

In another US study, conducted in 25 communities, the strength of the association between two-day averaged $PM_{2.5}$ mass and daily mortality increased when aluminium, arsenic, silicon, nickel or sulphate contributed larger proportions to the $PM_{2.5}$ mass (Franklin et al. 2008). Of 18 components of $PM_{2.5}$ assessed, aluminium and nickel explained most of the variability in the $PM_{2.5}$ mass-mortality associations between the different communities. In a study conducted in eight Canadian cities, among 47 elements measured in $PM_{2.5}$, nickel, iron and zinc were most strongly positively associated with daily mortality (Burnett et al. 2000). The total effect of these three elements plus sulphate on mortality was greater than the effect of $PM_{2.5}$ mass.

A synthesis of various methods of source apportionment found that copper-related PM was more strongly associated with all-cause mortality than PM attributed to traffic (Mar et al. 2006). Source apportionment of $PM_{2.5}$ in Barcelona found that $PM_{2.5}$ with high concentrations of zinc and lead were not associated with greater mortality risk (Ostro et al. 2011). Element-specific analysis of $PM_{2.5}$ from six US cities found that nickel and lead were significantly positively associated with daily deaths (Laden et al. 2000). When zinc was used as a tracer to source-apportion $PM_{2.5}$ it was found to be associated with emergency department presentations for cardiovascular, but not respiratory, disease (Sarnat et al. 2008). In a birth cohort in the Netherlands, iron, copper and zinc in PM, thought to be reflective of non-exhaust traffic emissions, were associated with aspects of asthma and allergy in children (Gehring et al. 2015). Long-term exposure to other metals in PM that point to impacts of resuspended road dust, namely potassium, silicon and iron, have been associated with the incidence of coronary events in cohorts across Europe (Wolf et al. 2015).

Some studies have failed to observe associations between metals in PM and outcomes such as cardiovascular and respiratory emergency department presentations (Chen and Lippmann 2009). Among annual average personal exposure (estimated by land use regression models) to eight different components of $PM_{2.5}$ and PM_{10} (copper, iron, potassium, nickel, sulphur, silicon, vanadium

and zinc) in seven European birth cohorts, pneumonia in early life was weakly associated with PM_{10}-zinc but not with the other elements (Fuertes et al. 2014). Exposure to PM_{10}-nickel, but not other metals (copper, iron, silicon, vanadium and zinc), has been associated with decreased lung function in children (Eeftens et al. 2014).

There has been a concerted effort to investigate the health impact of nickel and vanadium because these transition metals are in high abundance in emissions from fuel oil combustion. Across 60 US cities with particle speciation data, the estimated long-term mortality risk from a 10 $\mu g/m^3$ increase in ambient PM_{10} was higher in those cities where the nickel and vanadium content of $PM_{2.5}$ were significantly high (95^{th} percentile) (Lippmann et al. 2006). The concentrations of fourteen other components of $PM_{2.5}$ (including copper, lead, chromium, iron and arsenic) and $PM_{2.5}$ mass were not significantly associated with PM_{10} mortality risk estimates. These results, however, should be interpreted with some caution because they rely on comparisons between PM_{10}-associated daily mortality for 1987-1994 and $PM_{2.5}$ composition data for 2000-2003 (Lippmann et al. 2006). However, confirmatory results were reported from a recent analysis of the survival of Medicare enrolees in 81 US cities (2000-2010) (Kioumourtzoglou et al. 2015). The effect of annual $PM_{2.5}$ concentrations on survival was modified by particle composition, with the highest effect estimates observed in cities characterised by high nickel, vanadium and elemental carbon concentrations.

Indirect evidence of possible health impacts of nickel and vanadium was provided when Hong Kong switched to fossil fuels with low sulphur content in 1990. There was a decrease in cardiovascular and respiratory mortalities at this time (Hedley et al. 2002) and ambient concentrations of not only sulphates, but nickel and vanadium (but not other metals) decreased as a result of this intervention (Chen and Lippmann 2009).

Using three different models to assess the relationship between 18 constituents of ambient $PM_{2.5}$ and stroke risk among acute ischaemic stroke patients at a Boston medical centre, only concentrations of black carbon, nickel and vanadium were consistently positively associated with stroke onset (Mostofsky et al. 2012). An investigation into the impact of constituents of $PM_{2.5}$ on long-term mortality in a cohort of male US military veterans found that nickel, vanadium, elemental carbon and nitrate, but not other $PM_{2.5}$ constituents, were significantly associated with mortality (Lipfert et al. 2006). Nickel and vanadium have also been associated with cardiovascular and respiratory hospitalisations and, wheeze in children during the first two years of life (Kelly and Fussell 2012).

Numerous human exposure and animal toxicological studies have demonstrated that metals in PM are associated with health effects, including: respiratory inflammation, reduced lung function, oxidative stress, heart rate changes and abnormal heart beat (Kelly and Fussell 2012). Vanadium and zinc have been prominently associated with acute respiratory responses whereas nickel may play more of a role in cardiovascular effects (Lippmann and Chen 2009). In human volunteers, controlled exposure to concentrated PM has demonstrated that together copper, zinc and vanadium is associated with changes in biological markers consistent with systemic inflammation, while chromium and vanadium is associated with oxidative stress and DNA damage (Chen and Lippmann 2009). Vanadium is capable of disrupting the function of cells that line human lung airways (Zhang et al. 2009), while iron and chromium in ambient PM have been associated with the release of mediators of inflammation from human lung cells (Becker et al. 2005). Airway instillation of PM

collected in the Utah Valley whilst a steel mill was operational, induced lung injury and inflammation in humans, whereas PM collected when the steel mill was closed did not elicit these effects (Kelly and Fussell 2012). Particle analysis indicated the PM collected when the steel mill was in operation had a higher metal content (including iron, copper, nickel, lead and zinc).

Most of the evidence relating to the biological effects of metals has come from exposing animals to much higher metal concentrations than are environmentally relevant. Some toxicity studies using high doses of source-related PM (residual oil fly ash, coal fly ash) that contain multiple metals have produced effects that appear to be related to the metal content (Chen and Lippmann 2009). Those studies were not able to identify individual metals of concern. When rats were exposed to concentrated ambient PM, increased oxidative stress in the lung was strongly associated with the particle content of iron, manganese, copper and zinc while increased oxidative stress in the heart was associated with iron, aluminium and titanium (Gurgueira et al. 2002). A lack of association for total PM mass (rather than metal content) with oxidative stress in the lung (although not in the heart) suggested a cause-effect relationship between the presence of metals and the oxidant capability of concentrated ambient PM, as opposed to a non-specific effect caused by the physical interaction of particles with lung cells. Nickel and vanadium are both capable of producing respiratory and cardiovascular effects in animals (Zhang et al. 2009). Nickel can induce expression of oxidative stress-responsive and inflammatory genes and, alter blood vessel responsiveness to mediators of vascular tone (Cuevas et al. 2010).

When cardiovascular disease-prone mice were exposed to concentrated ambient $PM_{2.5}$, weak associations were observed between exposure to particles enriched in nickel, chromium and iron and increased heart rate and decreased heart rate variability, which are indicators of cardiac stress (Lippmann et al. 2006). When each metal was considered separately only nickel had a significant acute effect on heart rate and heart rate variability. This is the one example demonstrating that a specific PM metal may cause an effect that explains epidemiological observations. PM enriched with transition metals have also produced heart rate changes in a rat model of coronary heart disease, while in a dog model of cardiovascular disease, silicon- and lead-associated PM (but not nickel-associated PM) were associated with heart rate changes (Wellenius et al. 2003). There is some evidence that different transition metals may result in synergistic responses upon inhalation, and overall, it appears that the cardiovascular effects of ambient $PM_{2.5}$ are greatly influenced by their metal content (Lippmann and Chen 2009).

The qualitative coherence among epidemiological and toxicological studies suggests that metals contribute to the detrimental health outcomes associated with exposure to PM. Evidence is emerging that some metals in ambient PM are associated with adverse health effects at concentrations near to current ambient levels (Chen and Lippmann 2009) and, evidence is accumulating that transition metals emitted during combustion processes, particularly nickel and vanadium, may have health impacts.

7.2 Organic compounds

Although organic compounds constitute a considerable component of PM mass, there is much uncertainty over the contribution of organic components to the health effects of PM. This is largely owing to the complex, heterogeneous nature of organic compounds in PM, which has not been well

characterised. Also, because organic compounds are so ubiquitous in PM (10-70% of PM mass (US EPA 2004)) it is difficult to separate the effects of these constituents from the effects of PM in general. More than 200 organic species in PM have been identified (Seinfeld and Pandis 2006). The task of identifying potential toxicity of specific organic compounds is further complicated since they may undergo chemical modification that alters their ability to induce biological effects and, they can serve as carriers of adsorbed chemicals with toxic properties of their own (Kelly and Fussell 2012).

It can be said with reasonable confidence that exposure to the organic component of vehicle PM exhaust emissions has an adverse impact on health, even though there is no direct evidence. Extracts of organic material from vehicle emissions are known to be mutagenic to bacteria and mammalian cells and, carcinogenic when painted on mouse skin (Mauderly and Chow 2008). Individual organic compounds in exhaust emissions have dose-dependent toxicity in humans, such as the carcinogenicity of benzo[a]pyrene and the irritant potential of formaldehyde. The health effects of diesel exhaust particles are mediated by chemicals adsorbed on the particle surface and among these chemicals, the organic constituents, in particular PAHs and semi-VOCs are likely to be toxicologically active (Mills et al. 2011, WHO 2013c). Particle-bound PAHs have been associated with ischaemic heart disease mortality and adverse symptoms in survivors of myocardial infarctions. However the effects of PAHs have not been fully separated from the effects of the PM to which they are bound (WHO 2013c). Several adverse birth outcomes (pre-term birth, low birth weight, small for gestational age) associated with exposure of mothers to on-road vehicle emissions appear to be associated with PAHs (Grahame et al. 2014). It is possible that biologically active PAHs might be causally related to the impact that vehicle emissions have on birth outcomes. Pre-natal exposure to PAHs in air has been associated with morphological changes in the brains of children aged 7-9 years that are related to cognitive deficits and behavioural problems (Peterson et al. 2015).

Wood and biomass smoke have many deleterious effects on health, including premature mortality. These emissions have very high organic content (Lemieux et al. 2004) and because some of the organic molecules are known irritants, mutagens, and carcinogens, it is likely that the organic components of smoke contribute to the health effects of this source of PM, but as with vehicle emissions, there is no direct evidence of this.

Organic carbon in PM has been found to be associated with short-term changes in cardiovascular and respiratory health (Urch et al. 2005, WHO 2013c). Long-term exposure to organic carbon has been associated with ischaemic heart disease and lung disease mortality whereas elemental carbon was only associated with ischaemic heart disease mortality (Ostro et al. 2010). Thus there appears to be health responses that are related to carbon in the organic form. However not all organic carbon is the same and one study found differences in biological effect between exposure to organic carbon in primary and secondary PM (Delfino et al. 2010). There is no evidence that organic carbon is responsible for health effects that have not been associated with exposure to PM in general (Ostro et al. 2007, Franklin et al. 2008, Ostro et al. 2009, Lippmann 2014).

Associations between health effects and organic compounds attached to PM have been observed but it is unclear what specific role organic compounds have on the health impacts of PM. It is likely that the large number of organic compounds in PM from combustion sources will have some influence on the health impact of these particles. However, it is difficult to separate the effects of organic compounds from the effects of total particle mass and presently there is little direct

evidence that organic compounds on PM are responsible for the health effects associated with exposure to PM.

7.3 Sulphates

Sulphate in PM (largely present as ammonium sulphate) is mainly formed secondarily in the atmosphere from the oxidation of sulphur dioxide, a principal component of fossil fuel combustion but also arising from sulphur-containing gases released from oceans and wetlands (Reiss et al. 2007, Schlesinger 2007). Sulphate can be a considerable component of PM, and in the Sydney basin ammonium sulphate comprises approximately 20% of the total mass of $PM_{2.5}$ (Cohen et al. 2011), while in the Hunter Valley sulphate (ion) contributed approximately 12% to the total mass of $PM_{2.5}$ (Hibberd et al. 2013). Concentrations of sulphate in PM correlate with anthropogenic emissions of sulphur dioxide. In the US, the sulphate concentration in PM is highest in the Midwest and eastern states, where coal-fired power generation is responsible for sulphur dioxide emissions (Harrison and Yin 2000). Sulphate tends to be a more prominent component of $PM_{2.5}$ than it is in coarse particles (Brook et al. 1997).

Reiss *et al* summarised the results of time-series studies published up to 2005 that included health risk estimates for both $PM_{2.5}$ mass and $PM_{2.5}$-sulphate (Reiss et al. 2007). Across 11 study locations and 48 risk estimates for mortality and cardiorespiratory endpoints, total $PM_{2.5}$ mass and $PM_{2.5}$-sulphate were significantly positively associated with 16 and 9 (out of the 48) health outcomes, respectively. Relative risks for $PM_{2.5}$ and sulphate were reported on a per 10 $\mu g/m^3$ increase basis, which does not allow a direct comparison between $PM_{2.5}$ and sulphate. This is because a 10 $\mu g/m^3$ increase in sulphate represents a larger percentage change in this species than does a 10 $\mu g/m^3$ increase in total $PM_{2.5}$ mass. However, of the 9 health outcomes that were associated with sulphate, 8 of these were also associated with total $PM_{2.5}$ mass. If sulphate exerts effects in proportion to its mass in $PM_{2.5}$, the evaluation by Reiss *et al* suggests that sulphate has a weaker effect on mortality and cardiorespiratory endpoints (hospital admissions, mortality) than does total $PM_{2.5}$.

In a large time-series study in the US (75 cities; 4 million deaths), (elemental) sulphur increased the effect of $PM_{2.5}$ mass on daily all-cause and respiratory mortality (Dai et al. 2014). Pooled data from 11 time-series studies suggested that $PM_{2.5}$-sulphate was significantly associated with all-cause mortality (Levy et al. 2012). In another study, among 25 US communities, the daily mortality associated with a 10 $\mu g/m^3$ increase in ambient $PM_{2.5}$ was increased by 0.51% when the $PM_{2.5}$ contained a higher proportion of sulphate (Franklin et al. 2008). Three metals, aluminium, arsenic and nickel, were also associated with increased daily mortality. These results are consistent with the hypothesis that acid sulphates in the air may solubilize transition metals, thus making them bioavailable (Ghio et al. 1999).

When Hong Kong switched to low sulphur fuels in 1999, ambient sulphur dioxide and sulphate levels fell immediately and this change coincided with declines in cardiorespiratory mortality and respiratory symptoms (Hedley et al. 2002). However, as mentioned previously (*Section 7.1 Trace metals*), since ambient concentrations of the transition metals vanadium and nickel were also reduced by the change to low sulphur fuels, improved health outcomes cannot be wholly attributed to decreased ambient sulphate.

Several studies have evaluated the effect of PM-sulphate in children. $PM_{2.5}$-suphate has been associated with decreased lung function and increased respiratory symptoms in children with asthma in the Czech Republic (Peters et al. 1997). That study made comparisons with effect estimates for PM_{10} mass (not $PM_{2.5}$ from which the sulphate was measured) and therefore it cannot be concluded whether sulphate had an effect beyond that of $PM_{2.5}$ particle mass. In another study, among children in the Netherlands during winter, only those children with symptoms of chronic respiratory disease demonstrated significant associations between ambient concentrations of sulphate and decreased lung function and increased respiratory symptoms (van der Zee et al. 1999). However, the observed associations of respiratory outcomes and sulphate were no stronger than the associations with PM_{10} mass. $PM_{2.5}$-sulphate has been associated with decreased lung function and increased respiratory symptoms in children in the US that were free from chronic disease (Schlesinger 2007), however sulphate was highly correlated with $PM_{2.5}$ mass and it is questionable whether these results show an effect that is specific to sulphate.

The health effects of PM-sulphate have also been evaluated in other vulnerable populations. In diabetic patients, $PM_{2.5}$-sulphate had a stronger association with endothelium-dependent (but not endothelium-independent) vascular function (a marker of atherosclerotic change in blood vessels) than did total $PM_{2.5}$ mass (O'Neill et al. 2005). In patients with implanted cardiac pacemakers, $PM_{2.5}$-sulphate had a weaker association with episodes of abnormal heart rate than did $PM_{2.5}$ mass (Dockery et al. 2005). In COPD patients, $PM_{2.5}$-sulphate was not associated with lung function, blood pressure or a change in heart rate whereas $PM_{2.5}$ mass was associated with each of these effects (Ebelt et al. 2005). Overall, there is little evidence that PM-sulphate has health impacts on vulnerable populations beyond the effects associated with exposure to PM as a whole.

Source apportionment studies have provided indirect evidence of health impact of PM-sulphate. In a test of the robustness of source apportionment methods, various investigators used different apportionment methods to estimate the effect on daily mortality of source apportioned $PM_{2.5}$ in Washington, DC (Ito et al. 2006). Among nine $PM_{2.5}$ sources (including traffic, wood smoke and coal), $PM_{2.5}$ designated as "secondary sulphate" was most strongly associated with daily mortality. However *secondary sulphate* $PM_{2.5}$ was the largest constituent of $PM_{2.5}$, accounting for approximately 60% of total $PM_{2.5}$ mass. It is possible that the effect of *secondary sulphate* $PM_{2.5}$ on mortality simply reflected the effect of total $PM_{2.5}$. Indeed, the mortality relative risk estimates were similar for total $PM_{2.5}$ mass and *secondary sulphate* $PM_{2.5}$ (Ito et al. 2006).

Another synthesis of various source apportionment methods found that in Phoenix, Arizona, $PM_{2.5}$-sulphate was associated with daily cardiovascular, but not all-cause, mortality (Mar et al. 2006). The magnitude of effect was similar to that of total $PM_{2.5}$ mass. In a source apportionment study from Barcelona, $PM_{2.5}$ with a high sulphur and ammonia content (designated as *secondary sulphate*) was associated with a higher daily cardiovascular, but not all-cause, mortality risk than was total $PM_{2.5}$ mass (Ostro et al. 2011). Together these results suggest that $PM_{2.5}$-sulphate has cardiovascular health effects. However a study from Atlanta, Georgia, (using a variety of source-apportionment methods) found that $PM_{2.5}$ with a higher proportion of sulphate was strongly associated with hospital emergency department presentations for respiratory disease but not cardiovascular disease (Sarnat et al. 2008). The association between $PM_{2.5}$-sulphate and respiratory disease presentations was stark, with statistically significant associations via three different source apportionment

methods and no significant association between total $PM_{2.5}$ mass and respiratory disease presentations.

Most source apportionment studies have found health effect associations with $PM_{2.5}$-sulphate-however the magnitude of the effect has often been similar to that of total $PM_{2.5}$. Furthermore, these studies are difficult to interpret because the sulphate-related factor in source apportionment includes contributions from other particle constituents.

Evaluations of the effect of long-term exposure to sulphate and mortality have provided unconvincing evidence of an effect, primarily because sulphate in ambient air is highly correlated with ambient $PM_{2.5}$ mass and therefore it is not possible to differentiate the effect of sulphate from that of $PM_{2.5}$ (Reiss et al. 2007). The *Harvard Six Cities* cohort study found that ambient sulphate levels in the six cities were strongly correlated with mortality, although to a similar extent as was total $PM_{2.5}$ (Dockery et al. 1993). The *American Cancer Society* cohort study found ambient sulphate levels to be significantly associated with all-cause, lung cancer and cardiopulmonary mortality (Pope III et al. 1995). $PM_{2.5}$ was similarly associated with all-cause and cardiopulmonary mortality, but not lung cancer mortality. Subsequent analyses of the *American Cancer Society* study with additional years of follow-up have inconsistently associated sulphate with mortality (Schlesinger 2007). Other cohort studies have found no association between long-term exposure to ambient sulphate and mortality (Abbey et al. 1999, Lipfert et al. 2000, Lipfert et al. 2006).

Studies that have evaluated the biological effects of exposing humans and animals to concentrated ambient PM have not consistently identified sulphate content as being significant (Reiss et al. 2007, Schlesinger 2007). Studies have found that exposure to PM-sulphate is associated with suppressed bacterial defence in the lung (Clarke et al. 2000), blood vessel dysfunction (O'Neill et al. 2005) and, increased oxidative stress and blood coagulation (Chuang et al. 2007). However other toxicological studies have failed to provide biological plausibility for the reported health outcomes associated with exposure to sulphate (Schlesinger 2007).

In summary, a number of associations between $PM_{2.5}$-sulphate and health outcomes have been observed, however it is unclear whether these effects can be attributed specifically to sulphate. $PM_{2.5}$ mass is generally highly correlated with $PM_{2.5}$-sulphate and usually the same health outcomes associated with $PM_{2.5}$-sulphate are also associated with total $PM_{2.5}$. In the main, epidemiological and toxicological evidence linking $PM_{2.5}$-sulphate with health effects is inconsistent. Presently, there is some evidence to suggest that exposure to $PM_{2.5}$ that contains sulphate compounds is more detrimental to health than exposure to $PM_{2.5}$ without sulphate. However, it is not clear whether other constituents commonly associated with sulphate $PM_{2.5}$ may be contributing to these health effect-exposure associations.

7.4 Nitrates

As with sulphates, nitrates in PM are primarily formed secondarily in the atmosphere rather than being directly emitted. Nitrate in PM largely originates from nitrous oxide emissions, the largest source of which is on-road vehicles. Generally, nitrate is <10-15% of the mass of PM (Harrison and Yin 2000, Reiss et al. 2007). The western US, with high traffic levels relative to fossil fuel-generated

electricity generation, is an exception, there nitrate levels in PM are comparable to or greater than sulphate levels (Harrison and Yin 2000, Reiss et al. 2007).

Not many studies have addressed the association between PM-nitrate and health outcomes. In two reviews of evidence published in 2007 (Reiss et al. 2007, Schlesinger 2007), a total of four separate epidemiological investigations were cited. Nitrate was significantly associated with all-cause mortality in the Netherlands (Hoek et al. 2000) and California (Lipfert et al. 2006, Ostro et al. 2007) and, cardiovascular mortality in California (Fairley 1999). However, in another US study, nitrate was not significantly associated with all-cause mortality in Atlanta in people ≥65 years of age (Klemm et al. 2004). The study of Hoek *et al* measured aerosol nitrate rather than nitrate as a component of a PM size fraction, therefore it is not possible to directly compare the strength of the association of nitrate with that of PM mass (mortality risk estimates were marginally higher for nitrate than for PM_{10}). Of the other studies with significant mortality associations, one study found a stronger association with $PM_{2.5}$-nitrate than with total $PM_{2.5}$ (Lipfert et al. 2006). In the other two studies (Fairley 1999, Ostro et al. 2007), the association between $PM_{2.5}$-nitrate and mortality was the same or weaker than the association for total $PM_{2.5}$ mass.

Since those reviews, source apportionment studies from the US have found that $PM_{2.5}$-nitrate was not associated with daily all-cause and cardiovascular mortality or, hospital emergency department presentations for cardiorespiratory disease (Ito et al. 2006, Sarnat et al. 2008). In contrast, a source apportionment study from Barcelona found that $PM_{2.5}$-nitrate was positively associated with cardiovascular mortality and to a greater extent than was total $PM_{2.5}$ mass (Ostro et al. 2011). An analysis of a data set of 12.5 million Medicare enrolees (≥ 65 years of age) found that long-term exposure to increased $PM_{2.5}$-nitrate (a one-standard deviation increase in the 7-year average) was associated with a 1.2% increase in mortality (Chung et al. 2015). In that study, increased long-term $PM_{2.5}$-sulphate was associated with a decrease in mortality.

An examination of the effects of various components of $PM_{2.5}$ on cardiovascular mortality and hospitalisations in New York found that $PM_{2.5}$-nitrate was associated with cardiovascular hospitalisations but not mortality, while total $PM_{2.5}$ mass was associated with both outcomes (Ito et al. 2011). In another study of 25 US communities, $PM_{2.5}$-nitrate was not associated with all-cause mortality (Franklin et al. 2008). In London, $PM_{2.5}$-nitrate was positively associated with daily respiratory, but not all-cause or cardiovascular mortality (Atkinson et al. 2010). Cardiovascular and respiratory hospitalisations for persons 65 years or older in 106 US counties were not associated with $PM_{2.5}$-nitrate, although for cardiovascular hospitalisations the positive risk estimate was greater than for the majority of the other 19 constituents of $PM_{2.5}$ that were measured (Bell et al. 2009). In an analysis which utilised US Medicare billing claims in order to determine hospital admissions for a population of 12 million people from 119 US counties, it was demonstrated that hospital admissions for cardiovascular disease, but not respiratory disease, were positively associated with $PM_{2.5}$-nitrate (Levy et al. 2012). In a meta-analysis of estimates of effect from four time-series studies, a 10 $\mu g/m^3$ increase in $PM_{2.5}$-nitrate was associated with a non-statistically significant 2.7% increase in all-cause mortality (Levy et al. 2012). Long-term exposure to $PM_{2.5}$-nitrate has been associated with all-cause and cardiorespiratory mortality in female teachers in California, but not to a greater extent than was long-term exposure to total $PM_{2.5}$ (Ostro et al. 2010).

There have been few toxicological studies on which to evaluate the health effects of PM-nitrate. Generally toxicological studies have involved exposure to nitrated compounds rather than nitrate bound to PM, with mixed results (Schlesinger 2007).

In summary, the evidence base for health impacts associated with exposure to PM-nitrate is limited. As with sulphate, the evidence is difficult to interpret because $PM_{2.5}$-nitrate concentrations are highly correlated with total $PM_{2.5}$ mass, making it difficult to determine whether nitrate or whole particle mass is responsible for observed health outcomes. There is more evidence linking PM-nitrate with cardiovascular than respiratory outcomes.

7.5 Summary

Epidemiological studies that portray regional heterogeneity in the health effects of exposure to PM and, toxicological studies that demonstrate compositional variability in PM toxicity, strongly suggest that particle composition influences the health effects of PM. Thus it is not solely the mass concentration of ambient PM that determines the biological (and consequently health) response to exposure. It is unlikely that any single chemical component of PM is responsible for all adverse health outcomes. Rather, an array of elements/compounds may be associated with specific outcomes. Most of the evidence linking compositional elements of PM with health effects is inconsistent and conflicting. Although there are constituent candidates for causing adverse health effects (*e.g.* the transition metals vanadium and nickel), there is presently insufficient evidence to infer that a particular particle (or population of particles) will have a greater impact on health than other particles on the basis of composition alone.

- Some metals in PM, such as vanadium and nickel, are associated with adverse health effects but due to the complexity of PM composition it is not known whether it is specifically the metals in ambient PM which are responsible for adverse health outcomes.
- There are biologically plausible mechanisms for the adverse health effects associated with the inhalation of transition metals.
- Organic compounds in PM, particularly those emitted during the process of fossil fuel combustion, are likely to cause health effects, however it is presently unclear what the health impacts of the inhalation of organic compounds in ambient PM are.
- Health outcomes have been associated with both sulphate and nitrate in PM but there is no clear evidence that the presence of these constituents increases the toxicity of ambient PM over and above total $PM_{2.5}$ mass.

8. Future directions to address the health impact of PM emissions in NSW

The study results presented in this report can broadly be divided into two lines of evidence that can be used to guide policy:

1. **Evidence relating health effects to exposure to PM from specific emission sources.** A particular focus of this review was to evaluate the evidence pertaining to the health impacts of source-specific PM air pollution relevant to NSW. Therefore the majority of this report relates to this evidence. This evidence may be used to guide policies that aim to provide population health benefits through the reduction of air pollution emissions from specific types of industries or human activities.
2. **Evidence relating health effects to exposure to ambient particle mass concentration and, PM of specific size or chemical composition.** This evidence may be used to set ambient air quality standards. Given that the chemical composition of PM is partially dependent upon emission sources, this evidence may also guide source-specific emission reduction policies.

These two types of evidence are considered under *Chapter 8.1: What evidence is there to guide future emissions reduction policies in NSW?*

Sources of evidence

As this report has detailed, evidence of associations between exposure to PM and health impacts comes from a variety of different types of scientific investigation. These types of scientific investigations are:

- **Epidemiological studies**, where the health impact of exposure to ambient air PM is assessed in the population as a whole or in a segment of the population, such as in specific age groups or people with chronic diseases;
- **Controlled human exposure (or chamber) studies**, where volunteers (usually healthy individuals) are exposed to PM in air within a sealed chamber for a period of minutes to hours at a concentration that is at usual ambient concentrations or marginally higher;
- **Animal or cell culture toxicological studies**, where animals or cell cultures (human, animal or bacterial) are exposed to PM in concentrations that far exceed ambient concentrations. Such studies investigate biological mechanistic processes that help to explain or support the potential for health impacts.

Evidence is most robust when all study types provide evidence of consistent and biologically plausible health effects. The pros and cons of applying the evidence from these study types to guide policy are shown in *Table 8.1*. By considering evidence across all study types, the strengths and weaknesses of different studies are integrated and weaknesses from one study type are nullified by advantages from another. Although all evidence should be considered when developing a policy

response, this report is heavily weighted with evidence from epidemiological studies because epidemiological studies are conducted within the community (the population that policy serves to protect) at ambient air pollution concentrations, and is therefore directly relevant to policy development.

Table 8.1 Pros and cons of evidence from different study types

Study type	Pros	Cons
Epidemiological	• health and exposure measured as people go about their daily lives and therefore directly relevant to environmental health and air quality policy	• confounders of effect may obscure the true reason for measured health outcomes • exposure misclassification – individuals with detrimental health outcomes may not have been exposed to worse air quality than individuals with better health or vice versa
Controlled human exposure **(chamber study)**	• can be certain of exposure • can exclude most confounders, therefore are confident the response *is* due to the exposure • can measure health/biological response in great detail	• conducted in a controlled environment (not a real life situation) • usually conducted on healthy and fit volunteers whereas air pollution inordinately affects the young, old and ill • exposure over a relatively short time
Toxicological **(animal studies or cell culture)**	• can determine the biological mechanism behind an exposure-health response • can determine the potential for toxicity of PM constituents that may have very low ambient levels	• responses of animals and cells may not be applicable to humans • PM exposure concentration is usually an order of magnitude greater than ambient concentrations

Within epidemiological studies there are a variety of different study methods. Each method is designed to answer a different question in relation to the health impact of PM (*Table 8.2*). There are limitations with all epidemiological studies. Assuming that a study is well designed and conducted, the degree of confidence that the evidence can be used to inform policy is, among other things, dependent on the limitations associated with the study type. Robust evidence also requires verification of previous study results. For these reasons, policy development should be based upon an integration of results from several studies.

Table 8.2 Epidemiological studies used to investigate the health impact of PM air pollution

Study	Question and PM characteristic addressed	Limitations
Source apportionment applied to health outcome data	What are the health impacts of exposure to source-specific PM emissions? **Emission source**	Inherent uncertainties in assigning ambient air PM to a specific emission source
Community-based cohort (small population) (*e.g.* population living near an emission source)	What are the health impacts of living near a PM emission source (*e.g.* mine, roadway) **Emission source**	Exposure near an emission source can vary considerably depending on personal daily activities and the type of emission Living in a particular area is associated with many other variables that can impact health (*e.g.* socioeconomic status, other environmental pollutants) Individuals whose health is impacted may migrate out of the community and not be accounted for in the study
Time-series	What are the health impacts of short-term exposure to increases in ambient PM? **Temporal emission sources** **(Short-term) ambient PM mass concentration** **Particle size**	Daily changes in health outcomes can be caused by many factors that change on a daily basis
Intervention evaluation (*e.g.* mine closure, implementation of fuel regulations)	What were the health impacts of a change in PM emissions? **Emission source**	A variety of environmental and social changes often accompany an intervention, all of which may impact health
Cohort (large population)	What are the health impacts of long-term exposure to ambient air PM? **Ambient PM mass concentration** **Particle size**	Many confounding factors will affect the health of populations over a long period of time, not all of these can be accounted for in study design or analysis
Chemical speciation	What are the health impacts of exposure to specific chemical components of PM? **Chemical composition**	Inherent uncertainty using fixed-site monitoring to estimate exposure to chemical constituents of PM that are atmospherically dynamic

8.1 What evidence is there to guide future PM emissions reduction policies in NSW?

8.1.1 Evidence relating health effects to exposure to PM from specific emission sources

Source apportionment applied to health outcome data

The methods used to apportion PM measured in ambient air to different sources are still evolving and it is a relatively new area of study. Due to the dynamic nature of atmospheric processes that affect the chemical and physical characteristics of emitted PM, source apportionment models are associated with an inherent amount of uncertainty. Furthermore, attribution of the identified emission profiles (or 'factors') to specific sources requires judgement and, therefore, includes inherent subjective uncertainty. This uncertainty is added to the uncertainty associated with the use of fixed-site monitors to estimate human exposure. Hence overall, the findings of studies linking specific sources to human health effects using source apportionment need to be interpreted with some caution.

Despite these limitations, source apportionment is a way of estimating the health impact of exposure to PM from a specific source in contrast to the total ambient PM mixture comprising PM from multiple sources.

Source apportionment studies suggest:

- **There is strong evidence that exposure to ambient PM from combustion emissions (particularly coal-fired power stations, diesel and, on-road vehicle emissions) is associated with adverse health outcomes.**

Community-based cohort and cross-sectional studies

The health effects of PM emissions from coal mines and on-road vehicles have been investigated in communities that live close to these emission sources. A major limitation of these studies is that living in a particular location is invariably associated with a variety of factors that impact health and it can be difficult to find a directly comparable population with which to compare health outcomes. Nevertheless, these cohort and cross-sectional studies add to the evidence from source apportionment studies.

Community-based cohort studies show that:

- **living in close proximity to major roadways is associated with adverse health outcomes that are likely to be partially attributable to exposure to on-road vehicle PM emissions;**
- **living in close proximity to coal mines has been associated with adverse health outcomes however there is insufficient evidence (due to study limitations and the small number of studies) to conclude that these health outcomes are specifically related to exposure to coal dust.**

Time-series studies

Time-series studies have investigated daily health outcomes associated with short-term exposure to increased ambient PM levels during bushfires, severe dust storms and periods of high use of wood-fuelled domestic heating. Many factors that affect population health change on a daily basis (*e.g.* air temperature) however, properly controlled time-series studies can show strong evidence of associations between ambient PM and daily health outcomes.

Time-series studies show that:

- **exposure to increased ambient PM mass during severe dust storms and bushfires is associated with adverse health effects, particularly in people with chronic respiratory disease;**
- **exposure to ambient PM from wood-fired domestic heating is associated with an increase in respiratory disease symptoms and adverse respiratory events.**

Intervention evaluation

There have been very few evaluations of the health impacts of interventions that have changed source-specific PM emissions. Interventions that have been evaluated include: a ban on coal sales in Dublin (Clancy et al. 2002), re-opening of a steel mill in Utah (Pope III 1989) and, reductions in the sulphur content of fuel oil for power stations and vehicles in Hong Kong (Hedley et al. 2002).

Evaluations of interventions that have changed source-specific PM emissions suggest that:

- **exposures to PM emissions from combustion sources (domestic coal combustion, industrial combustion, power stations and vehicles) are associated with increased cardiovascular and respiratory mortality and morbidity.**

Controlled human exposure (chamber) and toxicological studies

Controlled human exposure (chamber) and animal/cellular toxicological studies have been used to examine the biological effects of a variety of PM emissions, particularly diesel exhaust and wood smoke.

Controlled human exposure and animal/cellular toxicological studies demonstrate that:

- **exposure to diesel exhaust PM results in a variety of cardiovascular, respiratory, carcinogenic and developmental biological effects, many of which are consistent with the health effects associated with occupational exposure to diesel exhaust;**
- **exposure to wood smoke PM results in compromised lung immune defence, oxidative stress responses and inflammation, effects that are consistent with adverse respiratory health endpoints in epidemiological studies.**

8.1.2 Evidence relating health effects to exposure to ambient particle mass concentration and, PM of specific size or chemical composition

Particle mass concentration

There is strong evidence from many large cohort studies and time-series studies that the ambient concentration of PM mass is associated with adverse health outcomes.

- **Long-term and short-term exposure to ambient PM mass is associated with adverse health outcomes, primarily related to respiratory and cardiovascular diseases but also including lung cancer, birth outcomes and developmental outcomes.**

There is limited evidence related to the health impacts of low concentrations (<8 µg/m^3) of ambient PM. There is no evidence of a concentration threshold below which ambient PM has no health effects, suggesting that improvements in population health will continue to occur as ambient mass concentrations of PM are reduced to "background levels".

Particle size

There is strong evidence from cohort, time-series and toxicological studies that particle size influences the health effects associated with exposure to PM.

- **There is evidence that smaller particles have a greater impact on health than larger particles however some studies have indicated that larger (coarse) particles may preferentially affect the airways and lungs and cause more respiratory health effects than smaller particles.**

- Particle size is the basis on which PM is classified and has defined the study of exposure-response functions however **particle composition is also important with regards to the health effects of PM.**

Particle chemical composition

Ambient PM is recognised as a "mix" of many particles, with different compositions. The composition of PM is thought to influence the health impact of exposure.

- **There is evidence that metals and organic compounds within PM have toxic effects.**

- **There is evidence of health effects associated with sulphates and nitrates within PM but this evidence is variable and generally effects are not greater than those associated with total $PM_{2.5}$ mass.**

- **Organic compounds in PM, particularly those emitted during the combustion of fossil fuels, are likely to cause health effects. However, the specific health impacts of organic compounds are unclear.**

The evidence relating health effects to exposure of ambient concentrations of specific constituents of PM is still emerging. **Presently, it is appropriate to continue to base emission reduction policies on minimising ambient particle mass rather than specific particle constituents**.

8.2 Which sources of PM should be targeted to maximise health benefits?

Exposure to PM from a variety of emission sources has been associated with adverse health outcomes. However, there is **inconsistent evidence that the health impact of PM from any specific source is greater than the health impact from exposure to total ambient PM mass**. Notwithstanding these inconsistencies, there is evidence that the health impact of PM from combustion sources may be greater than the health impact of PM from other sources (Cooke et al. 2007, Grahame and Schlesinger 2007, Janssen et al. 2011). In urban areas, on-road vehicle exhaust is a major source of combustion related PM. Other sources of combustion particles that contribute to ambient PM include: wood-fired domestic heating, off-road diesel exhaust and coal-fired power stations.

The sum of evidence of health effects associated with exposure to PM from combustion sources is considerable (Grahame et al. 2014), and substantially greater than for non-combustion sources. This is largely a consequence of many more investigations of combustion related PM. The number of studies that have directly compared the health effects of exposure to PM from combustion sources with exposure to PM from non-combustion sources is relatively small. However, the indication is that health effects are more likely to be associated with exposure to PM from combustion than from other emission sources.

Presently there is insufficient health effects evidence to conclude that there will be significant benefit from basing emission reduction policies on specific emission sources in NSW, in preference to minimising total ambient PM exposures via emissions reductions.

The limitations of studies in determining the health effects of exposure to ambient PM from specific sources have been outlined in this report. These limitations mean that it is very difficult to categorically determine that certain health effects are a consequence of a source-specific exposure. Some of the strongest evidence of health effects associated with source-specific exposures can come from the evaluation of the effects on health of interventions that reduce emissions. Thus, it can be prudent to intervene and reduce certain emissions based on the limited evidence that is available and to later assess whether these interventions have resulted in expected health benefits. Current evidence suggests that actions that reduce PM emissions from combustion sources are likely to have population health benefits. At the very least, it would be prudent to monitor community exposure to particulates from combustion sources and evaluate the health impacts of this exposure.

On current evidence, if a source of PM is to be targeted for emissions reduction policies, it should be on the basis of:

1. the source is a large contributor to ambient PM mass in the community and therefore regulation specific to the source will help to significantly reduce exposure to total ambient PM;
2. the source of PM emissions occurs in close proximity to people and therefore it could be expected that the emission source is responsible for considerable exposure to PM; or,
3. the source of PM is a combustion source and therefore the health benefits from exposure reduction are likely to be significant.

This report has not evaluated exposures of the NSW population to source-specific PM nor assessed risks to the population of NSW. Nevertheless, on-road vehicles are likely to be a significant source of PM exposure in NSW given the proximity of much of the population to high traffic density. There are likely to be significant health benefits from minimising this exposure.

Although there may be insufficient evidence from the scientific literature to support health effects being associated with exposure to particles from a specific emission source, if that emission source substantially increases the ambient PM mass concentration to which a community is exposed this is cause for concern. For example, if coal dust from mining operations significantly increased the ambient PM mass concentration to which a community was exposed then efforts should be made to reduce this exposure, regardless of what is, and is not, known about the health effects specific to coal dust exposure.

8.3 How can we address the knowledge gaps related to the health impacts of source-specific PM relevant to NSW?

Current knowledge does not allow precise quantification or a definitive ranking of the health impact of PM emissions from different sources. This deficiency can only be addressed by epidemiological studies designed to specifically examine source-specific PM exposure and health outcome associations, rather than focusing solely on ambient particle mass. To provide a solid evidence base on which to develop policy, findings from epidemiological studies should ideally be supported by studies of the biological effects of controlled exposures to source-specific PM.

The majority of evidence of the health effects of exposure to source-specific PM has been obtained from studies conducted elsewhere (outside of NSW and outside of Australia). There are regional differences in PM emission sources and population characteristics that may influence the effects of exposures. For example, vehicle emission standards in NSW may differ from those standards applying to past epidemiological investigations conducted elsewhere. Coal mining practices in NSW, as well as the coal itself, differ from other countries where the health impact of coal mining has been investigated. It is important that some of the evidence that is to be used to develop policies to protect the health of the population of NSW is obtained from studies conducted in NSW or Australia. International evidence should not be dismissed, but rather used in conjunction with local evidence to inform policy development.

To address the knowledge gaps related to the health impacts of source-specific PM relevant to NSW, requires:

- accurate characterisation of population exposure to source-specific PM in NSW; and
- determination of the effect of source-specific PM exposures on health outcomes in NSW.

Accurate characterisation of population exposure to source-specific PM in NSW

Accurate characterisation of population exposure to source-specific PM is a prerequisite for more informative studies of the health effects of exposure. Emissions inventories provide information on the relative contributions of different sources to total ambient PM but provide no evidence of actual exposures. Characterisation of population exposure to source-specific PM should entail:

- Ambient and strategic (for communities close to emission source) monitoring of the chemical constituents of PM (source speciation) in different regions;

- Improving the spatial resolution of exposure estimates whenever possible by increasing the number of sites monitoring chemical constituents of ambient PM, particularly constituents in combustion emissions;

- Source apportionment modelling with a NSW emissions context. Emission sources that might be considered less of a concern (based on current evidence), such as crustal dust and sea salt, should be included in these models;

- Monitoring particle size that is emission source-relevant. For example, it has been proposed that PM_1 (<1 µm) measurements provide better information about contributions to ambient PM from combustion processes than does monitoring of $PM_{2.5}$ (Morawska et al. 2008a). Motor vehicles are a major emitter of $PM_{0.1}$, which could be monitored near roadways. Since $PM_{0.1}$ has negligible mass, measurement of particle number would be a more useful unit of measure.

Determination of the effect of source-specific PM exposure on health outcomes

Characterisation of population exposure to source-specific PM can be used to address knowledge gaps in the health impacts of source-specific PM by:

- Applying source apportionment modelling to routinely collected health outcome data such as mortality and hospital admissions to determine exposure-health outcome associations;

- Using source apportionment data collected regularly over considerable time to estimate the health effects of long-term exposure;

- Using estimates of exposure from strategic monitoring of PM emissions (*e.g.* PM emitted from roadways) in studies of the health of populations that live close to emission sources.

Estimates of the health effects of exposure obtained from these studies should be expressed relative to the effect of total PM mass in order to determine whether reducing a source-specific PM emission is likely to result in more health benefit than minimising overall emissions of total PM mass.

Source-specific PM emissions in NSW that should be the focus of future health effects research

The evidence presented in this report shows that the magnitude and types of health effects resulting from exposure to source-specific PM are not fully understood. Thus, well conducted investigations of the health effects of any source-specific PM are likely to generate valuable new knowledge. However, the number of different emission sources is vast and studies of the health effects of ambient PM are expensive and resource intensive. Thus, there should be a focus on investigations of emission sources where new knowledge has the potential to provide the greatest health benefit in NSW. These are emission sources where current evidence demonstrates the potential for significant adverse health impacts from exposure and yet the evidence has provided uncertain estimates of effect. Emissions sources in NSW that fit these criteria are coal dust and combustion emissions. PM combustion emissions of particular concern in NSW based on emissions inventories and/or the potential for exposure are: coal-fired power stations, on-road vehicles and domestic wood-fired heating.

- **Coal dust** – There is significant evidence from studies of occupational exposure that inhalation of coal dust can have major impacts on health. Thus coal dust may pose a significant hazard to communities surrounding coal mining, coal transport and coal processing operations in NSW. Unfortunately most investigations of the health impacts of coal mining on surrounding communities, due to limitations of study methods, have provided poor quality evidence of the presence or absence of health effects associated with exposure to coal dust. If health studies are to be conducted of populations surrounding coal mining operations in NSW, they need to be appropriately powered to enable an effect to be detected if it truly exists, and should entail exposure assessment and health outcomes measurement with adjustment for potentially confounding factors. This may be difficult with current population sizes of communities living around NSW coal mines.
- **Coal-fired power stations** – There is evidence that exposure to PM that contains sulphate is detrimental to health. One of the major sources of these particles is emissions from coal-fired power stations.
- **On-road vehicles** – There is significant evidence that exposure to both exhaust and non-exhaust PM emissions from traffic are associated with adverse health effects. A significant proportion of the population of NSW is exposed to these emissions.
- **Domestic wood-fired heating** – The health impact of outdoor exposure to PM emitted from wood-fired heating in homes is unclear as this has not been extensively studied. There is evidence that in communities in which there is a high use of wood heaters, the respiratory health of residents can be impacted. Thus, there is potential for some populations in NSW to be impacted by these PM emissions.

Other considerations for addressing knowledge gaps

- Using personal exposure estimates (in combination with stationary monitoring data) in epidemiological cohort studies. It is acknowledged that personal monitoring can be difficult and costly to implement in epidemiological studies.
- Models which estimate individual exposures (*e.g.* land-use regression models or validated dispersion models) should be considered for determining estimates of exposure.
- Given that there is strong evidence of associations between exposure to PM from combustion sources and health effects and, $PM_{0.1}$ are a major component of combustion emissions, consideration should be given to project monitoring of $PM_{0.1}$ and modelling exposure near combustion sources in order to determine concentration-effect relationships.
- Conduct controlled human exposure (chamber) studies and animal toxicological studies to provide biological mechanistic support for exposure-health outcome associations observed in epidemiological investigations.
- Evaluate changes in population health from reductions/interventions in source-specific PM emissions.

8.4 What does the evidence suggest about the adequacy of current PM standards in NSW?

Table 8.4.1 Current PM standards in NSW

Standard	Averaging period	Maximum concentration	Goal
PM_{10} standard	1 day	50 µg/m^3	5 exceedances allowable per year
$PM_{2.5}$ advisory reporting standard	1 day 1 year	25 µg/m^3 8 µg/m^3	Gather data

The adequacy of current PM standards and possible courses of action

- Current evidence suggests that standards should continue to be set on the basis of total particle mass (speciation of PM is required to address knowledge gaps but should not presently be used to set standards).
- Health effects are associated with long-term exposure to PM_{10} and therefore an annual standard (1 year averaging period) is appropriate.
- No lower threshold of effect has been observed for both short- and long-term exposure to ambient $PM_{2.5}$. The $PM_{2.5}$ advisory reporting standards should become compliance standards with a goal of reducing ambient $PM_{2.5}$ to as low as practically possible. Furthermore, given that ambient PM concentrations in NSW are low relative to many other

areas of the world, exposure-response relationships for low concentrations of ambient PM mass could be examined in NSW.

- Evidence suggests that PM from combustion sources is more detrimental to health than PM from other sources. Black carbon is a measure of particles from combustion sources. The particles in black carbon can come from any combustion source (motor vehicles, diesel engines, coal-fired power station) and therefore from a regulatory standpoint there does not appear to be any advantage in monitoring black carbon on a routine basis. However, targeted monitoring of black carbon to address knowledge gaps would be useful.
- Health effects associated with exposure to coarse PM ($PM_{10-2.5}$) are different to the health effects observed with exposure to either PM_{10} or $PM_{2.5}$. Consideration should be given to monitoring ambient concentrations of coarse PM, possibly instead of PM_{10}.
- Ambient monitoring should be conducted with a view to providing epidemiological evidence. Therefore, monitoring should occur where people are exposed and also be used to examine spatial patterns of PM (and other pollutant) distributions.

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