Literature Review and Report on Potential Health Impacts of Exposure to Crustal Material in Port Hedland

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Executive Summary

Potential Health Impacts of Exposure to Crustal Material in Port Hedland

1. Introduction

The purpose of this literature review and report was to assist in the process of identifying whether there are potential health risks to the residents of Port Hedland from airborne particulate matter. The intention was to provide a reference document on the relevant issues for anyone involved or concerned about air quality standards and their application to Port Hedland. It was anticipated that by assessing the composition of the airborne dust, the concentration and relative toxicity of the dust constituents and the size of dust particles, and by comparing this information with data about the hazards from urban air particle pollution in large cities, a judgement could be made about the level of exposure that may be safely tolerated within the community.

This report reviews the available scientific information about the hazards associated with the various components of the airborne dust in Port Hedland and assesses them in the context of what is known about urban air particulate pollution. The document aims to provide sufficient information to address three key questions:

a) What are acceptable levels of particulate matter in Port Hedland, and should these be measured as average and/or peak levels?

b) What type of monitoring is appropriate for Port Hedland, to provide information on potential health effects?

c) What methods should be used to measure particulate matter, and what corrections need to be made to allow for Port Hedland conditions and/or other environmental factors?

2. Mineralogy of airborne particulate matter in Port Hedland

Port Hedland (population approximately 4,000) is situated virtually beside the BHP Billiton Iron Ore (BHPBIO) processing and loading facility at Nelson Point. BHPBIO ships approximately 80 – 100 million tonnes per annum (MTPA) of iron ore from Port Hedland. The Port Hedland Port Authority wharf ships salt (3 MTPA), manganese (0.5 MTPA), as well as small amounts of feldspar (0.05 MTPA) and copper concentrate (0.024 MTPA proposed). These materials are stockpiled close to residential and commercial areas. The town is primarily impacted by fugitive iron ore dusts, but is naturally dusty due to its semi-arid

climate (dry, hot and windy) and environmental factors. Regional winds also cause salt and crustal dust to become airborne and contribute to dust exposure in Port Hedland residents. There are no significant sources of combustion particles apart from a waste incinerator, 8 km directly south of the town. There are also very low levels of oxides of sulphur and nitrogen plus ozone at Port Hedland.

The mineral composition of iron ore from the Pilbara region is typically 93% iron oxides, 4% quartz as chert and 3% clays (aluminosilicates and iron-based clays). Consequently, the airborne dust arising from iron ore handling at Port Hedland consists mostly of iron oxides (haematite and goethite) as well as clays (aluminosilicates and iron silicates) and quartz. Manganese oxides, copper ore minerals, feldspar and salt are likely to represent minor components of airborne particulate matter in Port Hedland, although transport and ship loading activities and different weather patterns may impact on daily exposure levels. Concentrations of combustion particles derived from vehicle emissions and other combustion sources would be expected to be lower in Port Hedland than in large cities. An analysis of the elemental composition of a sample of airborne dust collected over a week at the Port Hedland wharf indicated an iron content of > 500g/kg, while the levels of sodium, magnesium, aluminium, calcium, manganese and copper were approximately 1g/kg. However, these could vary on a day to day basis depending on loading activities and weather patterns.

3. Particle behaviour and measurement

The effects of airborne particulate matter (PM) on health are determined by the inherent toxicity of particles and their pattern of deposition in and removal from the respiratory tract. Particles deposited on all but the most distal airways are removed rapidly (within 24 h) by the mucociliary escalator, although a substantial proportion of particles deposited on small ciliated airways are retained for more than 24 h. Particles that penetrate distally into the respiratory bronchioles and alveoli are cleared much less efficiently.

The terminology of biologically relevant fractions of particulates has been standardized: the *respirable fraction* is the mass fraction of inhaled particles penetrating to the unciliated lung parenchyma; the PM_{10} fraction is defined as particles which pass through a size selective inlet with a 50% efficiency cut-off at 10 μm aerodynamic diameter. Similarly $PM_{2.5}$ have been defined as particles which pass through a size selective inlet with a 50% efficiency cut-off at 2.5 μm aerodynamic diameter. The $PM_{2.5}$ fraction is alternatively called the *fine particle*

fraction; $PM_{10-2.5}$ is termed the *coarse particle fraction;* $PM_{0.1}$ is termed the *ultrafine (UF) particle fraction.* A large variety of instruments is used for measuring PM including many types of fixed location devices such as filter-based gravimetric samplers, reflectometers, direct-reading instruments for continuous measurements (TEOM and β-attenuation analyzers), optical analyzers, dichotomous samplers and devices for measuring particle counts. Portable devices for measuring personal ambient exposures to PM are increasingly being used.

4. Concentrations of airborne particulate matter in Port Hedland

From 1996, BHPBIO have made regular measurements at the Boodarie monitoring site using $PM_{10}/PM_{2.5}$ Tapered Element Oscillating Microbalance (TEOM) samplers. The 24-hour average PM_{10} concentrations at the Boodarie monitoring site ranged from 20 to 175 $\mu g/m^3$ over the four years from 1997-2000. Median annual PM_{10} concentrations were less than 25 $\mu g/m^3$. For $PM_{2.5}$, the minimum 24-hour average was approximately 10 $\mu g/m^3$ and the maximum concentration was about 42 $\mu g/m^3$. There were clear seasonal trends with the highest levels of airborne particulates occurring between October and March. The Ambient Air Quality National Environmental Protection Measure (AAQ-NEPM) standard for PM_{10} (50 $\mu g/m^3$, averaged over 24 hours, with no more than five exceedences of this value at a specific location each year) was exceeded between 8 and 16 times at Boodarie in each of the four years from 1997-2000. However, these data are clearly of limited relevance to ambient dust exposure levels at Port Hedland, given the distance of the Boodarie monitoring site from the town. There appear to be no systematic monitoring data from other independent sources.

A source of airborne particulate matter peculiar to Port Hedland is biomass burning as a result of wildfires started by lightning strikes. During one such event > 300 km from the Boodarie monitoring station, the maximum 24-hour PM_{10} concentration was 150 $\mu g/m^3$ over a four-day period. However, $PM_{2.5}$ comprised an unexpectedly small fraction (20 to 30%) of the PM_{10} aerosol measured during such fire events.

Currently available data on ambient particulate matter concentrations and composition in Port Hedland are insufficient for delineating community exposure levels. Prospective monitoring based on measurements at multiple sites within the township, as well as the use of devices that monitor personal exposure is recommended.

5. Health effects of exposure to crustal dust

Exposure to airborne quartz carries the risk of silicosis, but only with prolonged exposure to concentrations of > 200 μg/m^3. Exposure to airborne non-fibrous silicates, including coal mine dust, is associated with pneumoconiosis but only at very high concentrations seen in industrial settings. There appears to be an association between lung cancer and exposure to high concentrations of quartz or cristobalite dusts in occupational settings. It is unclear whether co-existing silicosis is a pre-requisite for carcinogenesis in this setting. Sufficient exposure to iron ore dust can cause pneumoconiosis, but this is believed to be primarily due to the quartz component. Iron oxide *per se* is relatively non-toxic in this regard. Haematite mining has been variably associated with a heightened risk of lung cancer. However this can probably be attributed to concomitant exposure to radioactivity during the mining of haematite rather than to the iron oxide *per se*.

Prolonged exposure to high concentrations of manganese in occupational settings is associated with neurotoxicity. Long-term exposure to average manganese concentrations > 200 μg/m^3 in respirable dust may adversely affect performance in neurobehavioral tests. No effects have been associated with workplace respirable dust manganese concentrations of 40–80 μg/m^3. There is no clear evidence of neurotoxicity from exposure to lower concentrations of airborne manganese in community settings. Manganese in combination with other metals may play a role in the toxic effects of particulate matter in urban air, but there is no evidence that manganese is of particular importance as opposed to other transition metals. It is unlikely that exposure to airborne manganese and copper ore is associated with an increased risk of cancer.

Exposure to dust comprising particles between 1 and 50 μm in diameter and to iron oxides within such dust may result in so-called nuisance effects, including minor or self-limiting irritation of the eyes, upper respiratory tract and/or skin.

6. Effects of urban particulate matter on cardiorespiratory health

Composition of particulate matter

The major components of airborne particulate matter are sulphate, nitrate, chloride, elemental and organic carbon, crustal material and biological material. Trace elements usually constitute less than 1% of total particle mass. In dry climates crustal material may be an important component of PM, while in urban areas SO_2, NO_2 and organic and elemental carbon, derived

from fossil fuel combustion and motor vehicle exhausts, are major components of PM. The composition of PM is influenced by geographical, meteorological and anthropogenic factors, including the degree of urbanization, industrial and agricultural activities, and the type and volume of vehicular traffic. Seasonal influences include the combustion of fossil fuels for heating, long-range transport of crustal material from desert areas in seasonal dust storms, increased photochemical oxidation during summer and seasonal burning of biomass. Motor vehicle emissions are the primary sources of ultrafine particles in urban areas, with limited data indicating that organic and elemental carbon is the major component of this size fraction.

Epidemiological studies of the health effects of urban particulate matter

Studies conducted at different locations worldwide indicate an association between ambient PM and mortality. In some locations there is evidence for an independent effect of coarse PM on short-term mortality, although many studies that have examined the effects of crustal or windblown particles have not identified a significant association with mortality. There is also little or no evidence that long-term exposure to coarse PM is related to increased mortality. In urban areas, there is stronger evidence that exposure to fine PM derived from anthropogenic sources, including fossil fuel combustion and industrial sources, might be associated with increased short-term mortality. Long-term exposure to fine PM also appears to be associated with increased mortality. It has been estimated that globally, $PM_{2.5}$ air pollution (primarily urban) causes ~3% of mortality from cardiopulmonary disease, ~5% of mortality from cancer of the trachea, bronchus and lung, and ~1% of mortality from respiratory infections in children < 5 years of age.

In many studies of hospital admissions for respiratory diseases, including asthma and COPD, the effect of exposure to coarse PM has been found to be as strong or stronger than that of fine PM. Coarse PM may induce adverse lung responses and trigger processes that result in an increased likelihood of hospitalization. Ambient PM levels appear to be consistently associated with increased asthma admissions among children, and with COPD admissions among the elderly. There is also a considerable body of evidence supporting an association between ambient coarse PM and cardiovascular hospital admissions.

Fine PM appears to have a stronger and more consistent association with reduction in lung function than coarse PM. Long-term exposure to $PM_{2.5}$ appears to adversely affect the development of lung function among teenagers. However, many studies have shown

significant associations between exposure to coarse PM and the incidence of respiratory symptoms in both healthy subjects and those with lung disease. Studies have also shown that exposure to PM, both coarse and fine, increases the likelihood of asthma symptoms, chronic bronchitis and cough among asthmatic children. However, adults with asthma are much less sensitive to the effects of increased levels of PM than children. Evidence that traffic-related pollutants contribute to the risk of asthma symptoms among adults is weak. Increased levels of PM are associated with higher rates of diagnosis of chronic bronchitis and emphysema. High levels of PM are associated with increased breathlessness and mucus production, and adverse effects on lung function in patients with COPD. Occupational exposure to various inorganic dusts is associated with the development and morbidity of COPD, independent of the effects of smoking. $PM_{2.5}$ or carcinogenic substances associated with fine PM may be causally related to the development of some lung cancers, either alone or in combination with tobacco smoke.

There is now a substantial body of evidence linking exposure to ambient PM with cardiovascular disease. Exposure to fine PM increases the risk for cardiovascular mortality by inducing pulmonary and systemic inflammation, accelerating atherosclerosis and altering cardiac autonomic function. Acute exposure to increased concentrations of fine PM may transiently elevate the risk of myocardial infarction. Exposure to $PM_{2.5}$ is also associated with a reduction in heart rate variability among the elderly and subjects with ischaemic heart disease, hypertension and diabetes. Increases in the levels of ambient PM have also been found to be associated with increases in blood pressure, plasma viscosity and serum C-reactive protein among healthy subjects, and with decreased endothelial dilation in subjects with diabetes.

Almost all epidemiological studies of the health effects of particulate matter have been conducted in urban areas, which are impacted by PM that is likely to be quite different in size distribution and composition to the PM impacting a semi-arid rural environment such as Port Hedland. Therefore the extent to which this epidemiological data can be extrapolated from urban environments to Port Hedland is uncertain. Such extrapolations should be interpreted with caution and be the subject of future studies.

Toxicology of airborne particulate matter

Inhaled PM elicits an inflammatory response in the lungs, with ultrafine particles eliciting a greater acute and persistent inflammatory response. Organic compounds are responsible for much of the toxicity of diesel exhaust particles, while oxygen free radical production also plays an important role. Acids and transition metals associated with PM have been shown to cause inflammation and lung injury in animal models. Some but not all of the cellular effects of $PM_{10-2.5}$ appear to be mediated by bacterially-derived endotoxin, whereas toxic effects caused by solvent-extractable organic compounds were mostly associated with the $PM_{2.5}$ fraction. The toxicity of transition metals associated with PM has been investigated on respiratory epithelial cells and *in vivo* in rats, using ambient PM collected in the vicinity of a steel mill. Urban PM_{10} and $PM_{2.5}$ collected in different European cities has been shown to have allergy-enhancing activity as assessed by cellular response in the lymph nodes and antigen-specific IgE antibody response in the serum of mice. These PM fractions also induced cytokine release from different types of rat and human lung cells and the content of both crustal- and combustion-derived metals, was positively correlated with cytokine release. The PAMCHAR project, which is currently in progress in Europe, will analyse associations between the physicochemical characteristics of PM_{10} subfractions and cytotoxic, pro-inflammatory and genotoxic effects on human and murine respiratory cells *in vitro*, and will test significant *in vitro* cytotoxic and pro-inflammatory effects in animal models.

7. Formulation of dust exposure guidelines for Port Hedland

The important question that needs to be addressed is what is the potential risk to the population of Port Hedland from the dust to which they are exposed at the concentrations which they experience. There is limited knowledge of the concentrations, composition and toxicity of the dust, or reliable measurements of dust exposure at Port Hedland, on which to come to firm conclusions and base recommendations. Therefore scientific data from other, broadly comparable settings has been considered as a possible basis for recommendations on acceptable levels of dust exposure in Port Hedland.

It is questionable whether coal dust is an appropriately analogous substance for ambient particulate matter in Port Hedland, and basing recommendations for dust control and air quality standards on the analogy with coal dust may exaggerate the adverse health effects of Port Hedland dust. Similarly, the significant differences in particle size and composition of welding fumes and London Underground dust mean that these are unlikely to be useful

analogies from which to derive an air quality standard for Port Hedland. It also seems inappropriate, given the current uncertainty regarding the concentration and size of silica particles present in the dust impacting Port Hedland, to attempt to derive an air quality standard based on the proposed reference exposure limit of 3 $\mu g/m^3$ for community exposure to respirable crystalline silica.

There is limited evidence that daily fluctuations in the concentration of the coarse fraction of air pollution aerosols may have a comparable effect on morbidity as the fine fraction, and a recent review by the US EPA found sufficient evidence to implicate urban $PM_{10-2.5}$ in adverse health effects. While specifically excluding contributions from mining operations, the EPA Clean Air Science Advisory Committee recommends that $PM_{10-2.5}$ be monitored in both urban and rural communities, and a short-term air quality standard of 70 $\mu g/m^3$ as a 24-h average has been proposed for $PM_{10-2.5}$.

In the absence of informative data, it is difficult to reach a conclusion regarding an Air Quality Standard that would be appropriate for the Port Hedland community. The most important recommendation that can be made at this time is that reliable, valid data should be collected in order to inform future decisions.

Specific recommendations are:

1) The establishment of a network of samplers that are capable of accurately and reliably measuring PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ mass concentrations at various locations around the Port Hedland town site and BHPBIO processing and loading facility at Port Nelson. Computer modelling should be used to assess the potential influences of wind direction and speed, daily activities at the iron ore loading facility and seasonal variations on the average daily $PM_{10-2.5}$ concentrations measured at specific monitors.

2) Information on the compositions of the PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ size fractions at various locations around Port Hedland should be obtained. This should include analyses of elements that are of specific interest such as Fe, Cu, Mn, Zn, V, Mg, Al and Si, as well as other components likely to occur in Port Hedland dust, including sulphate, nitrate, ammonia, sodium chloride and organic and elemental carbon. Using a well-designed network and appropriate sampling technology, it should be possible to measure PM concentrations and compositions precisely and accurately, even under the difficult environmental conditions prevalent in Port Hedland.

3) In order to obtain valid and representative measurements of the actual PM exposure experienced by the Port Hedland population, some form of personal sampling in representative groups of the population is needed. Ideally this should be combined with a prospective study carried out to determine whether there are any associated short-term adverse health outcomes.

4) A retrospective study should be undertaken to determine whether there has been any direct harm to the health of the community residing in Port Hedland. This could take the form of a questionnaire survey of respiratory health, lung function tests and chest radiographs.

5) Samples of Port Hedland dust should be tested in *in vitro* cell toxicity studies on human respiratory cells.

Because these proposals will require time to implement and there is a need to provide guidance on a shorter time scale, we would recommend that the control of air pollution in Port Hedland be achieved by imposing an interim Air Quality Standard whereby the $PM_{10-2.5}$ concentration (the difference between co-located PM_{10} and $PM_{2.5}$ mass concentration measurements) would not be allowed to exceed 70 $\mu g/m^3$ as a 24-h average. Because there may be manganese in the aerosol impacting Port Hedland, we also recommend that the annual average manganese concentration should be maintained below the World Health Organisation Air Quality Guideline of 0.15 $\mu g/m^3$ (as PM_{10}) to protect against potential neurotoxicity. No recommendation is made for other metals such as copper, as exposure to these is deemed to be negligible.

Any strategy to protect the health of the community and employees of the company from adverse effects of dust inhalation requires decisions on the numbers and siting of samplers. Such decisions should be made on the basis of a detailed understanding of the processes involved and local geography.

SECTION 1. INTRODUCTION

Summary

There are potential health hazards associated with inhalation of airborne dust, related to the concentration, particle size, and constituents of the dust. The purpose of this report is to help those trying to identify whether there are potential health risks to the residents of Port Hedland from airborne dust. The report reviews the available scientific data on the hazards associated with airborne dust in Port Hedland and particulate air pollution in other settings. When assessing these hazards it is important to have clarity about the amount of exposure, the size of the particles and the toxicity and nature of the particles involved. The BHP Billiton Iron Ore processing/loading facility is virtually adjacent to the Port Hedland township. The facility stockpiles large amounts of iron ore as well as salt, manganese, feldspar and copper. Port Hedland is impacted by dust, principally fugitive iron ore. Dust dispersion in Port Hedland is exacerbated by the dry, windy climate.

1.1 Hazard and risk from inhaling dusts

There is a hazard wherever dust gets into the air so that people may inhale it. The term "hazard" means the potential to do harm; the likelihood of harm occurring and its severity are quantified as "risk". In this report we shall distinguish these two concepts. Anything, even oxygen, inhaled into the lung may be regarded as a hazard, but whether this hazard implies risk depends on the amount and duration of exposure and on the inherent toxicity of the substance.

A number of different methods are used to quantify the risk from airborne substances (air pollutants) likely to be inhaled by exposed individuals. This quantification may be done at three levels – first (usually) by weighing the mass of the substance in a given volume of air; secondly, by analysing the chemical or mineralogical nature of that substance; and thirdly, by a toxicological analysis of the substance and/or by an epidemiological study of its effects on populations. Understanding and estimation of risk comes from the results of such an analysis, taking account where possible of previous studies of human beings exposed to the substances in question.

For over 200 years it has been an appreciated that some industrial dusts when inhaled in sufficient quantity may cause severe disease. There is a large literature for example on the effects of crystalline silicon dioxide (quartz) and coal dust in causing disease in miners. Similarly, since the mid 20th century, much information has been published on the harmful effects of combustion-generated air pollution (from coal and motor vehicles) on health. Quantitative information exists on risks to human populations from these very different pollutants and has been used in setting protective air quality standards. It is critically important to recognise that these standards are specific for the particular pollutant, having been based on studies of that pollutant. A common cause of misunderstanding arises from the use of apparently common units of quantification in these different circumstances. While no-one would regard a kilogram of chocolate as being as tasty as a kilogram of chalk, or 100 mg of salt as being as dangerous as 100 mg of arsenic, it is a not uncommon misconception that 100 μg of dust is equally hazardous whether it is derived from mining, combustion or the action of wind on the sea. It is not only knowledge of the weight of the hazardous substance inhaled that allows determination of risk; it is also an understanding of its constituents and of their inherent toxicity.

It is conventional to measure airborne dust as mass (mg or μg) in a cubic metre of air. In general, in industrial situations the measurements are expressed as mg/m^3 whereas in ambient air pollution the units are μg/m^3, 1,000 times lower, since the effects of combustion-generated particles appear to occur at much lower concentrations than those of dusts generated by mining processes. Air quality standards reflect this – Australian coal miners, for example, may be exposed to concentrations of coal dust up to 3 mg/m^3 (equivalent to 3,000 μg/m^3) whereas the urban air quality standard is currently 50 μg/m^3. To confuse matters even further, the mass of pollutant is measured in different ways in the two situations. In ambient air, it is usual to measure it as $PM_{2.5}$ or PM_{10}, that is the mass of particles with average size below, respectively, 2.5 and 10μm in aerodynamic diameter, whereas in industrial situations it is conventional to measure "respirable dust" which is predominantly less than about 3.5μm in diameter. These three slightly different metrics are used to reflect what is regarded as the component of the dust most likely to cause harm. For ease of comparison we have expressed all airborne dust concentrations in this report in μg/m^3.

The most important problems arise at what might be regarded as the interface between these two environments: the workplace, where protection of workers is paramount and drives

standards; and the general ambient environment where standards are driven by a desire to protect the vulnerable, children, older and sick people. This may give rise to apparent anomalies where within a mine boundary an air quality standard of say 3,000μg/m^3 applies whereas outside the standard may be 50μg/m^3, 60 times lower. Perhaps less commonly known is the fact that equally large gradients in exposure to pollutant mass can be created between the inside and outside of one's home, since cooking, smoking and vacuum cleaning can easily produce indoor concentrations of dust up to several hundred μg/m^3. But of course the dust in each case is different and implies different risks.

In spite of the confusion caused by these different measurement methods and similar means of expressing the results, there exists a well-tried method of estimating hazard and the risks implied. It is necessary to know what people may be exposed to, how great that exposure is/has been, and what is the toxicity of the substances to which they are exposed. Since there is much information in the literature on many substances, the first step is to find out what is already known; this is the basis of this report.

1.2 Port Hedland

Port Hedland is a small town (approximately 4,000 people) in the North West of Western Australia, and is situated virtually beside BHP Billiton Iron Ore's (BHPBIO) processing and loading facility at Nelson Point, which is part of Port Hedland. Approximately 1,000 people live near to the port facility. Port Hedland is a narrow strip of land (about 1 kilometre wide and 5 kilometres long) between the BHPBIO's processing and loading facility and the ocean. South Hedland (approximately 15,000 people) is a small town that has been built as a sister town about 15 kilometres south of Port Hedland. BHPBIO ships approximately 80 – 100 million tonnes per annum (MTPA) of iron ore from Port Hedland. Also near the town of Port Hedland, the Port Hedland Port Authority wharf ships salt (3 MTPA), manganese (0.5 MTPA) as well as small amounts of feldspar (0.05 MTPA) and copper concentrate (0.024 MTPA proposed). These materials are stockpiled very close to residential and commercial areas. There are proposals to increase the iron ore shipments with increased production from BHPBIO as well as new producers entering the market (Hope Downs and Fortescue Metals Group). BHPBIO is, however, proposing to move its crushing and screening facilities from Port Hedland to Newman, which will reduce dust generation at Nelson Point and Port Hedland.

The town is primarily impacted by fugitive iron ore dusts, but is naturally dusty due to its semi-arid climate (dry, hot and windy) and environmental factors. Port Hedland is hot (average temperatures are 40^0C in summer, and 28^0C in winter), as well as windy (mean 3pm wind speed is 25 km/h in summer, 15 km /h in winter) and dry.

There are no significant sources of combustion particles in the town apart from a waste incinerator, which is 7 to 8 km directly south of the town in an industrial area – Wedgefield. There are also very low levels of oxides of sulphur and nitrogen plus ozone at Port Hedland.

BHPBIO propose to significantly increase the production of iron ore to be shipped out of Port Hedland. It is acknowledged that this will provide a major boost to the Western Australian and Australian economies. Despite the best intentions the increased storage, loading and shipping of ore is likely to lead to an increase in dust exposure to residents and working staff. Regional winds also cause salt and crustal dust to be airborne and therefore will be a component of any dust exposure to Port Hedland residents.

1.3 The purpose of this report

An evaluation should be made of the potential impact of proposed changes to the ore loading operations on the health of Port Hedland residents. From an assessment of the composition of the airborne dust, the concentration and relative toxicity of the dust constituents and the size of dust particles it should be possible to identify whether there are potential risks to health for the residents. In addition, by comparing this information with data about the hazards from urban air particle pollution found in large cities it may be possible to decide what level of exposure may be safely tolerated within the community.

This report reviews the available scientific information about the hazards associated with the various components of the airborne dust in Port Hedland and assesses them in the context of what is known about urban air particulate pollution. The document aims to provide sufficient information to address three key questions:

a) What are acceptable levels of particulate matter or dust in the Port Hedland township, and should these be measured as average and/or peak levels of particulate matter?

b) What type of monitoring is appropriate for the Port Hedland township and appropriate monitoring sites, if needed, to provide information on potential health effects?

c) What methods should be used to measure particulate matter, and what if any corrections need to be made to the data to allow for Port Hedland conditions and/or other environmental factors?

The report also considers the need to undertake further research to substantiate the recommendations.

SECTION 2. MINERALOGY OF AIRBORNE PARTICULATE MATTER IN PORT HEDLAND

Summary

The mineral composition of iron ore from the Pilbara region is typically 93% iron oxides (haematite and goethite), 4% quartz as chert, and 3% clays. Manganese oxides, copper ore minerals, feldspar and salt are minor components of airborne particulate matter in Port Hedland. Concentrations of fossil fuel combustion products in airborne particles are likely to be lower in Port Hedland than in large cities.

2.1 Introduction

This chapter reviews the probable composition of airborne dust in Port Hedland. It is believed that metal ores exported through Port Hedland make a substantial contribution to local concentrations of airborne dust. The exported ores are predominantly iron, but copper and manganese ores, feldspar and salt are also handled. Other sources of wind-blown dust include soil-derived particles, which are likely to form a large component of the airborne dust in the town, together with salt particles derived from the sea.

2.2 Overview of local geology

The Pilbara Craton which lies between Karratha, Port Hedland, Marble Bar and Nullagine is the oldest piece of continental crust in Western Australia having formed about 2.6 billion years ago. The Pilbara Craton consists of mixed volcanic-sedimentary sequences (Greenstone Belts) and granite (a coarsely crystalline rock originally formed through the intrusion of silica-rich melts into the earth's crust at several kilometres depth). All Greenstone belts show some degree of recrystallisation and deformation as a result of increased temperature and pressure during burial combined with deformation associated with the development of folds in response to lateral rather than vertical forces within the crust. Life was at a very early stage of evolution at the time of formation of the Pilbara Craton and these rocks contain no fossils of multicellular animals. In the absence of widespread plant life, the earth's atmosphere contained only very low levels of oxygen. This may explain the development of iron and manganese ores at the earth's surface that are very different in mineralogy and texture from those formed in later geological history. The Pilbara Craton was subsequently joined to a similar piece of continental crust, the Yilgarn Craton, about 1.3 billion years ago.

Subsequently these very old pieces of continental crust have undergone relatively little deformation.

2.3 Mineralogy of ores exported through Port Hedland

Table 1 provides a brief description of the iron ore BHP Billiton currently mines in the Pilbara, all of which are types of Banded Ironstone Formation (BIF).

Table 1 Iron ores shipped through Port Hedland

Ore	Description
Mt Whaleback and Newman Satellites - Brockman Formation	Predominantly haematite that is hard and can be mined as lump material (> 6mm size). This is the most valuable iron ore deposits because of the iron ore being in lump form.
Mining Area C (MAC) - Marra Mamba Formation	Predominantly goethite and minor haematite that is more friable fines (<6mm size).
Yandi - Yandi Pisolitic Formation	Predominantly goethite and haematite that is in pea-size pebbles.

The term "formation" refers to a group of rocks that was deposited over a discrete period of time and differs in some way from the formation above and below. The mined formations differ from each other in texture and mineralogy because of the processes that the original ore has been exposed to over time.

BIF are characterised by fine laminations between 0.3 and 5 cm thick that are in turn laminated on a scale of sub millimetres to millimetres. The layering consists of silica rich and iron rich layers. They are found in rocks greater than 1.8 billion years old and differ from more recently formed ironstones in having very low aluminium content. They occur in sedimentary rock sequences that were deposited by water during the period of earth history when simple unicellular life forms were present but oxygen levels in the atmosphere were considerable lower than at present. BIF are typically found within greenstone belts.

The mineral composition of iron ore from the Pilbara region is typically 93% iron oxides, 4% quartz as chert, 3% clays (aluminosilicates and iron-based clays).

Currently BHP Billiton Iron Ore is exporting 6 iron ore products from Port Hedland – lump and fines from Brockman Formation (about 70% of quantity shipped), Marra Mamba Formation (about 10%) and Yandi Pisolitic Formation (about 20%). In the future, there will be increased quantities of Yandi and Marra Mamba ores shipped with the increase being greater for Marra Mamba. Greater dust is likely to be generated from handling ores from both these formations because they are more friable.

There is essentially no difference in mineralogy between lump and fines from each iron ore formation type.

2.4 Mineralogy of Banded Iron Formations

The iron layers in BIF are dominated by the minerals magnetite (Fe^{2+} Fe_2^{3+} O_4; black in colour) and haematite (Fe_2^{3+} O_3; red). A third of the iron in magnetite is in the reduced form whereas the iron in haematite is fully oxidised.

The silica layers are dominated by chert, a microcrystalline form of quartz that is formed from amorphous silica gel. Individual layers may extend for over 300 km. Individual quartz crystals range from less than 1 μm to 50 μm in size. Chert also contains smaller quantities of quartz as larger crystals and also in the form of chalcedony, a fibrous variety of quartz. Both the microcrystalline quartz and chalcedony contain small amounts of water within the crystal lattice and a large number of other defects. It is not certain whether all three forms of quartz display similar levels of toxicity as a number of investigations have shown quartz from different sources to have differing toxicities (Cakmak et al., 2004). The high prevalence of crystal defects in chert versus quartz from igneous rocks could lead to differences in toxic properties. The quartz crystals in chert form a tough interlocking fabric. During ore extraction and processing quartz crystals are likely to be shattered to give rise to particles with highly active surfaces. In contrast, quartz particles released from other types of sedimentary rock tend to have surfaces contaminated by iron oxides and/or clays (alumino-silicate minerals). These surface contaminants are thought to lead to differences in toxic properties (Soutar et al., 2000).

BIF also contain carbonate minerals, particularly siderite (Fe^{2+} CO_3; yellow) and also ankerite ((Fe^{2+} $Mg)_2$ $(CO_3)_2$; white to brown) inter-layered with chert. These minerals are particularly

abundant in pisolitic rocks. Pisolites are pea-sized grains composed of concentric layers of carbonate minerals such as siderite (which may have been replaced by other minerals subsequent to deposition) or iron-rich silicates. These minerals typically contain ferrous (Fe^{2+}) iron. The original mineralogy of iron silicate pisolites was probably dominated by the green coloured clay minerals greenalite and chamosite (green clay minerals closely related to chlorite with compositions of $Fe_6^{2+}\ Si_4O_{10}\ (0H)_5$ and $Fe_4^{2+}Al_2Si_2Al_2O_{10}(0H)_x$ respectively), and glauconite ($(K,Na,Ca)_{1.2\text{-}2.0}(Fe^{3+},Al,Fe^{2+},Mg)_4(Si_{7\text{-}7.6}Al_{1\text{-}0.4}O_{20})(OH)_4.n(H_2O)$), a green coloured mica. These minerals may weather to browns and reds. Subsequent low temperature alteration (metamorphism) has locally given rise to other iron silicate minerals, such as minnesotaite $(Fe^{2+},Mg)_3Si_4O_{10}(OH)_2$ and stilpnomelane $((K,Na,Ca)_{0\text{-}1.4}(Fe^{3+},Fe^{2+},Mg,Al,Mn)_{5.9\text{-}8.2}(Si_8O_{20})(OH)_4(O,OH,H_2O)_{3.6\text{-}8.5})$. Minnesotaite is pale green and stilpnomelane is a dark red brown in colour. Both minerals are sheet silicates with structures similar to that of talc. Little is known about the toxicity of these various iron silicates. Where BIF have undergone subsequent high temperature metamorphism and dehydration, other iron silica minerals may also be present. Veins of the amphibole asbestos minerals crocidolite $(Na_2Fe_3^{2+}Fe_2^{3+}(Si_8O_{22})(OH)_2$ and amosite $(Fe^{2+}Mg)_7(Si_8O_{22})(OH)_2)$ are sometimes found in metamorphosed BIF.

Other rock formations associated with BIF are quartzite (highly pure-quartz cemented, quartz sandstone), black carbonaceous shale, conglomerate, dolomite, massive chert, chert breccia and mudstone. These rock types were deposited in water of varying depth. They would not be present in the exported ore.

Goethite ($Fe^{2+}O.OH$) is a weathered (hydrated) form of haematite, typically yellow to brown in colour. Goethite usually occurs as a finer, dustier material in iron ore, than haematite. The process of extracting and processing ore is likely to liberate more dust from goethite dominated ore than from haematite. The goethite particles released however, are probably larger than those released from haematite.

2.5 Existing information about dust generated from iron ore exported through Port Hedland

A sample of representative dust from the combined mines, taken from near a transfer station close to the western end of Nelson Point (Port Hedland) was analysed by x-ray diffraction

(XRD) through the Chemistry Centre of Western Australia (unpublished study undertaken by Glossop Consultancy for BHP Billiton Iron Ore). The results are shown in Table 2.

Table 2 Mineralogy of a dust sample from a transfer station at Port Hedland

Size Fraction	Minerals					
	Haematite	Goethite	Kaolinite	Chlorite	Mica	Quartz
< 2µm	Major	Major	Dominant	Minor	Trace	-
2 to 10µm	Dominant	Major	Major	Trace	Trace	Minor
> 10µm	Dominant	Major	Minor	-	-	Minor

Dominant: >50%; Major: 10 to 50%; Minor: 2 to 10%; Trace: <2%

This analysis shows that most of the airborne dust from the iron ore handling at Port Hedland consists of iron oxides (haematite and goethite) as well as clays (aluminosilicates and iron silicates) and quartz (as chert). The quartz (as chert) is in the >2µm size range.

Nearly all studies on the chemical (elemental) composition of iron ore use the following description: Fe, SiO_2, Al_2O_3, TiO_2, CaO, MgO, MnO. The iron (Fe) is as the % of iron even though it is likely to be a mixture of oxides (magnetite, haematite and goethite). The Si, Al, Ti, Ca, Mg, Mn, etc are all quoted as if they are the oxide as stated above. The Si and Al are likely to be present as clay minerals. The quartz in the XRD analysis above is most likely chert.

2.6 Manganese Ore

Pilbara Manganese has a high manganese content with low iron and phosphorous content. The Pilbara Manganese Pty Ltd product specifications are shown in Table 3.

Table 3 Manganese ore shipped through Port Hedland

	Lump specifications Size: 6.5mm-75mm		Fines specifications Size: 1mm - 6.5mm	
	Guaranteed	Typical	Guaranteed	Typical
Mn	48.0%MIN	49.5%	45.0%MIN	46.5%
Fe	5.0%MAX	4.5%	8.0%MAX	7%
SiO_2	13.0%MAX	12%	13.0%MAX	10%
Al_2O_3	1.0%MAX	0.6%	1.0%MAX	0.8%
P	0.05%MAX	0.03%	0.07%MAX	0.05%

(from www.consminerals.com.au/pages/manganese/pilbara-manganese.htm)

The most common manganese ore minerals in Western Australia are the oxides, braunite ($Mn_2^{2+}Mn_6^{3+}SiO_{12}$), manganite ($Mn^{3+}O(OH)$) and hausmannite ($Mn^{4+}Mn^{2+}O_4$). These deposits are believed to have formed in shallow marine basins and like BIF are confined to rock sequences that are greater than 1.8 billion years in age. They are believed to have formed as chemical precipitates on the floor of the shallow oceanic basins in a highly oxidising environment.

2.7 Copper Ore

The copper ores of Pilbara were deposited by hydrothermal fluids (i.e. hot water) in mixed sedimentary-volcanic sequences deposited over 2 billion years ago. The circulation of superheated aqueous fluid through these sequences leached base metals such as copper out of the volcanic rocks and redeposited the metals in veins at higher levels in the sequence. The ore deposits are dominated by metal sulphides. Copper ore minerals include chalcopyrite ($CuFeS_2$: a gold coloured mineral).

2.8 Feldspar

The feldspars are framework silicates with structural similarities to quartz. There are two main groups of feldspar: the alkali feldspars ($(KNa)(AlSi_3O_8)$) and the plagioclase feldspars.

The alkali feldspars include sodium and potassium rich varieties. The plagioclase feldspars are a continuous solid solution series ranging from the sodium-rich albite ($Na_2Al_3Si_3O_8$) to the calcium-rich anorthite ($CaAl_2Si_2O_8$) with all the intervening compositions being found in nature. Feldspar mineralogy is dependent on the conditions of formation. The low temperature form of K-feldspar is microcline, at higher temperatures orthoclase is formed and at higher temperatures still sanidine is formed. Feldspar minerals are extremely common in the earth's crust, being found in most types of igneous rock and in many types of metamorphic rock. They weather to clays at the earth's surface and are therefore less common in unmetamorphosed sedimentary sequences.

Feldspars are usually dispersed as relatively small crystals in their host lithology and can constitute up to 30% of some igneous rocks such as granite, or of highly metamorphosed mudstones that have re-crystallised to the metamorphic rock, gneiss. Feldspars are usually mined from thick veins called pegmatites that consist of large crystals of quartz, feldspar, muscovite mica and sometimes metal ore minerals. Pegmatites are generally formed during the last stages of cooling of large bodies of granite that were intruded into the earth's crust at several kilometres depth. They were deposited from hot-water rich fluids in concurrently developing cracks in the granite and in the surrounding rocks. Possible contaminants of mined feldspar deposits include quartz, which is likely to be present as highly shattered crystals with highly reactive surfaces. Other possible contaminants include muscovite mica, a sheet silicate, and the clay mineral kaolinite ($Al_4(Si_4O_{10})(OH)_6$).

2.9 Salt

Salt deposits are usually formed through the evaporation of seawater and are typically found in sedimentary sequences. Salt deposits may also form in arid environments through evaporation of surface and groundwaters of non-marine origin. Both marine and non-marine salt deposits are dominated by the mineral halite (NaCl; clear or white in colour) and the sulphate mineral gypsum ($CaSO_4.2H_2O$; white) and/or anhydrite ($CaSO_4$; white) are also likely to be present. The mined salt is likely to be dominantly halite although a wide range of other salts including other halides and sulphates, chromates, dichromates and nitrates are mined in other parts of the world.

2.10 Composition of other dust sources

a) Soil particles

A range of soil types typical of an arid climate are found in Western Australia (www.grdc.com.au/growers/oft/soiltype.htm). Sodosols are typically red-brown in colour with a clay content that increases abruptly with depth, and a high sodium content. These soils are usually very hard when dry and are prone to crust formation. Tenosols are well drained red loamy soils with a red-brown hardpan at shallow depths. Kandosols are mostly well-drained, permeable soils although some yellow and most of the grey forms have impeded subsoil drainage. They are susceptible to surface soil degradation such as hard setting and crust formation. All of these soils would be susceptible to wind erosion. Soil derived dust would be expected to be dominated by quartz and clay minerals with smaller quantities of minerals such as muscovite ($K_2Al_4(Si_6Al_2O_{20})(OH,F)_4$); silvery sheet silicate common in metamorphosed mudstones and some silica-rich igneous rocks), calcite/aragonite ($CaCO_3$; white in colour) and gypsum that typically form hardpans in arid soils haematite and goethite. The clay minerals present would be expected to include illite (a pale clay mineral closely related to muscovite mica and indistinguishable by XRD), kaolinite and chlorite ($(Mg,Al,Fe)_{12}(Si,Al)_8O_{20})(OH)_{16}$; a green clay that may oxidise to a red colour).

b) Sea salt

Sea salt is dominated by halite (sodium chloride) that may react with other pollutants to form other soluble salts such as sodium nitrate.

c) Vehicle emissions and other combustion sources

Particles from combustion sources are dominated by carbon rich particles of small size (<10 μm). They are the dominant component of urban air pollution in most cities and the fraction of ambient PM_{10} that is thought to be most closely linked to adverse health effects. Concentrations of combustion particles would be expected to be much lower in Port Hedland than in large cities.

2.11 Elemental composition of airborne dust from Port Hedland

A sample of airborne dust (as Total Suspended Particulate – TSP) collected over a week from the wharf (named Town Centre Monitor) was analysed by Inductively Coupled Plasma Mass Spectrometry (ICP-MS) through CSIRO in Melbourne (unpublished study undertaken by

Glossop Consultancy for BHP Billiton Iron Ore). The analysis of a small sample of this dust is provided in the following figure.

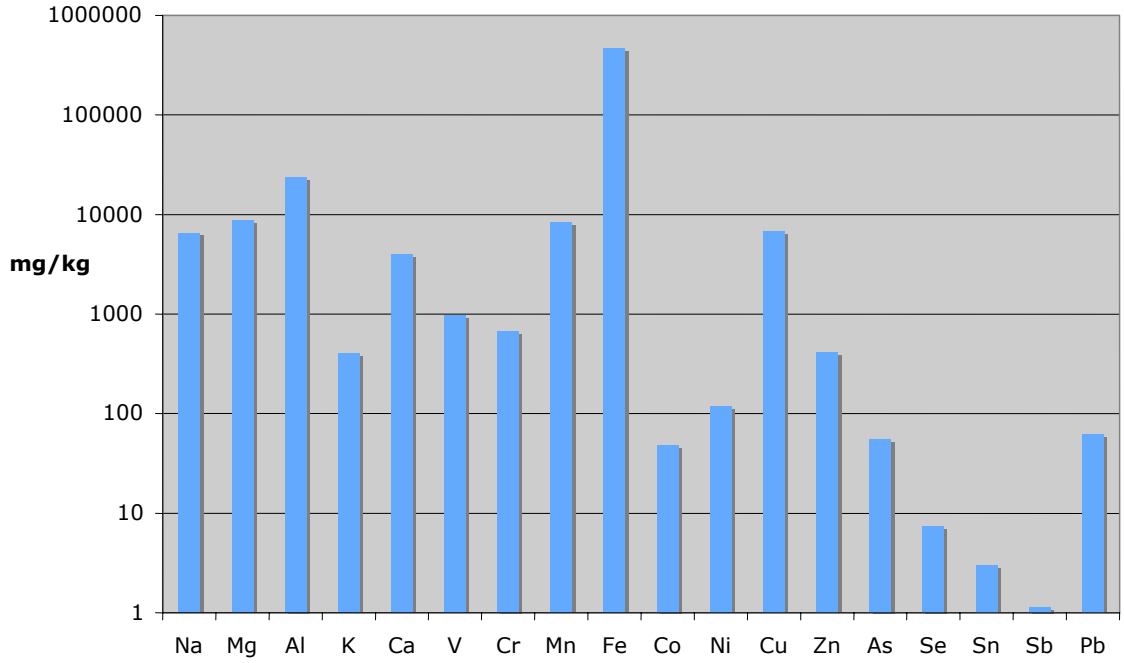


Figure 2.1 Summary of elemental composition of airborne dust

The authors of the report suggested the results for Na, Al, K, Ca and V may not be reliable indicators of dust from ore handling because of other potential environmental sources. In addition the potassium and selenium concentrations were close to the detection limit for the analytical technique. It was stated that the error associated with each element could have been significant because only a small sample was taken of the total TSP sample, but it is likely to be representative of the dust in Port Hedland. However, elemental composition of the dust is likely to vary on a day to day basis, depending on loading activities and weather patterns.

The copper and manganese is most likely from the loading of copper and manganese ores through Port Hedland. The copper and manganese ores also have significant quantities of other metals/metaloids such as arsenic. The analysis could not detect silicon. There appears to be a low level of iron and elevated level of aluminium compared to iron ore. This could be due to an apparent lower concentration of clays than of iron ore. The clays are likely to travel much further than the iron oxides because of the size of particles and their density.

SECTION 3. PARTICLE BEHAVIOUR AND MEASUREMENT

Summary

The effects of airborne particulate matter (PM) on health are determined by the inherent toxicity of particles and their pattern of deposition in and removal from the respiratory tract. The terminology of biologically relevant fractions of particulates has been standardized: the *respirable fraction* is the mass fraction of inhaled particles penetrating to the unciliated lung parenchyma; the $PM_{2.5}$ fraction is alternatively called the *fine particle fraction;* $PM_{2.5-10}$ is termed the *coarse particle fraction;* $PM_{0.1}$ is termed the *ultrafine (UF) particle fraction.* The large variety of instruments used for measuring PM includes many types of fixed location devices and, increasingly, portable devices for measuring personal ambient exposures.

3.1 Behaviour of inhaled particles in the respiratory tract

The physicochemical characteristics of airborne particles that are responsible for their adverse health effects are incompletely understood. It is clear, however, that both the inherent toxicity of particles and the pattern of their deposition in and removal from the respiratory tract are important.

The aerodynamic behaviour of particles within the respiratory tract is determined by physical properties such as size, shape, density and capacity to absorb moisture (hygroscopicity). Of principal importance is a particle's effective size, expressed as the *aerodynamic equivalent diameter*, i.e., the diameter of a unit-density spherical particle having the same settling velocity as the particle under consideration (Clarke and Yeates, 1994). Much of our understanding in this area has been derived from aerosol science which underpins the pharmaceutical treatment of conditions such as asthma (Dhand, 2000).

The predominant mechanisms of particle deposition in the respiratory tract are:

1. **Impaction** i.e., the inertial tendency, especially of larger particles, to follow a linear trajectory and deposit in the nose, pharynx or bifurcations of large airways. During resting breathing, essentially all particles >20μm and about 95% of particles >5μm are deposited in the nose by this means. Mouth breathing accentuates penetration of such particles into the tracheobronchial tree, and inertial impaction for particles >5μm passing the larynx is dominant in the major and segmental bronchi.

2. **Sedimentation** i.e., the gravitational settling of particles on airway surfaces. This is a principal means of deposition for medium-sized particles (1-5 μm), and occurs particularly in the small airways.
3. **Diffusion** i.e., collision of particles with airway surfaces due to random (Brownian) motion resulting from bombardment by gas molecules. Substantial deposition by this mechanism occurs only for fine particles (<1 μm), primarily in the small airways and alveoli.
4. **Electrostatic precipitation** enhances the deposition of smaller particles carrying electrical charges, but has negligible effects on particles ≥4 μm.
5. **Interception** by the airway wall accounts for the deposition of some particles, particularly fibres (Clarke and Yeates, 1994; West, 2005).

Clearance of particles deposited in the respiratory tract is dependent on the anatomical site of their deposition. The tracheobronchial tree is characterized by the production of mucus, which is propelled toward the oropharynx by the rhythmic motion of millions of microscopic cilia on the luminal surface of airway epithelial cells. In the smallest airways, mucus production is absent and the proportion of ciliated cells is diminished. It has been traditionally believed that particles deposited on all but the most distal airways are removed rapidly (within 24 hours) by this mucociliary escalator, but recent experimental data suggest that a substantial proportion of particles deposited on small ciliated airways are retained for more than 24 hours (Lastbom and Camner, 2000). Particles that penetrate distally into the lung acinus (respiratory bronchioles and alveoli) are cleared much less efficiently (principally by alveolar macrophages, the main scavenger cells of the immune system). If macrophage capacity is overwhelmed, however, ultrafine particles may perturb the epithelial barrier and enter the interstitium (a process sometimes termed “interstitialization”), provoking local and systemic inflammation (Seaton, 2000; Clarke and Yeates, 1994).

3.2 Classification of PM relevant to health

Terminology on biologically relevant fractions of particulates has been standardized by the International Standards Organisation (ISO). Three sampling conventions have been defined, based on studies in human volunteers (International Standards Organisation, 1994):

The **inhalable fraction** is defined as the mass fraction of inhaled particles which is inhaled into the nose or mouth

The **thoracic fraction** is the component of the inhalable fraction penetrating beyond the larynx.

The **respirable fraction** is a subset of the thoracic fraction, and is defined as the mass fraction of inhaled particles which penetrates to the unciliated lung parenchyma.

Standard particle size indices for scientific and regulatory purposes have been developed by the United States Environmental Protection Agency (EPA), independently of the ISO sampling conventions. PM conventions are based on the assumption of mouth breathing (Lastbom and Camner, 2000). The PM_{10} convention was introduced by the EPA in 1979 and can be defined as:

> Particulate matter less than 10 μm aerodynamic diameter (or, more strictly, particles which pass through a size selective inlet with a 50% efficiency cut-off at 10 μm aerodynamic diameter) (Expert Panel on Air Quality Standards, 2001a)

Given the disproportionate respirability of smaller particles (<2.5 μm), the EPA added a $PM_{2.5}$ convention in 2000:

> Particulate matter less than 2.5 μm aerodynamic diameter (or, more strictly, particles which pass through a size selective inlet with a 50% efficiency cut-off at 2.5 μm aerodynamic diameter) (Expert Panel on Air Quality Standards, 2001a)

(The inhalable fraction corresponds with a size threshold of about 50 μm. The thoracic fraction is almost identical to the PM_{10} fraction. The respirable fraction is, however, significantly different from the $PM_{2.5}$ fraction and approximates a particle size threshold of about 3.5-4 μm.

The $PM_{2.5}$ fraction is alternatively termed the **fine particle fraction**. $PM_{10-2.5}$ (i.e., particles of aerodynamic diameter in the range 10-2.5 μm) forms a **coarse particle fraction** which nowadays is often measured directly in epidemiological research.

Given the recent recognition of adverse effects specifically attributable to extremely small, combustion-generated (ultrafine) particulate matter, a $PM_{0.1}$ convention (i.e., particles of

aerodynamic diameter less than 0.1 μm) is increasingly used for measurement in research settings, but is not currently recognized in the EPA's regulatory framework.

3.3 Measurement of airborne particulate matter

Measurement of airborne particulate matter is not straightforward. Devices for measurement have evolved considerably over the course of the last half-century, as new technologies have become available, as research has progressively elucidated the relationship between ambient PM and adverse health effects, and as regulatory policies have been modified accordingly. However, none of the available methods is without shortcomings. It is therefore not surprising that there is a large variety of instruments in use, and that regulatory frameworks for measurement of PM are not internationally standardized. Additionally, reported measurements are highly dependent on the method of measurement selected, and cannot be taken at face value as representing absolute quantities. Results from different devices in a given setting are therefore not interchangeable, and the device used for measurement must be stated along with the reporting of results.

As research on airborne particulates has centred on urban settings affected predominantly by anthropogenic particulates, devices designed for this context have dominated advances in measurement technology. There is, therefore, a relative paucity of recent literature on measurement devices pertinent to analysis of crustal (i.e., predominantly coarse fraction) PM. Nevertheless, there have been substantial advances in the measurement of coarse fraction particulates.

a) Common features of measurement devices

Common to most devices is the principle that ambient air is drawn by a pump at a predetermined flow rate through a sampling head which may select particles on the basis of aerodynamic size, and these particles can then be analyzed for a range of quantitative and qualitative characteristics. An alternative form of particle separator is the elutriator (settling chamber), which relies on gravity to remove particles from ambient air. The horizontal elutriator, equipped with a British Medical Research Council (BMRC) sampler intended to capture respirable particles, is seldom used today. Devices of this type may nevertheless have utility in the unusual sampling conditions of Port Hedland (see Section 7.2).

Although measurements based on mass (weight) have predominated, with the recent recognition of the toxicological importance of ultrafine particles ($PM_{0.1}$) has come increasing recognition of the importance of measuring particle counts.

Commonly used methods include filter-based gravimetric samplers, from which the filter must be removed and analysed after collection, and instruments allowing continuous (real time) measurements (Air Quality Expert Group, 2005a; Expert Panel on Air Quality Standards, 2001a; Sarnat et al., 2003; Wilson et al., 2002).

Some older devices purport to measure all total suspended particulate matter (TSP). However, entrainment of particulates in these devices is highly dependent on wind speed and direction, and collection of larger particles is unpredictable. In order to exclude unwanted larger particles, devices other than those using optical methods for particle detection require a size-selective inlet. Particle selection by size follows aerodynamic principles. Particles are entrained onto the filter by a pressure gradient. A convoluted route forces larger particles to divert from the path of entrainment, either onto a central impactor, or via a cyclone (by inertial diversion onto an outer wall from a helical air streamline). Inlets are designed so that 50% of particles of the critical cut-off size are excluded. Impactors tend to provide a sharper cut-off, and thus are usually the reference methods. Regular cleaning is required and application of grease to the impaction site is necessary to prevent recoil of unwanted particles back into the streamline, especially in the case of central impactors.

b) Fixed location measurement devices

(i) Filter-based gravimetric samplers

These samplers entrain PM onto a filter, in contemporary models typically a Teflon filter disc (Air Quality Expert Group, 2005a; Expert Panel on Air Quality Standards, 2001a; Sarnat et al., 2003). Analysis of collected PM requires removal of the filter after a given period, most often 24 hours. However, shorter or longer intervals of measurement (hours to weeks) are generally possible. Measurement is therefore delayed from the time of particle impaction and is potentially labour intensive. Although there is great variety in the design and cost of the filter-based gravimetric samplers in use, these devices are generally easy to maintain, relatively inexpensive and cost-effective for monitoring networks. They are also more precise than the continuous monitoring alternatives. They are consequently the reference devices specified for monitoring of PM, although there is a notable absence of standardized

recommendations for use of specific devices among UK, European Union and North American regulatory bodies. For many years, a TSP high volume sampler (HVS) was in widespread use in the USA. This device lacked accuracy as a consequence of high sensitivity to wind speed and wind direction. It has now generally been replaced by devices bearing size-selective inlets.

The methods are prone to artifactual error. Chemical reactions between constituents may occur in the process of sampling. Negative artefacts arising from particle desorption from the filter during collection is possible, especially with high flow rate apparatus. Increased PM mass may result from an increase in water absorption from ambient humidity onto hygroscopic PM, and reduction in mass may occur due to loss of semi-volatile constituents. Filters thus require conditioning at standard temperature (20^oC) and humidity (50%) prior to weighing. Filters are removed and subsequently weighed after a set time interval (typically 24 hours). The measured PM concentration therefore does not correspond with the absolute mass of PM originally entrained onto the filter. Some of the newer devices allow multiple automatic filter changes after each 24 hour period, providing a series of measurements without the apparatus having to be visited each day.

Several PM_{10} reference samplers are used in the European Union, including the Superhigh volume sampler (Wide Range Aerosol Classifier – WRAC) which is not suitable for general deployment, a high volume sampler (HVS PM_{10} sampler, 68 m^3/h) and a low volume sampler (LVS PM_{10} sampler, 2.3 m^3/h, also known as the Kleinfiltergerat or KFG). The last of these is most widely deployed in the UK. There is also a wide range of devices in use that are based on the same principles as the reference methods.

(ii) Reflectometric measurements - Black Smoke method

Until very recently, PM measurement in the United Kingdom was dominated by the Black Smoke method, an indirect means of assessing particle mass developed over 50 years ago (Air Quality Expert Group, 2005a; Expert Panel on Air Quality Standards, 2001a). In this method, diminution of the light reflectance of the filter resulting from the "blackness" of collected matter is measured as a proxy for mass using a standard reflectometer. Consequently, reflectance measurements by the Black Smoke method only indicate the presence of light-absorbing PM, particularly elemental carbon (soot). A standard calibration curve is necessary to convert the reflectance to a measure of total mass concentrations of PM. The calibration is

predicated on patterns of air pollution from the 1960s, when ambient urban PM in the UK was dominated by pollutants derived from coal smoke. The sampling head commonly used in Black Smoke devices has been shown to select particles smaller than approximately 4.5 μm. The equipment is relatively simple, robust and inexpensive.

Clearly, reflectometric techniques such as the Black Smoke method are not applicable to measurement of coarse fraction crustal dusts, and their use is problematic even in contemporary urban settings for which historically developed calibration data are no longer valid. There is nevertheless a vast body of measurement data based on this method, and its use has made an important historical contribution to urban anthropogenic PM monitoring and epidemiological analysis.

(iii) Direct reading instruments for continuous (real time) measurements

Tapered element oscillating microbalance (TEOM) analyzer

The TEOM is widely used in the UK and throughout the world for continuous measurement of PM concentration. This device contains a tapered glass tube element bearing a small filter at one end (Air Quality Expert Group, 2005a; Expert Panel on Air Quality Standards, 2001a). Its operation is based on the principle that the oscillation frequency of the element is in direct proportion to its mass, and the measured frequency of the element will thus increase in relation to any additional mass of PM deposited on the filter. The inlet and filter are maintained at 50^{o}C to minimize measurement error from water evaporation and condensation. For the purposes of US EPA standardization, a default adjustment factor of 1.03 accounting for moisture loss is applied to the TEOM measurement for calibration to the US EPA HVS standard. The high temperature of operation required to eliminate error due to variable humidity has also resulted in disparity with measurements using EU gravimetric reference samplers, attributed to loss of semi-volatile components (especially ammonium nitrate) in the TEOM. By an interim arrangement, a default "scaling factor" of 1.3 is applied to all UK national TEOM-derived data reported to the EU. In a recent advance, a drying apparatus can be affixed to the TEOM inlet, reducing the humidity of the sample stream and allowing the device to operate at a lower temperature (30^{o}C).

β-attenuation analyzer

In continental Europe, the β-attenuation analyzer is the device most commonly used to monitor ambient PM_{10}. Similarly to other apparatus, a size selective inlet is used, and this

method can therefore be used to measure TSP, $PM_{2.5}$ or other particle fractions if desired. The method is based on the capacity of the filter material and collected dust to absorb β particles produced by a radioactive source (typically carbon-14, krypton-85 or promethium-147). Continuous recording of transmitted β radiation is possible by this means. There is a close exponential relationship between the mass of dust collected and the absorption of β particles (and therefore the attenuation of transmitted β radiation). This relationship is reasonably independent of the chemical composition of the PM collected on the filter. As with the TEOM, there is a trade-off between artefacts arising from heating the sample stream to remove humidity and underestimation of mass due to dispersal of semi-volatile components. The filter in β-attenuation devices is usually not heated. Calibration factors for comparison with EU standard measures depend on whether an inlet heater is used. In the absence of a heater the conversion factor approximates 1:1 (Air Quality Expert Group, 2005a).

(iv) Optical analyzers

Recently developed devices use laser light scattering both to count and measure the size of particles, allowing devices with a TSP inlet to monitor PM_{10}, $PM_{2.5}$ and/or other size fractions simultaneously. As well as directly counting particles, the mass of collected PM can be measured indirectly, by calculations based on a number of assumptions about the chemical constituents of the PM. Clearly the composition of the PM and the count-mass conversion factor vary greatly with the setting. Additionally, the optical device can incorporate a filter for subsequent gravimetric analysis, allowing calibration for the specific application. Optical devices are generally small, light and battery-operated, and thus highly portable. They have not yet been used for national monitoring networks in the UK (Air Quality Expert Group, 2005a).

(v) Coarse particle fraction measurement – dichotomous samplers

Measurements of the coarse particle fraction ($PM_{10-2.5}$) have often been derived by calculating the difference between separate direct measures of PM_{10} and direct measures of fine particles ($PM_{2.5}$). This results in two measurement errors, compromising measurement precision (Brunekreef and Forsberg, 2005). Such imprecision is of particular importance in urban settings where the coarse fraction concentration is very low and the $PM_{2.5}$ to PM_{10} ratio approaches unity (Allen et al., 1999). This problem can, however, be circumvented with a dichotomous gravimetric sampler such as the Andersen PM_{10} device which divides the entrained particle population into two distinct size fractions during sampling (Quality of

Urban Air Review Group, 1996). This is achieved by following the PM_{10} filter with a $PM_{2.5}$ cut-off impactor and collecting both fractions on Teflon membrane filters, providing separate samples for $PM_{10-2.5}$ and $PM_{2.5}$, suitable for both gravimetric and chemical analysis. A shortcoming of this device is that variations in humidity influence the size of hygroscopic PM, particularly that with a high sulphate content. Under conditions of high humidity, particles otherwise appearing in the fine fraction may be collected with the coarse. A coarse PM measuring device for continuous monitoring has recently been developed in the form of a TEOM coupled with a PM_{10} inlet followed by a 2.5 μm cut-point nozzle impactor (Misra et al., 2001).

(vi) Devices for particle counts

Given that the toxicological impact of ultrafine PM is markedly disproportionate to its contribution to PM mass, and that particles < 0.1 μm in diameter dominate counts in the urban context, measures of particle counts are of particular importance for the UF fraction (Expert Panel on Air Quality Standards, 2001a). Most experience has been with devices such as the Condensation Nuclei Counter (CNC), which is based on the principle of condensing a vapour (e.g., n-butanol) onto particles, which are then counted by optical methods. Such instruments are increasingly used in field studies on combustion-derived PM (Sarnat et al., 2003).

c) Personal measurement devices

Almost all epidemiological data on the health effects of PM are based on fixed-location background measurements as a surrogate for personal exposure. There is however an increasing range of devices available for measuring ambient personal exposure, largely adapted from use in occupational settings. These devices typically consist of a small battery-operated pump, a size-selective sampling head and a collection system such as a filter (Expert Panel on Air Quality Standards, 2001b; Sarnat et al., 2003). Such equipment must be portable, preferably quiet, and able to sample the breathing zone of an individual, which extends approximately 30 cm from the nose and mouth. Apparatus ranges in size from passive badge samplers, worn for example as a lapel, to HVS pumps weighing about 3 kg, which can be used, for example, at the bedside for overnight monitoring.

Personal PM measurements typically give higher readings than background samplers. The discrepancy is often substantial, and predominantly influences the coarse particle fraction. It appears to be largely attributable to particulates generated by indoor activities such as

cleaning and cooking, as well as the generation of a "personal cloud" of PM consisting of skin flakes and clothing fragments (Expert Panel on Air Quality Standards, 2001b; Sarnat et al., 2003). Artefacts of this nature have clear implications for the use of personal monitoring devices in the context of monitoring exposure to crustal PM.

d) Sample preparation and particle properties

There is almost no published evidence on the influence of sample preparation following collection on the biological properties of PM. A single study from Finland suggested that methanol extraction and sonication of reasonable duration (when dissolving dried samples in liquids prior to exposure studies) were unlikely to modify the cytotoxic and inflammatory properties of ambient PM (Jalava et al., 2005).

SECTION 4. CONCENTRATIONS OF AIRBORNE PARTICULATE MATTER IN PORT HEDLAND

Summary

Currently available data on ambient particulate matter in Port Hedland are insufficient for delineating community exposure levels. Prospective monitoring based on measurements at multiple sites within the township, as well as the use of devices that monitor personal exposure is recommended.

In order to formulate appropriate guidelines for dust exposure at Port Hedland, it is clearly important to have accurate information on existing local conditions. Ambient dust concentrations in the township are likely to vary according to distance from ore stockpiles and possibly in relation to topographical contours. Additionally, temporal fluctuation in dust levels is expected, related to industrial activity and prevailing weather conditions, particularly wind speed and direction, rainfall and humidity. Airborne dust concentrations in the vicinity of Port Hedland have been measured for a number of years by BHP Billiton. There appear to be no systematic monitoring data from other independent sources.

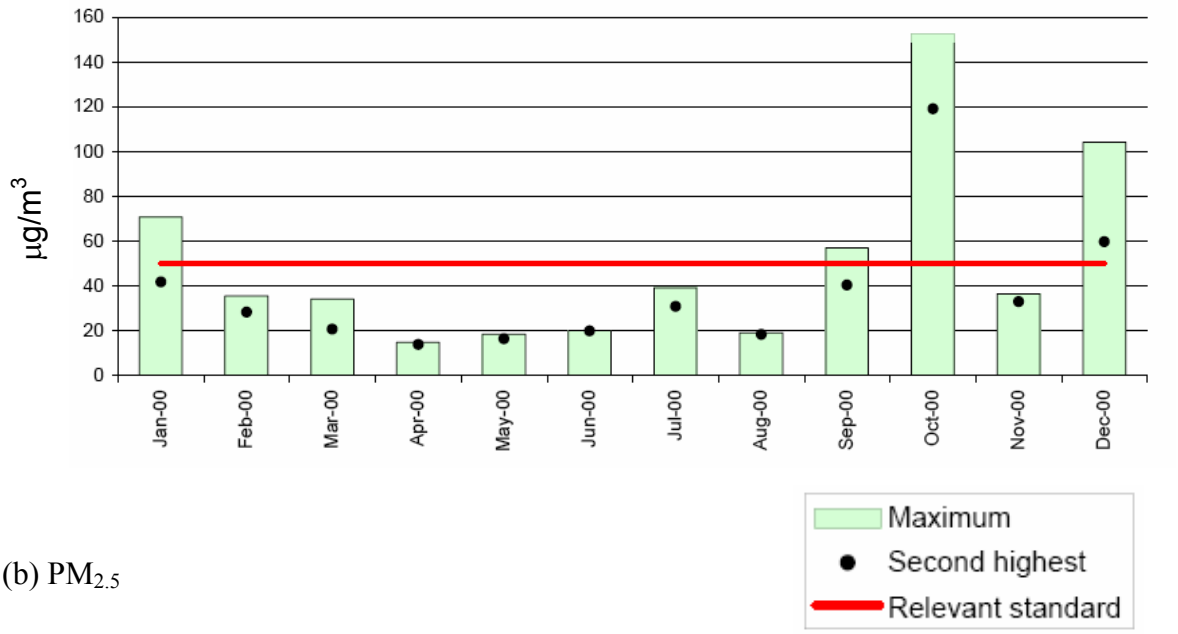
4.1 Existing data on dust levels in the Port Hedland area

Since the late 1970s, BHPBIO has used a High Volume sampling system to collect 24-hour measurements of Total Suspended Particulates (TSP) in the vicinity of Port Hedland. From 1996, regular measurements have been made at the Boodarie monitoring site (about 7 km from Port Hedland, close to South Hedland) using PM_{10}/$PM_{2.5}$ Tapered Element Oscillating Microbalance (TEOM) samplers, along with an assessment of TSP. The data have been summarised by the Western Australian Department of Environmental Protection in their report on the Monitoring of Ambient Air Quality and Meteorology during the Pilbara Air Quality Study (Department of Environmental Protection, 2002b). However, these data are clearly of limited relevance to ambient dust exposure levels at Port Hedland, given the distance of the Boodarie monitoring site from the town. Additionally, the Boodarie measurements may have been influenced by emissions from a nearby hot briquette production facility, which was operational for part of the period during which the measurements were taken.

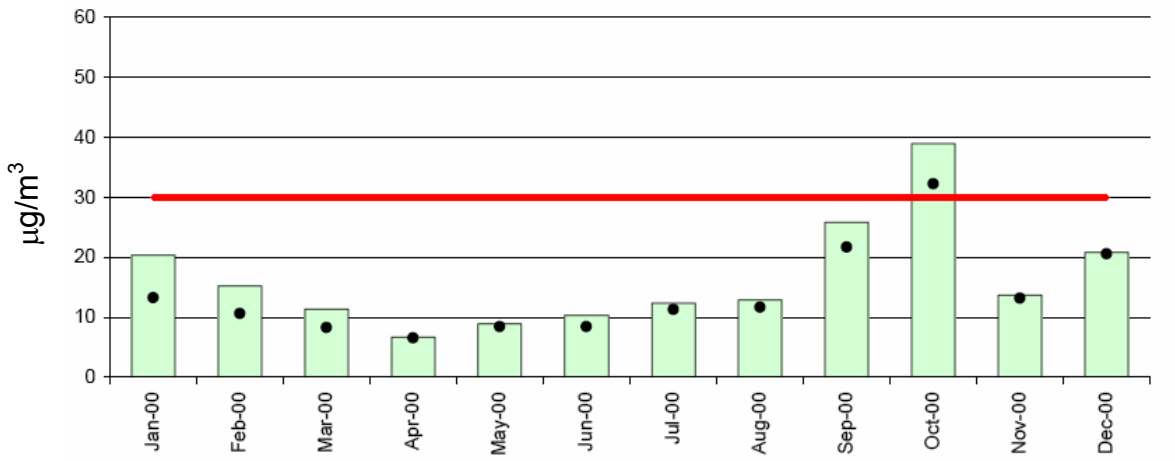
The 24-hour average PM_{10} concentrations at the Boodarie monitoring site ranged from about 20 $\mu g/m^3$ to approximately 175 $\mu g/m^3$ during the four years from 1997 (Department of Environmental Protection, 2002a). Median annual PM_{10} concentrations were all less than 25 $\mu g/m^3$. The corresponding data for $PM_{2.5}$ were consistently lower than the PM_{10}, with the minimum 24-hour average being approximately 10 $\mu g/m^3$ and the maximum about 42 $\mu g/m^3$. There were clear seasonal trends in both measures of airborne particulates, with the highest levels generally occurring between about October and March in each year. Figure 4.1 shows the monthly data for 2000 for both PM_{10} and $PM_{2.5}$. At Boodarie there were 12 exceedences of the AAQ-NEPM PM_{10} standard (50 $\mu g/m^3$ averaged over 24 hours) in 2000, and between 8 and 16 exceedences in each of the three preceding years.

Figure 4.1 Monthly PM10 (a) and PM2.5 (b) data for Boodarie for the year 2000
(Department of Environmental Protection, 2002a)

(a) PM_{10}



(b) $PM_{2.5}$



One important extraneous source of airborne particles highlighted by the Department of Environmental Protection is biomass burning (Department of Environmental Protection, 2002b). During the period from September to December 2000 several wildfires were started in north-west Australia due to lightning strikes. During the worst fire event, which was more than 300 km from the Boodarie monitoring station, the maximum 24-hour PM_{10} concentration was 150 $\mu g/m^3$ over about a four-day period.

4.2 Ideal monitoring strategy

Prospective measurement of dust conditions in the township, incorporating both fixed and personal measurement devices is recommended, and could form the basis of an epidemiological investigation. Ideally, monitoring of dust levels in the town would be derived from fixed measurement devices placed appropriately with respect to population distribution, at a sufficient number of sites to elucidate important spatial variations in dust levels. Data from the fixed devices could be supplemented by readings from small portable personal measurement devices distributed to a selected sample of the town population. Given the complexity of dust dispersion patterns within the town, computer modelling of data on measured dust concentrations with respect to site, land contour, population distribution, and meteorological conditions may be useful.

There is contention about the dust measurement device best suited to the specific meteorological conditions and dust profile at Port Hedland. Optimal monitoring may require the simultaneous utilisation of a variety of devices in order to circumvent the limitations of each. Measurement of both the PM_{10} and $PM_{2.5}$ fractions would be required, with the recognition that comparative measurement of these PM fractions provides only limited information on particle size distribution. More fundamentally, it is important to understand what is the specific nature of the particles in the various fractions and particularly in the respirable fraction.

SECTION 5. CRUSTAL DUST AND HEALTH

Summary

Exposure to airborne quartz carries the risk of silicosis, but only at prolonged exposure to concentrations of > 200 µg/m^3. Exposure to airborne non-fibrous silicates is associated with pneumoconiosis but only at very high concentrations seen in industrial settings. Sufficient exposure to iron ore dust can cause pneumoconiosis, but this is believed to be primarily due to the quartz component. Iron oxide *per se* is relatively non-toxic in this regard. Prolonged exposure to high concentrations of manganese in occupational settings is associated with neurotoxicity. There is no clear evidence of neurotoxicity from exposure to lower concentrations of airborne manganese in community settings. Haematite mining has been variably associated with a heightened risk of lung cancer, although this can probably be attributed to concomitant exposure to radioactivity rather than to the iron oxide *per se*. There appears to be an association between lung cancer and exposure to high concentrations of quartz or cristobalite dusts in occupational settings. It is unclear whether co-existing silicosis is a pre-requisite for carcinogenesis in this setting. It is unlikely that exposure to airborne manganese and copper ore is associated with an increased risk of cancer. Exposure to dust, including iron oxides, may result in minor "nuisance" effects such as irritation of the eyes, upper respiratory tract and skin.

5.1 Health impacts of exposure to crustal dust particulates, including quartz and clay, on population health

For the purposes of this review crustal dust components are defined as those components of the mixed dust sampled in the air that are not of commercial origin. It would be misleading to use a size metric to differentiate crustal from ore dust, as many iron particles, being of high density, have falling speeds that put their aerodynamic diameters well outside the conventional respirable range and would be regarded as "coarse dust" in air pollution terms. The dusts we have regarded as crustal are therefore quartz and various clays and silicates.

There is sufficient knowledge of the effects of quartz (crystalline silicon dioxide) to predict with reasonable accuracy the risks to human health from exposure to known concentrations. These risks are the development of lung fibrosis (silicosis) and, in certain circumstances, lung

cancer. Risks of silicosis only apply to workers exposed to relatively high concentrations. Ambient exposures have not been reported to lead to this disease, save among desert dwellers exposed to regular dust storms. In general it can be stated that average air concentrations of quartz below 50 $\mu g/m^3$ are unlikely to be associated with the risk of silicosis. Potentially disabling silicosis occurs only with exposure to higher concentrations, of the order of 200–500 $\mu g/m^3$, over prolonged periods. Furthermore, there is evidence from studies in the coal industry that the risks of quartz inhalation are reduced if the mixed dust inhaled also contains silicate minerals, such that in that industry concentrations of up to 10% quartz were not associated with a silicosis risk.

There is however a certain amount of evidence that inhalation of some non-fibrous silicate minerals themselves entails a risk of pneumoconiosis, a condition in this case characterised by largely non-collagenous nodule formation in the peripheral lung and only rarely with progression to a disabling form of disease. This has been recognised with exposure to kaolin, mica, oil shale and some coals, for example. The dust concentrations required to cause these variants of pneumoconiosis are greater than those required to cause silicosis, and are never likely to be reached in a non-industrial situation.

5.2 Health impacts of iron oxides

a) Experimental data from animals and humans

In a study in which guinea pigs were exposed to extremely high concentrations of iron oxide (as haematite) for 10 days, Carleton (1927/8) reported an immediate proliferation and thickening of the lung epithelium and bronchitis. Subsequently, the haematite was rapidly cleared from the lung and there was only very limited development of lung fibrosis. Given the inferred high levels of exposure and poor description of exposure concentrations, the results of this study are of limited value in the prediction of effects that might arise through community exposure to iron oxide in ambient air in Port Hedland. What the data do demonstrate, however, is that haematite appears to be considerably less toxic that other dusts such as quartz or coal mine dust. It should be noted, however, that ambient PM_{10} in Port Hedland does contain small quantities of quartz, and so is not directly comparable to a pure haematite dust.

In a more recent study, Hubbs et al. (2001) exposed rats to doses of 2,500 and 10,000 μg of a range of mineral dusts by intratracheal injection. Lung inflammation was assessed four weeks later from the lactate dehydrogenase (LDH – a marker of cellular damage) content and cell counts in lavage fluids. In this assay, haematite had an extremely small effect on LDH release at the higher dose level and no impact on the prevalence of inflammatory cells in lavage fluids. In contrast other abrasive blasting agents – garnet, coal slag, blasting sand and staurolite, all had marked effects on levels of LDH and cell counts.

In a human volunteer experiment, Lay et al. (2001) exposed 16 healthy subjects to aerosols of iron oxide particles (1.5 μm mass median aerodynamic diameter) for 30 minutes at an average mass concentration of 12,700 $\mu g/m^3$. Two preparations of particles were used with water-soluble iron contents of 3.26 and 0.14 μg/mg. The exposures did not cause an appreciable alteration of alveolar epithelial permeability, lung diffusing capacity, or lung function in healthy subjects. Therefore the limited toxicological data that is available does not suggest that inhalation of pure iron oxide in the form of haematite is significantly toxic to the lungs.

In cellular assays that measured the potential of dusts to cause cell damage and death (release of LDH) and to induce the release of inflammatory cytokines (IL-8), London Underground dusts were more toxic than titanium dioxide (Seaton et al., 2005). Two of three dust samples were more active than urban PM_{10} in the LDH assay and all three dusts were more active than urban $PM_{2.5}$ in the IL-8 assay. The Underground dust samples also caused significantly more damage to plasmid DNA (an index of free radical release) than urban PM_{10}, but had similar activities in the LDH and IL-8 assays to that of welding fume.

b) Welding fumes

For some, but not all, applications and steel types, welding fumes would be dominated by iron oxides. Taylor et al. (2003) demonstrated, for example, that different welding fumes caused varied responses in the lungs of rats that were correlated to their metal composition and ability to produce free radicals. Both the soluble and insoluble fractions of the fume were required to produce most effects, indicating that the responses are not dependent exclusively on the soluble metals. Antonini et al. (1998) demonstrated that freshly generated stainless steel welding fume induces greater lung inflammation than 'aged' fume, probably as a result of a higher concentration of reactive oxygen species on fresh fume surfaces. Particles in

ambient air might therefore be expected to have a much lower toxic potential that than those present in freshly generated welding fume.

Palmer et al. (2006) measured various markers of host defence function in sputum and venous blood samples collected from 27 welders with regular long-term exposure to ferrous metal fume and from 31 unexposed matched controls. The welders had significantly higher iron levels and a substantially lower unsaturated iron-binding capacity in their sputum, but showed little evidence of an inflammatory response. Only blood counts of eosinophils and basophils were significantly related to the extent of welding

Kim et al. (2005) measured the exposure and inflammatory response of 24 welders (42% smokers) exposed to a median $PM_{2.5}$ concentration of 1,660 $\mu g/m^3$ in comparison to 13 non-exposed controls (23% smokers) exposed to a median concentration of 40 $\mu g/m^3$. In non-smokers, welding fume exposure was associated with a significant increase in white blood cell counts and neutrophil counts in peripheral blood samples combined with a significant decrease in fibrinogen levels. No significant changes in white blood cell counts, neutrophil or fibrinogen levels were found in smokers. Sixteen hours after welding exposure, C-reactive protein levels were found to be significantly increased in both non-smokers and smokers.

Yu et al. (2004) exposed rats to manual metal arc-stainless steel welding fumes at concentrations of 65,600 +/- 2,900 (low dose) and 116,800 +/- 3,900 $\mu g/m^3$ (high dose) total suspended particulate for 2 hours per day for 30 days. When compared with unexposed controls, exposed rats showed a decrease in body weight combined with evidence of DNA damage and an inflammatory lung response, namely an elevated cellular differential count and higher levels of albumin, LDH, and beta-NAG, but not elevated TNF-α, and IL-1β in the acellular bronchoalveolar lavage fluid.

c) Epidemiological data on haematite mining

Haematite had been mined in Cumbria, in the English Lake District, since mediaeval and probably Roman times. The author AJ Cronin, while working there as a doctor in the 1920s, was probably the first to draw attention to the dustiness of the mines and to the prevalence of lung disease and tuberculosis among the miners. The suspicion that this was contributed to by silicosis resulted in an investigation by the UK Medical Research Council. They reported in

1933 that a specific form of pneumoconiosis affected haematite miners, and this led to action in the mines to control dust levels. In 1947, Craw reported a marked reduction in risk of pneumoconiosis among younger miners, employed after dust control measures had been implemented (Craw, 1947). Some idea of the concentrations that might have prevailed and been responsible for pneumoconiosis comes from the study of dust levels in these mines by Bradshaw et al. (1962). By that time concentrations were regarded as "safe" but averaged between 2,000 and 5,000 $\mu g/m^3$ during drilling and reached 14,000 $\mu g/m^3$ after shot firing. Detailed pathological and mineralogical studies of lungs of deceased miners led Faulds and Nagelschmidt (1962) to conclude that the pneumoconiosis was similar to that occurring in coalminers and distinct from classical silicosis. In other words, it seemed likely that the inhalation of haematite in large quantities could by itself cause the disease.

Two unusual reports have drawn attention to the possibility of disease in iron ore workers being caused by other associated minerals. Edstrom and Rice (1982) reported "Labrador lung" in 48 subjects with pneumoconiosis resulting from a dry grinding process associated with open pit iron ore mining. Dust exposures were described as being between 3 and 7 times the relevant threshold limit values for the components of the dust. The men had a diffuse interstitial fibrosis and their lungs contained not only iron oxide and quartz, but also anthophyllite asbestos. The findings suggested a mixed pneumoconiosis due mainly to quartz but with contributions from asbestosis. Although the men were relatively well at the time, it was anticipated that further problems might well be identified at a later stage if they were followed up. No further record of this condition can be found in the literature. Nolan and colleagues (1999) described the finding of a seam of amosite asbestos in an iron ore mine in the United States, and reported a risk assessment for workers exposed to this dust. The average airborne fibre concentration from 179 personal samples collected from men employed in the mine was 0.05 fibres/ml.

Two papers give some indication of the dust concentrations to which men developing pneumoconiosis had been exposed. Chen et al. (1989) reported on a cohort of 5,406 Chinese underground miners with a 7-8 fold increase in risk of death from non-malignant respiratory disease among the highest exposed. These exposures had averaged 2,000-9,000 $\mu g/m^3$ in recent years but it was admitted that exposures may have been up to hundreds of thousands of $\mu g/m^3$ earlier. It is not clear from the paper what proportion of the dust was quartz (Chen et

al., 1989). Moore et al. (1987) reported a case-control study of 40 Canadian iron ore surface miners with International Labour Organization (ILO) category 1 or greater radiological changes. Careful estimates of individual exposure were based on detailed occupational histories and fixed-point workplace dust monitoring results. For early pneumoconiosis, peak exposure to quartz and cumulative exposure to dust related to risk of radiological change, while for more advanced disease (categories 2 and 3 pneumoconiosis according to the scale devised by the ILO), both peak and cumulative exposures to dust and quartz were significantly associated with radiological change. In neither case did exposure to iron oxide influence the risk. Median cumulative exposures of the men with pneumoconiosis to dust and to quartz, respectively, appear to have been 80,000 μg.years/m^3 and 10,500 μg.years/m^3. This equates to 4,000 μg/m^3 of dust and 580 μg/m^3 of quartz for 20 years, higher exposures than one would expect in open pit mining generally.

One paper of particular relevance to the Pilbara mining operations was published by Musk et al. (1988). They commented on the possible exposure of some miners in this area to quartz and fibres (noting that the Marra Mamba ore in particular had been associated with fibrous minerals). In a cross-sectional radiological survey of 788 miners they were able to show an association of radiological change with age and years of mining work, but no dust exposure indices or smoking histories were available. They were thus unable to conclude whether or not these changes were true pneumoconiosis, but ended with a caution that they might well be, and recommended further investigation.

In conclusion, there is no doubt that sufficient exposure to iron ore dusts can cause pneumoconiosis. The evidence suggests that this is primarily due to the quartz component and that iron oxide in the concentrations to which workers have been exposed is relatively non-toxic in this respect. Concern has been raised about the possible carcinogenicity of iron oxide, but in this industry it seems more likely that excesses of lung cancer have been due to irradiation from underground radon. The exposures associated with the development of pneumoconiosis have been high, of the order of 1,000 μg/m^3 of mixed dust or 50 μg/m^3 of quartz over several decades. It would be prudent to assume that such exposures would also, as with coal dust, be expected to increase risks of chronic obstructive lung disease, although there is no direct evidence of this.

5.3 Health impacts of quartz

Respirable silica (or quartz) was one of the earliest recognised occupational hazards, particularly in quarries and mines. Studies have been carried out in a number of occupational groups, and have amassed ample evidence of respiratory effects of exposure. The main risk has been of developing fibrotic nodules (silicosis) in the lung tissue. In the smaller number of areas where quantitative exposure estimates have been available, there has also been clear evidence of exposure-response relationships for silicosis.

However, a number of factors complicate the picture and make it hard to derive definitive exposure-response relationships. Many less hazardous mineral dusts demonstrate a response that relates well to estimates of cumulative exposure, i.e. the cumulative product of time spent and concentration experienced in various jobs over a working lifetime. Once deposited in the lungs, however, silica remains biologically active and hazardous for a long period, so the length of residence is an additional factor. This means that, while progression of pneumoconiosis due to other mineral dusts is likely to cease after exposure ceases, silicosis can develop anew or progress well after exposure to silica has ended. In addition, there is good evidence that the risks of silicosis do not increase uniformly with increases in airborne concentrations, but increase disproportionately at higher concentrations.

A further complication is that the risks of silicosis vary, depending on other characteristics of the exposure. The most hazardous form is pure crystalline silica with freshly fractured surfaces, which are particularly active in creating or promoting biological effects. This activity can be greatly reduced by the presence of other substances, including some metallic ions, for instance aluminium. This may explain why exposure to silica in clays, which contain aluminium compounds, is associated with much lower risks than exposure to pure, fresh silica.

Some of the most detailed exposure-response data on silica has come from a group of coalmine workers, some with atypical exposures to respirable silica abraded from sandstone strata (Buchanan et al., 2003). These data included very detailed exposure measurements, and the derived exposure-response relationships have been a primary influence on British occupational limits for silica. Since the coalmine dusts contained up to 25-30% of silica, these data also demonstrate that, where silica exposures are high, the presence of other components such as coal and even clay minerals does not necessarily protect against silicosis.

The current occupational exposure limit for respirable crystalline silica is 100 μg/m^3, although there have been recommendations that the limit of exposure, below which the risk of silicosis is considered negligible, should be lowered to 50 or 40 μg/m^3 (WHO, 1986; Rosenman et al., 1996; Finkelstein, 2000). More recently the Californian Office of Environmental Health Hazard Assessment has proposed a reference exposure limit of 3 μg/m^3 for chronic community exposure to respirable crystalline silica (California OEHHA, 2005). The derivation of such a limit is supported by long-term studies of the incidence of silicosis following occupational silica exposure, and the finding of a dose-response effect for silicosis in several studies. However, the no observed adverse effect levels varied widely from 7 to 100 μg/m^3 (Rice & Stayner, 1995).

5.4 Health impacts of manganese

The main health effect of concern that is associated with exposure to airborne manganese is neurotoxicity. Workers who have experienced prolonged exposure to high concentrations of airborne manganese compounds (>1-5 mg Mn/m^3) (Dobson et al., 2004) may develop symptoms similar to those observed in Parkinson's disease, including tremor and loss of motor control, although the two diseases are distinct (Olanow, 2004). Pathological data from rodents, primates and humans indicate that manganese neurotoxicity results in neuronal loss and gliosis of the basal ganglia, principally affecting the globus pallidus (Olanow, 2004). Effects may also include mood disturbances, irritability, apathy and anorexia. In addition to neurological effects, high levels of exposure to manganese in workplace air have been associated with respiratory effects (Levy et al., 2004). Most of the available epidemiological information is from workplace studies, but a number of investigators have also examined the possible effects of environmental exposure to manganese.

a) Neurobehavioral effects

A number of studies have demonstrated subtle neurotoxic effects associated with exposure to low concentrations of manganese in workplace air. Some of the studies report a manganese cumulative exposure index in addition to cross-sectional manganese dust exposure levels, although none has provided well-defined exposure-response relationships. Additionally, not all of the studies explicitly distinguish manganese concentrations in the respirable fraction of dust from those in total dust.

The effects found at the lowest exposure concentrations would not have a detectable effect on an individual's quality of life or workplace performance. It is not clear whether these effects are associated with an increased risk of neuro-degenerative disease in later life, although there is no evidence to link prolonged low level manganese exposure to an increased risk of Parkinson's disease in humans (Aschner, 2000).

In a study of battery workers exposed to manganese dioxide at mean concentrations of manganese in respirable dust of 200 $\mu g/m^3$, 15% of workers developed subtle neurological symptoms that could be detected in appropriate tests (Roels et al., 1992). An earlier study of chemical workers exposed to a wider range of manganese compounds had detected effects on reaction time, short term memory and hand steadiness, in workers exposed to mean concentrations of 970 $\mu g/m^3$ as manganese in total dust (Roels et al., 1987). Mergler et al. (1994) reported effects on motor function in metal alloy workers exposed to manganese concentrations of 1,200 $\mu g/m^3$ in total dust and 120 $\mu g/m^3$ in respirable dust. Bast-Pettersen et al. (2004) found increased postural tremor, compared with a control group, in manganese alloy workers exposed to mean concentrations of manganese in inhalable dust of 300 $\mu g/m^3$.

An excess of subtle neuropsychological symptoms (emotional irritability, memory disturbance, impaired concentration and sleepiness) was reported among workers in the shipping and electrical industries who were exposed to manganese concentrations averaging 400 $\mu g/m^3$. However, no objective neurological findings were detectable (Sinczuk-Walczak et al., 2001).

In a Canadian study of manganese alloy production workers, exposed to a respirable manganese concentration of 230 $\mu g/m^3$, evidence was found of an interactive effect on mood states of blood Mn levels and reported alcohol consumption (Bouchard et al., 2003).

However, Gibbs et al. (1999) found no evidence of neurological damage in metal workers exposed to mean respirable concentrations of 0.04 mg/m^3. Myers et al. (2003a) found no effects in miners exposed to total manganese concentrations of 200 $\mu g/m^3$ that have been interpreted to be equivalent to respirable concentrations of about 40 – 80 $\mu g/m^3$ (Levy et al., 2004). In smelter workers exposed to concentrations that generally exceeded 200 $\mu g/m^3$, Myers et al. (2003b) found evidence for effects in selected tests such as hand tapping, but no

evidence of tremor. In a case-control study of enamel production workers, exposure to a manganese concentration of approximately 200 μg/m^3 over a period averaging nearly 20 years resulted in no detectable disturbance of function as assessed by a battery of neuropsychological tests, although the exposed workers reported more non-specific subjective health complaints than the controls (Deschamps et al., 2001). Psychomotor function scores in a sample of ferro-alloy workers exposed to airborne manganese oxide, at a concentration in total dust of 193 μg Mn/m^3 for an average duration of 14.5 years, did not differ significantly from those in controls (Lucchini et al., 1997).

Even in the absence of clinically evident neuropsychological abnormalities, prolonged occupational exposure to manganese dust at concentrations < 400 μg/m^3 may result in abnormalities of the basal ganglia that are detectable by magnetic resonance imaging of the brain, namely, increased signal intensity in the globus pallidus on T_1-weighted images (Dietz et al., 2001).

The typical duration of exposure in most workplace studies has been less than 20 years and, in most studies, the mean duration of exposure was less than 10 years. There is therefore little information about the longer term effects of continued manganese exposure. Notably however, Hochberg et al. (1996) detected a significant excess of movement abnormalities (tremor and impaired repetitive hand movements) among asymptomatic former miners with a history of chronic exposure to manganese ore dust (mean duration > 20 years), ending at least 5 years previously.

There is limited evidence to suggest that symptoms cease to progress following a reduction in exposure (Lucchini et al., 1999; Crump and Rousseau, 1999). In the Lucchini study, the reported range of manganese concentrations decreased from 70 – 1,600 μg/m^3 to 30 – 270 μg/m^3 as manganese in total dust. Roels et al. (1999) found some limited evidence for the possible reversibility of effects in battery workers following a reduction in exposure. In their study, mean concentrations of manganese in total dust had fallen from 800 to 250 μg/m^3 over an 8 year period.

It is not known how individual manganese compounds vary in their toxicity for humans. The characterisation of exposure in workplace studies has generally been reported only in terms of

manganese metal concentrations in air or blood. It seems likely that the manganese compounds present in ambient air in Port Hedland would be similar to those to which manganese miners are exposed, and that miners would therefore provide the most appropriate comparison group as, for example, in the study by Myers et al. (2003a).

A number of studies have examined the effects of environmental exposure to manganese on health. These have included both studies of populations exposed to airborne manganese, as well as populations exposed to manganese in drinking water. Considerable public controversy has arisen over the potential hazard of exposure to airborne manganese as a result of the use of the petrol additive, methylcyclopentadienyl manganese tricarbonyl (MMT), although the additional Mn exposure resulting from inhalation of MMT combustion products is likely to be minimal (< 0.15 $\mu g/m^3$) for at least 99% of the general population (Kaiser, 2003; Finley, 2004). It is unlikely that the manganese compounds typically associated with elevated environmental manganese exposure arising from the use of MMT would be directly comparable with those arising from windblown dusts generated from manganese ore. The drinking water studies are also unlikely to be of direct relevance to understanding the potential impact of inhaled manganese dust, because of the differences in the kinetics of absorption of manganese from the gastrointestinal and respiratory tracts.

Although the results of some studies of environmental exposure to manganese have suggested possible links to neurodegenerative disease, other studies have failed to find an association. Rodríguez-Agudelo et al. (2006) assessed alterations in motor function among 288 individuals from eight communities at various distances from manganese extraction and processing facilities in a Mexican mining district. Ambient manganese concentrations averaged 0.42 $\mu g/m^3$ (range 0.003 – 5.86 $\mu g/m^3$). An extensive battery of over 20 neuromotor tests was performed on the subjects. Associations between community manganese exposure and neuromotor function that were of marginal statistical significance, were documented for only two of the neuromotor tests.

In a Canadian study of 273 subjects exposed to manganese from a variety of environmental sources, including residents living downwind of a manganese alloy plant, Mergler et al. (1999) reported an association between raised manganese concentrations in blood and reduced performance in neurobehavioral and neuropsychiatric tests. Mean total-particulate airborne manganese concentrations in ambient air were 0.022 $\mu g/m^3$ with a range of 0.009 to

0.035 μg/m^3 across four sampling sites (Hudnell, 1999). Neurological effects were more common in older individuals (over 50 years of age). It is not clear whether this reflects confounding in the study design (i.e. effects attributed to manganese were actually due to the normal processes of aging) or an increased susceptibility of the elderly to manganese toxicity. In contrast, Smorgon et al. (2004) found no association between blood manganese concentrations and cognitive function in a study of 35 elderly patients.

In a comparison of populations exposed to 3.6 – 15 μg/l, 82 – 250 μg/l, and 1,800 - 2,300 μg/l, respectively, of Mn in drinking water, Kondakis et al. (1989) found evidence of neurological symptoms in the highest exposure group. Consumption of 2 litres of water per day at the highest manganese concentration would have given an intake similar to that associated with exposure to 230 μg/m^3 in ambient air (assuming comparable levels of absorption). In a comparison of a population exposed to more than 300 μg/l (range 300 to 2,160) of Mn in drinking water with a population exposed to less than 50 μg/l, Vieregge et al. (1995) found no detectable neurological impairment or increased risk of Parkinson's disease in the high exposure group. Consumption of 2 litres of water per day at the highest concentrations would have been similar to exposure to 210 μg/m^3 in ambient air. Although it seems possible that high levels of exposure to manganese in drinking water might have adverse effects, the level of manganese intake in these studies far exceeds what is likely to arise from ambient air. The drinking water studies are therefore not particularly informative as to potential risks associated with airborne manganese at environmental concentrations. Overall there is no strong evidence to suggest that elevated levels of exposure to manganese in ambient air are associated with an increased risk of developing neurodegenerative disease.

b) Respiratory and other potential adverse health effects

Roels et al. (1987) found some evidence of subjective respiratory symptoms and reduced lung function in chemical workers exposed to mean concentrations of manganese in respirable dust of about 1,000 μg/m^3. However, in their later study of battery workers exposed to manganese concentrations of about 200 μg/m^3, Roels et al. (1992) found no evidence of an association between manganese exposure and either lung function or respiratory symptoms. In a study of miners exposed to manganese concentrations in respirable dust of 43 μg/m^3 (arithmetic mean), Boojar and Goodarzi (2002) reported associations between manganese and respiratory symptoms and also lung function. Given such high exposure concentrations, the observed

effects may have been a non-specific response to dust exposure rather than to manganese specifically.

A number of studies have investigated the role of metals in causing the toxic effects of environmental particulate air pollution on the respiratory system. Typical concentrations of manganese in urban air are extremely small. In a Canadian study, Bolte et al. (2004) reported respirable manganese concentrations of 0.025 $\mu g/m^3$ in urban air compared with 0.005 $\mu g/m^3$ in rural air. A substantial proportion of the manganese in urban air came from the use of MMT. Although the results from animal toxicity studies suggest a possible role for trace metals, including manganese, in the development of airways inflammation, there is no clear specific link to manganese (e.g. Pagan et al., 2003; Dye et al., 2001). Magari et al. (2002) reported an association between exposure to airborne metals, including manganese, and significant alterations in cardiac autonomic function in humans, but the effects were not specific to manganese.

c) Conclusions

The main health effect of concern associated with exposure to manganese is neurotoxicity. High levels of exposure in the workplace have also given rise to respiratory disease, but the specific importance of manganese as opposed to dust exposure in causing respiratory illness is unclear. The results of studies of the health effects of environmental exposure to manganese have been inconclusive. There is limited evidence that suggests that manganese in combination with other metals may play a role in the toxic effects of particulate matter in urban air, but there is no evidence that manganese is of particular importance as opposed to other transition metals.

The results of workplace studies suggest that long term exposure to average manganese concentrations exceeding approximately 200 $\mu g/m^3$ in respirable workplace dust may give rise to adverse effects on performance in neurobehavioral tests. No effects have been observed following exposure to workplace concentrations of 40 – 80 $\mu g/m^3$ of manganese in the respirable dust fraction. Workplace exposure to respirable manganese concentrations of 200 $\mu g/m^3$ over a calendar year would be approximately equivalent to continuous exposure to 40 $\mu g/m^3$ in ambient air over the same time period. The comparable concentration of

manganese in PM_{10} would be slightly greater because PM_{10} includes a larger proportion of airborne particles than the respirable fraction.

5.5 Health impacts of other non-fibrous silicates

The silicates of relevance in this context are likely to be kaolinite and mica. Kaolinite has been exploited commercially in the china clay industry and it is recognised that exposure to the fine dust resulting from drying processes may lead to a pneumoconiosis similar to that of coalminers. Exposure to powdered mica has also been reported to lead to a similar pneumoconiosis, although it appears that many fewer workers have been at risk of this condition.

Records of a dust hazard in relation to exposure to kaolinite go back to the 1930s in the UK, and the term kaolinosis was introduced in the USA by Lynch and McIver (1954) to denote a characteristic pneumoconiosis among these workers. The pathological changes in six patients, and the presence of kaolin in their lungs were described by Gough and co-workers (1956). In spite of these reports, the dust was widely regarded as harmless until a high prevalence of the condition was described in Cornish china clay workers (Sheers, 1964). It was noted that the risk was related to duration of exposure and was markedly greater among workers exposed to high dust levels, including millers, baggers and loaders. It is clear that very high concentrations of dust pertained in this industry at that time, but no measurements were recorded. A further prevalence study was reported among these workers (Oldham, 1983). Again, an association was observed between likely (but not measured) dust exposure and risk of pneumoconiosis and the presence of radiological change was associated with some loss of lung vital capacity but not the forced expired air volume in one second (FEV_1). Wagner and colleagues (1986) drew attention to the occasional influence of quartz dust in causing silicosis among workers exposed to china stone but showed that in the majority of subjects with kaolinosis there was an interstitial fibrosis, associated with high concentrations of kaolin in the lungs (up to 100 mg/g dry lung tissue) at autopsy. Morgan and colleagues (1988) reported a large prevalence study of US kaolin workers. Again the risk of pneumoconiosis appeared to be related to duration and intensity of dust exposure, being greater among workers involved in dry processing work. In this case, functional abnormalities were found in the FEV_1 in relation to dustiness of work. Finally, Baser and colleagues (1989) reported that the risks of pneumoconiosis differed between two plants in the US, and in the one with the higher risk there was evidence of deterioration in vital capacity.

It is striking that in spite of the evidence suggesting dust exposure-related effects on radiographs and lung function, no papers prior to 1989 were able to comment on the basis of

actual measurements of dust exposure among different categories of china clay workers. In the Cornish industry, to which most of the above reports refer, according to Ogle and colleagues (1989), "…measurements of a rudimentary nature had been carried out since 1965 but only since 1978 had personal respirable and total dust measurements been made". It was claimed in this paper that many changes had been made during the 1970s to reduce dust exposure, with a reduction in airborne concentrations. At the time of the survey, average personal respirable dust exposure among driers, calciners and millers ranged from 1,900 to 2,700 $\mu g/m^3$. The authors did not use this data to make estimates of exposure, rather relating disease to duration in different occupations. There was a clear exposure duration-response relationship in terms of radiological change and also an association of such change with vital capacity. However, the authors commented that exposures were such that it was likely that "the average worker exposed to dust only after 1971 would not expect to develop category 1 pneumoconiosis through a full working life". However in 1993, the authors of a further study of all china clay workers in the UK did make estimates of exposure, based on measured concentrations since 1978 and estimated concentrations before that date (Rundle et al., 1993). They showed that radiological appearances of pneumoconiosis increased in relation to increasing exposure to kaolin, and that FEV_1 declined in relation to radiographic category. Although they did not report the relationship between exposure and FEV_1, their data were used by the Health and Safety Executive (HSE) in producing a Criteria Document for Kaolin (Standring et al., 1994). The HSE proposed an occupational exposure limit of 2,500 $\mu g/m^3$ respirable dust, on the basis that 40 years' exposure to this mineral over 8-hour working shifts was associated with an average radiological score for pneumoconiosis of 1, a level at which disablement did not occur. The average decrease in FEV_1 for this level exposure was estimated to be 220 ml.

The evidence with respect to exposure to respirable kaolin is thus unequivocal that at industrial concentrations it may cause both radiological pneumoconiosis and decline of lung function. The UK OEL of 2,500 $\mu g/m^3$ refers to eight hours of exposure daily for 40 years. On the assumption that general environmental exposure could theoretically last 24 hours daily, for say 80 years, similar consequences would be associated with a concentration six times lower, so consideration of an ambient standard for kaolin should start at approximately 400 $\mu g/m^3$ and would normally include a safety factor to take account of variations in population susceptibility.

Reports of adverse effects of mica are scanty in the literature, and some individual case reports may well have documented coincidental lung disease in workers exposed to the mineral. However, there is sufficient evidence to conclude that a specific pneumoconiosis may occur in heavily exposed workers. Dreesen et al. (1940) found an 18% prevalence of radiological change among workers exposed to ground mica, while Vestal et al. (1943) found an 11% prevalence among grinders and millers. No recent papers have reported pneumoconiosis or adverse pulmonary effects in workers exposed to mica.

In discussing these minerals, it should be noted that kaolin and other clays are normal constituents of coal and are inhaled by coalminers along with the carbon and quartz that are the more toxic components of the coal dust. In such circumstances there is evidence that the main role of clays is to reduce the toxicity of quartz by coating the active surface of the crystals.

5.6 Occupational and community exposure to mine dusts – the analogy of coal-dust

A number of studies have shown coalmine dust to have a greater toxicity than titanium dioxide. In animal inhalation studies, coalmine dust showed toxicity intermediate between that of titanium dioxide and quartz (Donaldson et al., 1990). The results of cellular assays are less clear-cut. Churg et al. (1997) reported that coal was more active than quartz in an assay to determine the relative potential to cause breakdown of connective tissue in the airways and, by inference, would have a greater potential to cause emphysema. In an assay of the potential of dusts to cause nitric oxide (NO) production following intratracheal instillation in rats, Blackford et al. (1997) found that when exposure was normalised for an equal number of particles, crystalline silica and coal caused more inflammation and NO production than titanium dioxide. In assays with human and non-human macrophage cell lines, Kuhn et al. (1993) reported that the inflammatory response, as determined by eicosanoid production following a single bolus exposure, decreased in the order silica > anthracite > bituminous coal > titanium dioxide.

The results of a number of US studies of coalmines have suggested adverse respiratory effects following long-term exposure to mean coal dust concentrations of < 2,000 $\mu g/m^3$ at work (the occupational exposure standard). Goodwin and Attfield (1998) reported on the occurrence of pneumoconiosis in workers who were employed after the introduction of the 2,000 $\mu g/m^3$ limit. An analysis performed by Kuempel et al. (1995) established that miners exposed at or

below the U.S. coal dust standard of 2,000 μg/m^3 over a working lifetime had an elevated risk of dying from pneumoconiosis, chronic bronchitis or emphysema. In a study of 1,866 miners, Henneberger and Attfield (1997) found evidence of shortness of breath and wheeze associated with exposures controlled to the 2,000 μg/m^3 coal mine dust standard. Seixas et al. (1993) reported a significant non-linear effect of exposure to dust on pulmonary function, at dust concentrations present after the implementation of the 2,000 μg/m^3 standard, with effects being greatest on initial exposure. The initial changes in both FVC and FEV_1 were stated to be consistent with inflammation of the small airways in response to exposure to dust. The equivalent concentration for lifetime exposure in ambient air would be about 200 μg/m^3, as respirable dust. A study of 3,194 underground bituminous coal miners and ex-miners established that 40 years exposure to coal mine dust, at the federal dust limit of 2,000 μg/m^3, was associated with a 1.4% risk of having progressive massive fibrosis on retirement (Attfield and Seixas, 1995). In a study of over 9,000 US miners, Attfield and Morring (1992) reported that 40 years exposure to an average concentration of 1,000 μg/m^3 of respirable coal mine dust at work was associated with a risk of developing Category I (or greater) pneumoconiosis of between 5.5 and 12.8%, and a risk of developing Category II (or greater) pneumoconiosis of between 1.4 and 4.6%. Category I pneumoconiosis, while identifiable on chest X-rays would not be expected to give rise to noticeable respiratory illness. Concerns have been expressed about the reliability of the dust concentration data used in US studies (Brower and Attfield, 1998). Under-measurement of dust concentrations would give rise to inflated estimates of disease risk.

It is therefore well recognised that miners occupationally exposed to prolonged high levels of coal dust are at risk of developing lung disease (coal workers' pneumoconiosis), even in the absence of concomitant significant exposure to silica (Castranova and Vallyathan, 2000). However, the health effects of coal dust on communities living in proximity to opencast coal mines are uncertain. A study of community exposure to mineral dust arising from opencast coal mining in the UK found no evidence to link acute respiratory symptoms in children to residential proximity to opencast operations (Howel et al., 2001a; Pless-Mulloli et al., 2001). There was limited evidence of an increase in GP consultations for children living in communities close to opencast workings relative to those living in more distant communities (Howel et al., 2001b).

5.7 Carcinogenesis

a) Introduction

Assessment of the human carcinogenicity of a given substance is based on data from observational epidemiology, supplemented by experimental studies in animals and *in vitro* models (Wogan et al., 2004). Establishing the carcinogenicity of environmental agents is not straightforward. Interpretation of data from animal experiments for this purpose is problematic, given inter-species variation in biological responses, and the uncertain relationship between the generally extreme doses of potential carcinogens administered in animal models compared with real-life human exposures (Luch, 2005). It is well known that interpretation of epidemiological studies on chemical carcinogenesis is potentially fraught because of unmeasured confounding factors, biases in the selection of subjects and in ascertainment of exposures and health outcomes, and chance effects (Hennekens and Buring, 1987).

Additionally, epidemiological studies on the carcinogenicity of inhaled dusts are generally conducted in occupational settings, in which levels of exposure to dust are very high. There are unavoidable caveats on extrapolating inferences on carcinogenicity from these studies to lower level exposures encountered in non-occupational settings. Linear non-threshold models have traditionally been applied in low-dose risk extrapolation for carcinogens, but the principles underlying this convention remain controversial (Calabrese and Baldwin, 2003).

b) Exposure to haematite/ferric oxide dusts

The carcinogenicity of iron oxides has been a matter of some contention. Epidemiological examination of the subject has been hindered by the fact that exposure to “pure” iron oxides is exceptional. Mining and processing of iron ores is commonly associated with concomitant exposure to other agents believed to be potentially carcinogenic including ionizing radiation, silica dust, amphibole asbestos fibres, and fumes from combustion of fossil fuels such as diesel (Stokinger, 1984).

(i) Experimental data

The plausibility of a cancer risk associated with exposure to iron oxide dusts can be established by *in vitro* carcinogenicity tests. The scanty published *in vitro* data have not confirmed a carcinogenic role for ferric oxides such as haematite dust in the absence of other carcinogens such as polycyclic aromatic hydrocarbons. An early study showed an increase in

lung tumours in mice exposed for long periods to dust composed of precipitated ferric oxide (Argyll Campbell, 1940). The interpretation of this finding is uncertain as no statistical inferential data were reported. This purported evidence for the carcinogenicity of "pure" iron oxide dusts has not been replicated in other animal experiments. In a subsequent study on rats, intratracheal instillation and intraperitoneal injection of a range of test iron oxides did not induce tumours (Steinhoff et al., 1991). Ferric oxides are believed not to be genotoxic and are generally not active in the production of free radicals (Costa et al., 1989).

(ii) Epidemiological studies

There are a number of published epidemiological studies on cancer and occupational exposure to iron oxide dusts among miners. A risk of lung cancer caused by exposure to iron oxide was first mooted on the basis of autopsy data from haematite miners for the period 1930-1967 in the Cumberland region of the United Kingdom. However, these data were age-confounded and unavoidably biased by autopsy practices (Anonymous editorial, 1970). Subsequently, a number of studies have documented an increased risk of lung cancer among underground miners of a range of metallic ores, including iron ore (Boyd et al., 1970; Edling, 1982; Edling and Axelson, 1983; Radford and Renard, 1984; Jorgensen, 1984; Damber and Larsson, 1985; Chen et al., 1990). The increased risk has been documented even among non-smoking miners (Edling, 1982; Radford and Renard, 1984). Smoking and underground iron ore mining appear to act additively (Edling and Axelson, 1983; Radford and Renard, 1984), or perhaps multiplicatively (Damber and Larsson, 1985), in generating excess lung cancer risk. Early studies suggested that the effect is specific for lung tumours, i.e., it is not accompanied by an accentuated risk of non-respiratory tumours (Boyd et al., 1970), although data published more recently highlight a possible association of iron ore mining with malignancies outside the respiratory tract, including cancers of the stomach and rectum (Darby et al., 1995).

Importantly, however, the excess lung cancer risk appears to be restricted to mines in which there are excessive levels of ambient radioactivity. Uranium is frequently present in association with deposits of a range of metallic ores. Radon, the gaseous product of the radioactive decay of uranium, is formed directly from radium. Such decay products are readily dissipated in the atmosphere but can become highly concentrated in an underground mine setting, especially when ventilation is poor. Although radon itself releases relatively little radiation, its short-lived daughter products, including polonium-218, lead-214, bismuth-

214 and polonium-214 are highly radioactive. They are readily adsorbed onto ambient dust particles, inhaled and deposited in the tracheobronchial tree, where they deliver a potentially carcinogenic dose of radiation (Harley, 1984). Coal deposits are typically not associated with uranium and its decay products, and studies of coal miners have not shown an elevated risk of lung cancer (Boyd et al., 1970; Miller and Jacobsen, 1985). The causal relationship between underground radioactivity and lung cancer risk is supported by the high magnitude of hazard in settings where exposures are extreme (Greenberg and Selikoff, 1993). There is a clear biological gradient between occupational exposure to radiation and risk (Radford and Renard, 1984).

No excess lung cancer risk was evident in an American iron ore mine in which background levels of alpha radiation were relatively very low (Lawler et al., 1985). A corresponding increase in lung cancer has not been seen among surface workers in the same mines, who were exposed to very high levels of iron oxide dust, but not to the high levels of radon daughters experienced by their counterparts working underground (Boyd et al., 1970; Radford and Renard, 1984). Additionally, the increased lung cancer risk is not evident among workers in secondary industries, such as processing plants, in which measured levels of iron oxide dust are extremely high but exposure to radioactivity is negligible (Axelson and Sjoberg, 1979).

One discordant finding requires specific mention. A series of studies were published purporting to show an increased lung cancer risk among a cohort of iron ore miners in Lorraine, France, despite the low levels of radiation that were measured (Pham et al., 1983; Pham et al., 1992; Mur et al., 1987). It was conceded, however that unmeasured historical levels of radon daughters in this mine, prior to the advent of improved underground ventilation measures, may have been materially higher than those measured close to the time that the retrospective cohort study was undertaken (Pham et al., 1983). Given the long latency of the disease, these unmeasured historical radiation levels are likely to have been pertinent to the aetiology of many of the recorded cases of lung cancer.

In summary, although haematite mining is variably associated with excess risk of lung cancer, the available epidemiological data generally do not strongly support a causal relationship between exposure to haematite dust and carcinogenesis (Stokinger, 1984). Experimental data lend low plausibility to the documented association, which is strongly confounded by

exposure to known carcinogens, and is not entirely consistent. Although an association between lung cancer and exposure to dusts containing iron oxides has been noted in the mining context in several locations worldwide, the heightened cancer risk can probably be attributed to concomitant exposure to radioactivity on several grounds. The association has been consistently observed in a variety of mining settings, in which dusts of the ore *per se*, such as zinc, lead and gold are believed not to be carcinogenic, but where there is concomitant exposure to radon daughters (Edling, 1982). The consistent observation that the excess cancer risk appears to apply exclusively to underground miners of haematite, and not to their peers employed at the mine surface (Boyd et al., 1970; Pham et al., 1983), incriminates an exposure unique to the underground setting, with radon daughters being a plausible candidate. Haematite mining in locations where exposure to radon daughters is demonstrably low is apparently not associated with cancer risk (Axelson and Sjoberg, 1979).

There are no published epidemiological studies specifically on the cancer risk associated with non-occupational exposure to dust composed predominantly of haematite or other iron oxides, such as would be encountered in communities living near to an iron ore mine or a surface stockpile of ore. However, from the foregoing discussion, it may be concluded that there are no firm grounds for suspecting a risk of cancer associated with dust exposure in a non-occupational setting such as this, but such a risk cannot be entirely excluded.

The International Agency for Research on Cancer (IARC) has classified haematite and iron oxides as Group 3 carcinogens, i.e., currently available data are inadequate to permit a firm conclusion regarding a causal association between exposure and cancer in humans (IARC, 1998, 1999).

c) Haematite/ferric oxide as a co-carcinogen with organic compounds

There is considerable evidence from experimental animal models that haematite (Fe_2O_3) accentuates the carcinogenic properties of organic hydrocarbons that are known to be carcinogenic, including diethyl nitrosamine (Nettesheim et al., 1975) and benzo[a]pyrene (B[a]P). Although the weight of evidence suggests that ferric oxide alone does not induce respiratory tract tumours in animal models when instilled into the respiratory tract (Steinhoff et al., 1991), experiments using intratracheal instillation of B[a]P and/or haematite particles into Syrian golden hamsters have demonstrated that B[a]P coated onto haematite particles reduces the latency of, and increases the incidence of tumours. The mechanistic basis of this

effect remains incompletely understood. It was initially attributed entirely to the physical properties of the haematite as an inert carrier necessary for high tumour yield (Saffiotti et al., 1968, Saffiotti et al., 1972), as particles were understood to increase penetration of B[a]P and facilitate retention of B[a]P in the respiratory tract, increasing residence time of the carcinogen and thereby effectively increasing the B[a]P dose (Henry et al., 1975, Boutin et al., 1996). Subsequent experiments with Sprague-Dawley rats demonstrated that lipid peroxidation was induced by haematite, and could be accentuated by coating B[a]P onto haematite particles (Garcon et al., 2000, Boutin et al., 1996). Additionally, it was shown that induction of the inflammatory mediators interleukin-1β, nitric oxide and tumour necrosis factor-α by B[a]P was more pronounced when B[a]P was coated onto haematite particles (Garcon et al., 2001). It has recently been shown experimentally that haematite, although not genotoxic *per se*, accentuates the genotoxicity of B[a]P in rat lung cells and peripheral lymphocytes (Garry et al., 2003).

Some authors (Garry et al., 2003; Garcon et al., 2004) have attributed the epidemiological data linking lung cancer and iron ore mining to combined exposure to iron oxide dust and organic hydrocarbon carcinogens from diesel fumes in the mines. This however, is clearly not a valid interpretation of the aetiology of all excess lung cancers in this occupational setting, as many such cancers developed in the decades before miners were exposed to concentrated diesel fumes (Boyd et al., 1970; Pham et al., 1983). Also, this explanation ignores the robust evidence attributing these malignancies to α-radiation from inhalation of high concentrations of radon daughters in poorly ventilated underground settings.

d) Silica dusts

Not unexpectedly, all published epidemiological studies on the relationship between exposure to silica and cancer appear to have been conducted in occupational settings.

Crystalline silica in the occupational setting, inhaled in the form of quartz or its polymorph cristobalite, has been classified as a Group 1 carcinogen (definitely carcinogenic) by the IARC, based on data from both human epidemiological studies demonstrating an excess risk of lung cancer, and animal experiments (IARC, 1997).

This IARC classification has been a matter of some controversy, and the human carcinogenicity of inhaled crystalline silica has not been uniformly accepted (Soutar et al.,

2000; Hessel et al., 2000). Experimental verification of the carcinogenesis of silica in the respiratory tract is essentially limited to studies on rats, which may be considered an inappropriate animal model, as rats are notoriously prone to developing lung cancers when exposed to high doses of substances believed to be of low toxicity to humans e.g., titanium dioxide (Soutar et al., 2000). There has been some inconsistency in the detection of excess lung cancer risk in published epidemiological studies. Additionally, the magnitude of the measured association is low in comparison with other inhaled occupational carcinogens such as arsenic and cadmium (Steenland et al., 2001). Consequently there is a theoretical potential for spurious association between silica inhalation and lung cancer based on inadequate measurement of confounding factors such as smoking.

Nevertheless, the majority of published epidemiological studies demonstrate a significant association between lung cancer and occupational exposure to quartz or cristobalite dusts, at concentrations around the current workplace dust concentration regulatory threshold (0.1 mg/m^3) (Steenland et al., 2001). There is a biological gradient between cumulative exposure and risk. The link is seen in a range of occupational settings including ceramic production and brick and sand plants, in which confounding exposures such as radon and arsenic are almost certainly negligible, as well as in the mining contexts in which these concomitant exposures are possible. A recent large cohort study documenting excess lung cancer among workers in a sand plant exposed to silica dust (McDonald et al., 2001) has addressed methodological shortcomings of previous research (Wong, 2000). Cumulative exposure to silica dust has been ascertained carefully in job-matrix data (Rando et al., 2001), with scrupulous adjustment for smoking in a nested case-control design (Hughes et al., 2001).

It remains uncertain whether pulmonary silicosis is a pre-requisite or risk factor for the development of lung cancer, or whether the damage and disease processes involved can progress independently. The rat lung tumour response to silica appears to be the result of severe persistent inflammation (Nehls et al., 1997). Attempts to link lung cancer and pre-existing silicosis in epidemiological studies are fraught by the diagnostic limitations of chest radiography for detection of silicotic changes, and the biases of case ascertainment inherent in retrospective analysis of workers' records in which chest radiography has been used in the assessment of compensation claims (Wong, 2002; Checkoway and Franzblau, 2000).

The implications of the published data for low-level exposures to quartz dust in non-occupational settings are uncertain. A threshold exposure if any, below which excess cancer risk is negligible, is uncertain (Finkelstein, 2000). However, in view of the relatively low excess lung cancer risk documented even with sustained relatively high-level occupational exposures, and the possible dependency of silica-induced lung cancer on pre-existing inflammation in silicotic nodules, the risk is almost certainly very low at the most.

e) Manganese and copper

Based on evidence of low mutagenicity in *in vitro* studies, the carcinogenicity of manganese is believed to be low (Desoize, 2003). Published epidemiological evidence on cancer in relation to manganese mining appears to be restricted to two Japanese studies, in which an increased risk of prostatic cancer was found in proximity to manganese mines (Watanabe et al., 1981; Nakata et al., 1995). In Nakata's study, the magnitude of association was marginal (age-adjusted incidence rate 12.0/100,000/yr for districts with 'a history of manganese mining' versus 10.5/100,000/yr for those without), and no statistical inferential data were reported. Catalytic copper has a theoretical carcinogenic potential by virtue of its role in the formation of DNA-binding reactive oxygen species (Theophanides and Anastassopoulou, 2002). However there are no published epidemiological studies linking cancer and exposure to copper ores.

f) Conclusions

Although dust particles of haematite and related iron oxides may augment the carcinogenicity of certain organic carcinogens, there is no conclusive evidence that the dust containing iron oxide is carcinogenic in its own right.

The IARC's classification of crystalline silica dust in the occupational context is supported by the weight of published epidemiological data. It is very unlikely that dust containing low levels of crystalline silica in non-occupational settings constitutes a substantial cancer hazard. However, given a non-threshold model of carcinogenesis risk, an extremely small risk cannot be excluded.

5.8 "Nuisance" morbidity of Port Hedland dust exposure

Even in the absence of major adverse effects on human health, exposure to dusts may result in so-called nuisance effects – minor or self-limiting irritation of the eyes, upper respiratory tract

and/or skin. The Australian Department of the Environment and Heritage (Department of the Environment and Heritage, 1998) defines nuisance dust as:

> "dust which reduces environmental amenity without necessarily resulting in material environmental harm. Nuisance dust comprises particles with diameters nominally from about 1 mm [sic] up to 50 μm. This generally equates with 'total suspended particulates' (TSP). The TSP range of dust particles is broad, and may be produced from sources such as industrial and mining processes, agricultural practices and from wind erosion of the natural environment."

Notably, "(I)mpacts of mine dust on near neighbours is [sic] most often due to nuisance dust".

Iron oxides are considered for regulatory purposes by the US Occupational Health and Safety Administration to have nuisance effects such as irritation of the eyes, nose and upper respiratory tract (Occupational Health and Safety Administration, 1989).

There are essentially no published peer-reviewed journal data specifically concerning the nuisance effects of ferric oxide dusts. Contact dermatitis from exposure to red iron oxide (haematite) is a documented occupational hazard among enamellers and decorators in the ceramics industry (Motolese et al., 1993). There are also case reports of allergic contact dermatitis caused by yellow iron oxide (limonite, of which goethite [$Fe^{3+}O(OH)\cdot nH_2O$] is the major constituent) (Zugerman, 1985) and black iron oxide (magnetite [$Fe^{3+}{}_2Fe^{2+}O_4$]) (Saxena et al., 2001) applied cosmetically to the eyelids as mascara.

SECTION 6. PARTICLE SIZE AND HEALTH

Summary

The major components of PM vary according to the geographical setting; they include sulphate, nitrate, chloride, elemental and organic carbon, crustal material and biological material, with trace elements accounting for < 1%. In urban PM, SO_2, NOx and carbon compounds derived from fossil fuel combustion predominate. In urban settings, there is evidence that both short- & long-term exposure to fine PM are associated with increased mortality. There is inconsistent evidence for an independent effect of coarse PM on short-term mortality – many studies on the effects of crustal/windblown PM are negative in this regard. There is little or no evidence for increased mortality associated with long-term exposure to coarse PM. Exposure to both fine and coarse PM is associated with respiratory symptoms and hospital admissions for respiratory diseases (asthma and COPD). Exposure to fine PM has been more consistently associated with diminished lung function. Increased levels of PM are associated with higher levels of COPD diagnosis. Exposure to fine PM is associated with an increased risk of cardiovascular disease and cardiovascular mortality. Toxicological studies demonstrate that PM, particularly the UF fraction, elicits an inflammatory response in the lung. Organic compounds and oxygen free radical production play important roles in the toxic affects of $PM_{2.5}$. Some of the cellular toxicity of coarse PM can be attributed to bacterially-derived endotoxin. The vast majority of this data is derived from studies in urban settings.

6.1 Constituents and physicochemical properties of particulate matter

The physicochemical properties of airborne particles have been recently reviewed in detail, in public access governmental reports available on the internet (Air Quality Expert Group, 2005b), as well as in peer-reviewed journal literature (Wilson et al., 2002). Atmospheric particles are highly diverse in origin and correspondingly heterogeneous in composition and physical properties. Particles may be solid, liquid or both, for example composed of a solid core enveloped by liquid. Primary particles are dispersed directly from a source (such as a mechanical or combustion process) whereas secondary particles are formed *de novo* in the atmosphere by chemical reactions

(principally oxidation) that result in the production of substances having relatively low volatility and that therefore condense from the gaseous phase into solids or liquids.

The formation of particulate matter by condensation of gases is termed nucleation. The most common type is heterogeneous nucleation, i.e., enlargement of existing (primary) particles by condensation on their surface. Some molecules (e.g. H_2SO_4 formed from atmospheric oxidation of SO_2) are of sufficiently low volatility to nucleate with water and ammonia to form sulphate droplets (homogeneous nucleation) (Air Quality Expert Group, 2005b).

Ambient PM can be conceptualized as having a trimodal distribution with respect to size and behaviour in the atmosphere. The smallest, newly nucleated particles are typically transient as they tend to enlarge, either by condensation or by aggregation, to form larger particles. The brief survival of the smallest particles (≤ 50 nm) can also be attributed to their efficient deposition onto surfaces, given their particular tendency to Brownian motion and consequent diffusion. The largest particles (> 2.5 μm) settle rapidly by gravitational sedimentation. By contrast, particles in the size range 50 nm to 1 μm can persist in the atmosphere for long periods (typically several weeks) although they are subject to incorporation into rain and removal by precipitation in raindrops (Air Quality Expert Group, 2005b).

Airborne particulate matter (PM) is composed of solid and liquid particles generated by a wide variety of natural (wind-borne soil, sea-spray, forest fires, volcanic emissions) and anthropogenic (combustion of fossil fuels, vehicle and industrial emissions, road and rail dust) sources. PM has been broadly classified, on the basis of size, as coarse particles (diameter > 2.5 μm) that are mainly derived from windblown soil and sea spray, and fine (0.1 to 2.5 μm diameter) and ultrafine particles (< 0.1 μm) that are predominantly derived from combustion of fossil fuel (Nel, 2005). The health risks posed by PM are determined by its size, surface area and chemical composition (Donaldson and Tran, 2002). Investigations of the health effects of airborne particulate matter have mainly focused on PM_{10} (all particles < 10 μm in aerodynamic diameter), of which a large fraction would be $PM_{2.5}$ (particles < 2.5μm in diameter), or specifically on $PM_{2.5}$. It is only recently that more attention has been given to dissecting out the specific adverse health effects of the coarse fraction of PM_{10} (usually defined as

particles > 2.5 and < 10 μm in diameter) or fine and ultrafine particles (Brunekreef and Forsberg, 2005).

a) Constituents of PM_{10} and $PM_{2.5}$

In order to estimate the effects of particles on health, the concentration and composition of particles collected at specific locations need to be measured. Wilson et al., (2002) have listed numerous PM parameters and components that are likely to be relevant to the effects of particles on health, including particle number, surface area, density and size distribution. PM mass, including fine-mode and coarse-mode mass as well as $PM_{2.5}$ and PM_{10}, non-volatile mass and mass including semi-volatile components, the content of ions, elemental and organic carbon, transition metals, toxic organic compounds, crustal elements and bio-aerosols may all be relevant.

(i) Bulk chemical and trace element composition

Airborne particulate matter is made up of several major components, the relative abundance of which is referred to as the 'bulk chemical composition' (Harrison and Yin, 2000). The major components typically identified in air samples are 1) sulphate – derived from the oxidation of SO_2 in the atmosphere and present mainly as the ammonium salt, 2) nitrate – derived from oxidation of atmospheric NO_2 and also present as the ammonium salt, 3) chloride – mainly derived from sea spray, even at locations distant from the coast, 4) elemental and organic carbon – derived mainly from combustion of fossil fuels, and in particular vehicle exhausts, which emit soot particles containing a core of solid elemental carbon coated with semi-volatile organic compounds, 5) crustal materials, including soil dusts and windblown minerals, which are present mainly in the coarse particle fraction, and 6) biological materials, including bacteria, spores, pollens and fragments of plant and animal origin, although in some studies these are classified as organic carbon. Trace elements usually represent less than 1% of total particle mass, but their bioavailability and propensity to produce reactive species through participation in specific reactions such as the Fenton reaction, suggests that trace elements are likely to play an important role in the deleterious effects of particulate matter on human health (Harrison and Yin, 2000).

The bulk chemical composition of particulate matter varies widely between different urban areas and between urban and rural areas (Harrison and Yin, 2000). Climatic

factors have a major influence on the abundance of crustal materials, and in dry climates these may constitute an important major component of particulate matter. Intensive agriculture is a major source of ammonia and thus the concentration of ammonium ions in particulate matter is likely to show considerable geographic variation. Significant differences in bulk chemical composition may also occur within a large country such as the USA or Australia. PM_{10} samples from the western US show higher concentrations of nitrate (24%) and elemental and organic carbon (30%), but lower concentrations of sulphate (4.6%) compared to those from the eastern US (nitrate 1.2%, organic carbon 8.5%, sulphate 27.8%) (US EPA, 1996). These differences probably relate to the greater use of SO_2 emitting fossil fuels for electricity generation in the east, and the relatively greater contribution of NO_2 emissions from vehicles and the greater abundance of ammonia in the western US (Harrison and Yin, 2000). The mineral content also differed between these locations, constituting 36% of PM_{10} in the western US, but only 19.6% in the east.

Organic and elemental carbon accounted for 20% and 18%, respectively, of the bulk chemical composition of PM_{10} samples collected in Birmingham, UK in 1995 (Harrison et al., 1997). Sulphate comprised 17%, nitrate 6% and ammonium 6% of these samples. Generally similar bulk chemical compositions of PM_{10} have been reported for samples collected in Sapporo, Japan (Kaneyasu et al., 1995), Hong Kong (Qin et al., 1997) and urban southern California (Chow et al., 1994). However, in some studies crustal material comprised a significant percentage of the bulk chemical composition of the PM_{10} or coarse particle fraction. Thus, in a study from the south-western US, crustal material comprised 88% of the coarse fraction (2.5 – 15 μm) and 24% of $PM_{2.5}$ (Vasconcelas et al., 1994), while in regionally representative locations in California, crustal material comprised 46% of PM_{10} (Chow et al., 1996). In a study of samples collected in Brisbane, Australia, crustal material represented 29%, organic compounds 13%, nitrate 4%, ammonium 3%, and sulphate 1.4% of the bulk chemical composition, while chloride (22%), probably derived from sea-spray, was also an important component of PM_{10} (Chan et al., 1997).

A more recent study used a high volume cascade impactor to fractionate PM_{10} samples collected in Brisbane, Australia into 6 size fractions (<0.5, 0.5-0.61, 0.61-1.3, 1.3-2.7, 2.7-4.9 and 4.9-10 μm) (Chan et al., 2000). More than 40% of the mass of the aerosols

was in the >2.7 μm fraction with a further 40% in the < 0.5 μm fraction. Elements derived from natural sources (Na, Cl, Si, Al, K, Ca, Fe, Mn, Zn, Br) were mainly present in the >2.7 μm fraction, whereas components related to anthropogenic sources (soot, organics, lead bromide) were predominantly in the < 0.5 μm fraction. The $PM_{2.7-10}$ fraction was composed of crustal matter (24%), sea salt (16%), organic compounds (9%), soot (3%), ammonium sulphate (1.5%) and other components, including nitrates (20%). In contrast the $PM_{2.7}$ fraction contained less crustal matter (8%) and sea salt (3%), but more organics (25%) and soot (9%). Analyses of $PM_{2.5}$ samples from Sydney (ERDC, 1995) and Melbourne (Gras et al., 1991) showed similar levels of crustal matter and sea salt, although ammonium sulphate and soot contents were higher in the Sydney samples and organic compounds were higher in the Melbourne samples. Both Sydney and Melbourne $PM_{2.5}$ samples contained higher levels of lead bromide than the more recent Brisbane samples and this probably relates to the phasing out of the use of leaded petrol. The aerosol content of ammonium sulphate and organic compounds is likely to be related to the level of industrial activity near the sampling site, while nitrate content may reflect local traffic volumes and soot, the numbers of diesel-fuelled vehicles (Chan et al., 2000).

In the Brisbane study, the source factors contributing to PM composition were identified as soil, sea salt, organics and vehicle exhausts, although the analysis indicated that mixing of two or more sources was likely to have occurred (Chan et al., 2000). Thus, the 'soil' source factor showed an abundance of crustal elements including Si, Ca and Fe, although contributions of soot and organics were also apparent. The major fingerprints for sea salt were Na and Cl, but included contributions of elemental and organic carbon. The 'organic' source showed not only the major contribution of organic carbon but also those from Si, Na, Al and Fe, while the 'vehicle exhaust' source was made up mainly of contributions from elemental carbon, Si and Fe. The 'soil' and 'sea salt' sources contributed more than 80% of the >2.7 μm aerosol mass, while the 'organics' and 'vehicle exhaust' sources accounted for almost all of the < 0.61 μm mass (Chan et al., 2000).

(ii) Chemical composition of PM at different locations

In recent years numerous studies have attempted to characterize the composition of PM collected at a variety of urban and rural sites or adjacent to areas of specific human

activity or under different climatic or meteorological conditions. Large-scale monitoring of fine and coarse PM_{10} has been performed in four major Italian cities (Florence, Genoa, Milan, Naples) and the concentrations of >20 elements were determined (D'Alessandro et al., 2003). Vehicle emissions, particles generated by the wearing of road surfaces, soil dust, industrial components, oil combustion and sea salt were identified as the sources of PM in all cities. Traffic was a major source of Cu, Pb, Br and the light attenuating fine fraction. A subsequent study monitored the concentrations of >20 different elements in PM_{10} aerosols at three different urban settings in Florence, on a daily basis over a 1-year period (Lucarelli et al., 2004). Traffic was again identified as the major source of PM both at a high traffic site and at an urban background site. Long-term sampling and elemental analysis of the PM_{10}, $PM_{2.5}$ and PM_1 fractions has also been performed on samples collected in an urban area of Genoa (Ariola et al., 2006). The levels of PM_{10} collected at rural, urban and industrial sites in six regions of Spain have recently been summarized (Querol et al., 2004).

The sources and composition of ambient $PM_{2.5}$ in Amsterdam (The Netherlands), Erfurt (Germany) and Helsinki (Finland) were compared using principal component analysis and multiple linear regression. Six sources of $PM_{2.5}$ were identified in Amsterdam, including traffic (30%), secondary particles (mainly ammonium sulphate, 34%), crustal material (7%), oil combustion (11%), industrial and incineration processes (9%) and sea salt (2%). The results from Erfurt were similar except that there was more crustal material (21%), whereas secondary particles constituted as much as half the total average $PM_{2.5}$ in Helsinki (Vallius et al., 2005). These differences were thought to reflect the lower population density and smaller scale of energy production and industrial activities in Finland.

Systematic sampling and detailed analysis has been performed to determine the chemical composition of PM_{10} from different geological sources in the San Joaquin Valley, California (Chow et al., 2003). The sources included urban and rural paved and unpaved roads, agricultural and dairy fields, canal drainage deposits and building and roadway construction/earthmoving sites. Elemental carbon and Pb were markers for paved road dust, while Na, S, and sulphate marked salt deposits from canal drainage. Animal husbandry sources were characterised by organic carbon, phosphate, K and Ca, while the abundances of metals such as Ti, V and Mn was higher in construction soils.

(iii) Seasonal influences on the chemical composition of PM

Seasonal influences on the characteristics and composition of PM_{10} have been studied in Rome, Italy, which has cool winters during which a dry wind blows from the north-east, while moist south-west winds carry a fine suspension of dust from the Sahara desert during the warm summers. There is little pollution derived from industrial or energy generation sources and the main source of atmospheric pollution in the Rome urban area is vehicle traffic, and in winter, the combustion of fuel oil or methane for heating (Paoletti et al., 2002). Mean airborne PM_{10} concentration decreased from 56 $\mu g/m^3$ in winter to 41 $\mu g/m^3$ in summer. Using hierarchical cluster analysis, seven groups (clusters) of similar particles were identified. The 'carbon-rich particle' cluster comprised 65-85% of the $PM_{3.3}$ (fine) fraction and 12-25% of the $PM_{10-3.3}$ (coarse) fraction. Two different particle morphologies were observed in this carbonaceous cluster, a) aggregates of tens to hundreds of spherules, each ~40 nm in diameter, and b) rarely observed larger 'flake-like' particles. A surface coating of S and N or sometimes Si, K, Ca and Fe was variably observed on both the spherules and flakes in the fine particle fraction. The most significant source of this carbonaceous PM was motor vehicle exhausts. 'Carbonate', 'silica' and 'silicate' clusters originated from soil erosion and/or erosion of urban structures. On the whole, these clusters comprised 9% of the $PM_{3.3}$ fraction in winter and 24% in summer, while they represented more than 50% of the $PM_{10-3.3}$ fraction in both winter and summer. A coating of S was also detected on a variable proportion of the fine 'Si-rich' particles. The 'sulphate' cluster, composed mainly of Ca sulphates, had an abundance in the coarse fraction of ~13% and in the fine fraction of < 7%. They were thought to arise from reactions between Ca carbonate materials such as marble, and sulphur-rich compounds in the atmosphere, as well as from wearing of paints, plaster and paper. The 'Fe-rich' particle cluster was composed of Fe (>50%) and oxygen, while the 'metals' cluster was composed of oxides of Al, Ni, Cr, Ti, Cu and Zn, with these two particle clusters comprising < 6% of $PM_{3.3}$ and < 10% of the $PM_{10-3.3}$ fractions (Paoletti et al., 2002).

Three significant seasonal trends were identified in this study from Rome (Paoletti et al., 2002). Firstly, the greater frequency of soil particles in the $PM_{3.3}$ fraction in summer was though to be almost exclusively due to the fine alumino-silicate particles transported by the winds from North Africa. Second, an increase in carbonaceous particles in the fine fraction in winter was probably due to an increase in vehicular

traffic and possibly the combustion of fossil fuels for heating. The third and most obvious seasonal effect was the big increase in the abundance of sulphur-coated particles in the $PM_{3.3}$ fractions collected during summer. This observation was in agreement with previous data from the urban environments of Phoenix in the USA (Katrinak et al., 1995) and Beijing, China (He et al., 2001). It was postulated that the photo-chemical oxidation of SO_2, which is dependent on the amount of solar radiation, might be an important factor determining the abundance of sulphur-coated particles (Paoletti et al., 2002).

Long-range transport of Saharan dust from Africa was also found to contribute to episodes of high PM_{10} in Zaragoza, Spain, in a study that determined the concentrations of 16 elements in samples collected in a heavy traffic area (Lopez et al., 2005). However, enrichment for Pb, Zn and Cu compared to crustal element compositions reflected the contribution of anthropogenic sources of PM_{10}, mainly emissions from vehicles and industrial processes. Calm weather with low wind speeds was found to favour the accumulation of PM_{10} and increase trace element pollution.

The chemical composition of $PM_{2.5}$ and PM_{10} were investigated in Mexico City during the winter of 1997, with samples being collected from regional, central, commercial, residential and industrial areas of the city (Chow et al., 2002). PM_{10} concentrations were highly variable, with fugitive dust and carbon concentrations being the primary causes of this variability. The PM mass was mainly comprised of carbon, sulphate, nitrate, ammonium and crustal materials, but the composition varied on different days and with location. Carbonaceous particles constituted ~50% of the $PM_{2.5}$ mass followed by inorganic aerosols (30%) and crustal material (15%). The PM_{10} mass was made up of crustal material (50%) and carbonaceous (32%) and inorganic (17%) aerosols. Diurnal variations were observed, with morning samples having the greatest $PM_{2.5}$ and PM_{10} mass, inorganic aerosols and carbon concentrations, probably due to the effects of surface inversion and rush-hour traffic.

A study of PM_{10} samples collected in five Chinese cities (Dongying, Jinan, Qingdao, Shanghai, Beijing) showed the presence of quartz, feldspar, clays, calcium sulphates, and carbonates at all locations, with carbonaceous particles predominating in winter-time samples (Davis and Guo, 2000). Calcium carbonate, an important constituent of

fugitive dust, was observed to react with sulphuric acid aerosols from industrial sources to form hydrated calcium sulphate. A more detailed analysis was performed of the characteristics of $PM_{2.5}$ samples collected at residential and downtown sites in Beijing and Shanghai (Yang et al., 2005b, He et al., 2001). There was significant seasonal variation in $PM_{2.5}$ concentrations, with the highest concentrations occurring in winter and the lowest in summer. The concentrations of crustal species such as Al, Fe, Si, Ca and Mg increased markedly during the spring, probably as a consequence of the frequent dust storms that occur in Beijing at that time of the year. Organic carbon was the most abundant species, accounting for > 30% of the $PM_{2.5}$ mass at both sites, with another 25-30% being made up of ammonium, sulphate and nitrate.

The chemical characteristics and sources of $PM_{2.5}$ particles have been investigated in the Sihwa area of South Korea (Park et al., 2001). Ammonium sulphate was found to be the major component of $PM_{2.5}$ particles, followed by organic carbon emitted from an industrial complex, ammonium nitrate, products from the combustion of agricultural waste, vehicle emissions, road dust and marine aerosols. Comparisons have also been made of the ionic composition of fine and coarse particle fractions collected at two urban locations in South Korea (Kim et al., 2006). The site in Seoul represented a moderately developed complex urban area that was likely to be influenced by various anthropogenic sources, whereas the site in Busan (the second largest city) was a lightly developed grassland area close to the seashore. The mean PM_{10} concentrations were higher for Seoul (79 $\mu g/m^3$) than Busan (34 $\mu g/m^3$), but the difference was even more marked for the $PM_{2.5}$ concentrations (Seoul 58 and Busan 19 $\mu g/m^3$), and this enrichment of the fine fraction in Seoul suggested a strong influence of anthropogenic activities. Differences in ionic compositions between the coarse and fine fractions and between the two locations indicated the dominance of oceanic processes at the Busan site as well as the influence of air mass movement patterns.

The chemical composition of PM_{10}, divided into coarse (2.1-10 μm) and fine (< 2.1 μm) fractions, has also been investigated in the ambient air of Delhi, India (Balachandran et al., 2000). Pb, Cr, Cd and Ni concentrations were up to six times greater in the fine than the coarse fraction, whereas the Fe concentration was greater in the coarse fraction suggesting that the source of Fe was mainly resuspension of crustal material. Principal component analysis identified vehicle and industrial emissions and soil resuspension as

the major sources of PM_{10} in Delhi. A two step procedure that combines air dispersion and receptor models was used to estimate the contributions of a thermal power plant, an integrated steel mill, motor vehicle emissions and dust from paved and unpaved roads to PM_{10} levels in San Nicolas, Argentina. The most likely contributing sources were differentiated from those that were insignificant by diagonalization of the covariance matrix (Gomez et al., 2005).

Some studies have investigated episodes of high PM pollution related to unusual meteorological conditions or proximity to specific industries or other human activities. During PM measurements in Milan and Erba in the Po valley of Italy, daily variability in mass concentration values and $PM_{2.5}/PM_{10}$ ratios was strongly dependent on the stability of meteorological and atmospheric conditions, and in particular wind. These were also major factors influencing a high-PM pollution episode that was recorded at many locations in the Po plain (Marcazzan et al., 2002). The characteristics of PM collected during dust storm episodes in Beijing have been compared to those of samples collected in haze pollution episodes. During dust storms PM_{10} levels reached 1,500 $\mu g/m^3$, while the $PM_{2.5}$ concentration was ~230 $\mu g/m^3$ (Xie et al., 2005). Due to strong winds during the dust storms SO_2, NO_2 and ozone levels were low, whereas crustal elements constituted about 66% of the $PM_{2.5}$ composition. The coarse fraction ($PM_{10-2.5}$) collected in central Taiwan during an Asian dust event in 2000 was enriched in the crustal elements, Ca, Mg, Al and Fe as well as Na^+ and Cl^- from sea salt (Cheng et al., 2005).

The relationships among meteorological data, pollution sources and receptors have been investigated in a petrochemical industrial area of northern Taiwan (Chiang et al., 2005). Particle and elemental concentrations were lower in summer due to greater convection of the air mass and vertical dispersion of pollutants. In addition high pressure systems and typhoons accompanied by high wind speeds and unstable weather conditions dispersed PM in summer. In winter the prevailing north-easterly winds may have carried PM and pollutants from mainland China and together with stable weather conditions may have led to an accumulation of PM.

The levels and composition of PM_{10} were investigated at a mining-industrial site in the city of Lavrion, Greece (Protonotarios et al., 2002). The soil at this site is heavily

contaminated with heavy and toxic metals due to intensive mining and metallurgical activities over the past 3,000 years. Wind erosion has resulted in surface deposits that are easily resuspended as PM in the surrounding atmosphere and there are also potential sources of industrial pollution close to the site. PM_{10} and elemental concentrations increased significantly during summer, with Fe, Pb, Zn, Mn and Cu considered to originate from contaminated soil, while enrichment for As, Ni, Cd and Cr was attributed to adjacent industrial plants. Another study investigated the chemical composition of PM arising from the burning of post-harvest biomass in Gwangju, South Korea. The fine fraction was unusually enriched in chlorine and potassium derived from the burning of semi-dry barley and rice straw, and organic and elemental carbon concentrations were also increased during burning of biomass (Ryu et al., 2004).

The size and composition of PM arising specifically from pavement wear, tyres and traction sanding has been investigated in a test facility (Kupiainen et al., 2005). Mass size distributions were dominated by coarse particles with concentrations increasing when traction sand was used. PM compositions were also affected by the type of tyres, properties of the traction sand aggregate and driving speed. Over 90% of PM_{10} was mineral particles originating from traction sand and paving aggregates, with the remainder being carbonaceous material from tyres and road bitumen. PM derived specifically from roads and motor vehicle emissions have been measured in two tunnels in Milwaukee, USA. The PM_{10} was composed mainly of organic carbon (30%), inorganic ions (20%), metals (19%) and elemental carbon (9%) with the metals being predominantly crustal elements (Si, Fe, Ca, Na, Mg, Al, K) and elements associated with exhaust emissions and brake and tyre wear (Cu, Zn, Sb, Ba, Pb, S). Resuspension of roadway dust depended on weather and road surface conditions, with increased emissions being associated with higher traffic volumes and the numbers of heavy vehicles (Lough et al., 2005).

b) Composition of ultrafine particles

Recently more attention has been focused on ambient ultrafine particles ($PM_{0.1}$, UFP) as there is evidence to suggest they may be more toxic than larger particles due to the orders of magnitude greater particle numbers and surface area, and greater concentrations of adsorbed or condensed toxic pollutants per unit mass (Sioutas et al., 2005). UFP are formed by at least three processes, a) direct emission into the

atmosphere during combustion processes associated with vehicular or industrial sources, b) nucleation and condensation during cooling of hot supersaturated vapours emitted during combustion and c) atmospheric chemical reactions that generate chemical species with low vapour pressures at ambient temperatures. Motor vehicles are the primary sources of direct emissions of UFP in urban areas (Zhu et al., 2002, Hitchins et al., 2000), with the most numerous emission particles ranging in size from 20-60 nm for petrol engines (Ristovski et al., 1998) and 20-130 nm for diesel engines (Morawska et al., 1998). In addition, atmospheric photochemical reactions generate low-volatility chemical species that form UFPs by a variety of nucleation processes involving sulphuric acid, ammonia and water vapours as well as trace gases and organic compounds (Kulmala et al., 2004).

Although concern about the health impacts of UFP is increasing, there is limited information on the concentrations and physico-chemical properties of these particles in places where people live and work (Sioutas et al., 2005). The chemical composition of ultrafine particles collected in the Los Angeles basin over three different seasons ranged from 32 to 69% for organic carbon, 1-34% for elemental carbon, 0-24% for sulphate and 0-4% for nitrate, with the highest ultrafine mass concentrations occurring in the autumn (Sardar et al., 2005). Other recent studies have attempted to characterise the formation and composition of UFP in Mexico City (Baumgardner et al., 2004), London (Charron and Harrison, 2003), Rochester, New York (Jeong et al., 2004), Atlanta (Woo et al., 2001) and Detroit (Young and Keeler, 2004). The trace elements Pb, As, La, Ce, Sr, Zn, Cr, Se, Sn, Y, Zr, Au, and Ag, were detected in the UFP range of samples collected in the Detroit urban area (Utsunomiya et al., 2004). UFP concentrations were greater in the large city of Copenhagen compared to the medium-size city of Gothenburg and lowest at more rural sites (Matson, 2005).

Seasonal and spatial trends in particle number concentrations (PNC), a proxy for UFP, have been analysed at eight urban, suburban and remote sites in southern California. Average PNC were higher in winter at all urban sites and, during the strike of port workers at Long Beach in 2002, there was a significant increase in PNC due to emissions from idling ships in the port (Singh et al., 2006). In the San Joaquin Valley, California, which has a severe air quality problem in winter, $PM_{0.1}$ concentrations were distinctly diurnal, peaking at ~2.4 $\mu g/m^3$ at night. The ultrafine particle mass was

mostly associated with carbonaceous material and it was suggested that the high ultrafine particle concentrations posed a potentially serious threat to public health in the San Joaquin Valley (Herner et al., 2005).

c) Summary – composition of particulate matter

The major components of airborne particulate matter are sulphate, nitrate, chloride, elemental and organic carbon, crustal material and biological material. Trace elements usually constitute less than 1% of total particle mass. In dry climates crustal material may be an important component of PM, while in urban areas SO_2, NO_2 and organic and elemental carbon, derived from fossil fuel combustion and motor vehicle exhausts, are major components of PM. The composition of PM is influenced by geographical, meteorological and anthropogenic factors, including the degree of urbanization, industrial and agricultural activities, and the type and volume of vehicular traffic. Seasonal influences include the combustion of fossil fuels for heating, long-range transport of crustal material from desert areas in seasonal dust storms, increased photochemical oxidation during summer and seasonal burning of biomass. Motor vehicle emissions are the primary sources of ultrafine particles in urban areas, with limited data indicating that organic and elemental carbon is the major component of this size fraction.

6.2 Effects of urban particulate matter on human health – epidemiological studies

The adverse effects of urban air pollution on human health are increasingly well delineated, especially in the case of particulate matter (Katsouyanni, 2003). Adverse effects can be assessed by measurement of various indices, including mortality, a range of symptoms and clinical morbid outcomes, and physiological alterations (e.g., diminished lung function) (American Thoracic Society, 2000).

a) Effects of urban PM on short-term mortality

(i) US and Canadian studies

In the largest time series study conducted to date, Schwartz et al. (1996) and Klemm et al. (2000) investigated the association between daily mortality and various PM fractions measured in six US cities (Boston, Knoxville, St Louis, Steubenville, Portage and Topeka) over 8 years (1979-1987). Overall, there was a small but significant increase in total mortality (~1.5%) associated with an increase of 10 $\mu g/m^3$ in the fine fraction

($PM_{2.5}$), but there was no significant association with the coarse fraction ($PM_{10-2.5}$) (Brunekreef and Forsberg, 2005). An analysis of cause-specific deaths in people > 65 years of age showed significant positive associations of $PM_{2.5}$ with ischaemic heart disease and pneumonia (Klemm et al., 2000).

The association between daily mortality and air pollution in Santa Clara County, California was investigated in a time series study conducted between 1989 and 1996 (Fairley, 1999). Overall there was a significant effect for fine particles (2.9% increase in total mortality per 10 $\mu g/m^3$) but not coarse particles (Brunekreef and Forsberg, 2005). The levels of CO and sulphate were significantly associated with respiratory mortality, while $PM_{2.5}$ and nitrate on the current day were associated with cardiovascular mortality. Daily mortality was also significantly associated with PM_{10} pollution in the Utah Valley from 1985 to 1989. The association was strongest for mortality due to respiratory disease, followed by cardiovascular disease (Pope and Dockery, 1992).

The association between PM and daily mortality was studied in the Coachella Valley, California using data collected between 1989 and 1992 (Ostro et al., 1999b). In the area studied, which included a desert resort and retirement area east of Los Angeles, coarse particles of geological origin constituted 50 to 60% of the PM_{10} fraction and could exceed 90%, depending on the wind. A 10 $\mu g/m^3$ increase in daily PM_{10} was associated with an increase in total mortality of ~1%, providing evidence for an effect of PM_{10} on mortality in an area where airborne PM was dominated by coarse particles. The strength of the association was similar to that observed in urban areas in which increases in PM are due mainly to combustion-related fine particles. In a repeat study in the Coachella Valley, using data from 1989 to 1998, Ostro et al. (2000) found that several pollutants, including $PM_{2.5}$, CO and NO_2 were associated with all-cause mortality. A consistent association was also observed between cardiovascular-specific mortality and coarse particles, although none of the pollutants was specifically associated with respiratory mortality.

The association between PM and mortality was investigated in elderly residents of Phoenix, Arizona between 1995 and 1997 (Mar et al., 2000). The 3-year daily mean $PM_{10-2.5}$ concentration (33.5 $\mu g/m^3$) was higher than the $PM_{2.5}$ concentration (13 $\mu g/m^3$), possibly due to the arid conditions. Total mortality was significantly associated with CO

and NO_2, while cardiovascular mortality was significantly associated with CO, NO_2, SO_2, $PM_{2.5}$, PM_{10}, $PM_{10-2.5}$, and elemental carbon. Factor analysis showed that combustion-related pollutants and sulphate aerosols were both associated with cardiovascular mortality.

Daily data for fine ($PM_{2.5}$) and coarse ($PM_{10-2.5}$) particles, collected in Phoenix, Arizona between 1995-7, were used to investigate the effects on mortality of the population > 65 years of age, living in the city and in a surrounding area of about 80 km (Smith et al., 2000). In Phoenix, the coarse fraction is thought to be mainly composed of crustal material and construction/road dust, while the fine fraction originates primarily from vehicles. Therefore city mortality data were used when considering the effects of the fine fraction and regional mortality data when considering the effects of the coarse fraction. Statistically significant effects were observed between $PM_{10-2.5}$ and total regional mortality and between $PM_{2.5}$ and total mortality in the city, with the coarse fraction effects being significant only in spring and summer. There was no evidence of a threshold-based effect for the coarse fraction, while a threshold of 20 – 25 $\mu g/m^3$ was identified for the effect of the fine fraction.

Contrary evidence regarding the contribution of coarse particles to mortality was provided by a study from Spokane, Washington, which is located in an arid area that is subject to occasional dust storms after the harvesting of crops. Between 1989 and 1995, 17 dust storm episodes were identified in Spokane and the 24-h mean PM_{10} concentration during those episodes was 263 $\mu g/m^3$. There was little evidence of an increased mortality risk on the episode days compared with control non-episode days when the mean PM_{10} concentration was 42 $\mu g/m^3$, suggesting that coarse particles from windblown dust in this location were not associated with an increased mortality risk (Schwartz et al., 1999).

Mortality data from the Six Cities Study (1979-88) (Schwartz et al., 1996) were used to examine the effects of fine PM from different sources on daily mortality. The elemental composition of size fractionated particles was used to identify sources of fine PM, including soil and crustal material (silicon), motor vehicle exhaust (lead) and coal combustion (selenium). A 10 $\mu g/m^3$ increase in $PM_{2.5}$ from motor vehicles accounted for a 3.4% increase in daily mortality, and a similar increase in $PM_{2.5}$ from coal

combustion sources accounted for a 1.1% increase. However, $PM_{2.5}$ crustal particles were not associated with daily mortality (Laden et al., 2000).

Evidence for the lack of an association between crustal PM and daily mortality was also provided by a study of air pollutants from three sites in New Jersey between 1981 and 1983 (Tsai et al., 2000). A factor analysis of trace elements was used to identify major source types, including motor vehicle emissions (Pb, CO), oil combustion (V, Ni), industrial (Zn, Cu, Pb, Cd), geological (Mn, Fe) and secondary aerosols (SO_4). Oil combustion, industrial sources, motor vehicle emissions and secondary aerosols were all significantly associated with mortality, whereas geological sources were not.

The associations of PM components with daily mortality and morbidity in urban populations of Detroit, Michigan have been investigated. The data were collected during two periods (1985-90 and 1992-4) and showed positive but non-significant increases in the relative risks for daily mortality associated with PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$. However PM_{10} was significantly associated with respiratory mortality for the 1985-90 period. Ozone and NO_2 were also significantly associated with total and cardiovascular mortality (Lippmann et al., 2000). A time-series study of associations between size-classified PM and daily mortality in the Philadelphia metropolitan area showed positive and significant results for $PM_{2.5}$. However no systematic differences were observed according to particle size or chemistry, and an effect of the coarse fraction could not be excluded (Lipfert et al., 2000a). In a study of the association between daily mortality in two age groups (< 75 and ≥ 75 years) and ambient air pollution in Pittsburgh, Pennsylvania during 1989-91, no significant associations were found for any of the PM components. It was noted that instability of the pollutant coefficients due to data limitations made it impossible to ascertain confidently the relative importance of the fine and coarse fractions in relation to daily mortality (Chock et al., 2000).

Using PM air pollution, weather and mortality data collected between 1985-95, comparisons have been made between three metropolitan areas of Utah (Ogden, Salt Lake City and Provo/Orem) (Pope et al., 1999a). There were more high PM episodes dominated by windblown dust in Salt Lake City, but when the data were screened to exclude these episodes, comparable small increases in mortality of 0.8 to 1.6% per 10

μg/m^3 increase in PM_{10} were identified. The authors concluded that air pollution episodes caused by combustion-derived particles were more likely to be associated with increases in mortality than episodes of windblown dust with relatively higher concentrations of coarse crustal particles.

The association of mortality with air pollution over the period 1987-95 has been investigated in three major metropolitan US counties centred on Los Angeles, Chicago (Cook County) and Phoenix (Maricopa County). In Los Angeles County, the median 24-h average concentrations of PM_{10} and $PM_{2.5}$ were 44 and 22 μg/m^3, respectively. There was no evidence of an association between the coarse fraction and total daily mortality, cardiovascular, cerebrovascular or COPD mortality (Moolgavkar, 2000a). Although there was considerable heterogeneity for air pollution effects in the different geographic locations and between seasons (Moolgavkar, 2003), the gaseous pollutants, particularly CO, were more strongly associated with mortality than was particulate matter.

Using air quality and weather data for Atlanta, Georgia that were collected for the 2 years from August 1998, associations with daily mortality have been analysed according to age group (all ages, < 65 years, > 65 years) and cause of death (all causes, cardiovascular, respiratory, cancer, and other) (Klemm et al., 2004). The regressions for single air quality indicators on all-cause mortality in those > 65 years of age showed that CO, NO_2, $PM_{2.5}$, coarse PM mass, SO_2, and O_3, followed by elemental and organic carbon, consistently had the best model fits. Cardiovascular deaths in the elderly were consistently associated with CO.

PM-mortality dose-response curves and threshold levels have been estimated using time-series data for the 20 largest US cities, collected between 1987 and 1994, with PM_{10} as the exposure measure (Daniels et al., 2000). The results showed a linear relationship, with no indication of a threshold, between PM_{10} and the relative risk of death from all causes and cardiorespiratory causes. In contrast for other causes, the risk of death increased only when a PM_{10} concentration of ~50 μg/m^3 was reached.

The association between particulate and gas-phase urban air pollution and total daily mortality was investigated in eight Canadian cities, using data collected between 1986

and 1996 (Burnett et al., 2000). Mean 24-h average concentrations of $PM_{10-2.5}$ (12.6 $\mu g/m^3$) and $PM_{2.5}$ (13.3 $\mu g/m^3$) were similar. Daily fluctuations in mortality were significantly associated with daily variations in both gas- and particulate-phase pollution, with $PM_{2.5}$ being a better predictor of mortality than $PM_{10-2.5}$. There were strong associations between mortality and sulphate, iron, nickel and zinc in the fine fraction and it was suggested that the complex chemical mixture of the fine fraction may be a better predictor of mortality than mass, although the concentrations and effects of the specific toxic components of fine PM are likely to vary between locations.

Using data from 1986-99 for Vancouver, Canada, the relationship of daily levels of particulate and gaseous pollutants with mortality within a cohort of ~550,000 individuals has been investigated (Villeneuve et al., 2003). The cohort was also stratified according to socioeconomic status by quintiles of income. Increases in the 3-day average concentrations of NO_2 and SO_2 were estimated to increase total daily mortality by 4% and 1.3%, respectively. However, SO_2 was associated with a greater increase (5.6%) in deaths due to respiratory disease. The coarse fraction ($PM_{10-2.5}$) was associated with a 5.9% increase in cardiovascular mortality, while $PM_{2.5}$ was not an important predictor of mortality. For NO_2, CO and SO_2, there was a suggestion that the risk of all-cause and cardiovascular mortality was increased among the population with a lower socioeconomic status.

(ii) Central and South American studies

Associations between airborne PM and daily mortality in Mexico City have been investigated (Castillejos et al., 2000) and the influence of measurement device, region and modelling strategy assessed (O'Neill et al., 2004). Increases in total daily mortality of 1.5%, 1.8% and 4% were observed per 10 $\mu g/m^3$ increments in PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$, respectively. The associations were strongest for $PM_{10-2.5}$ with an 8% increase in the risk of respiratory mortality and a 4.5% increase in the risk of cardiovascular mortality per 10 $\mu g/m^3$ increase in $PM_{10-2.5}$. There were no consistent differences in associations across regions that might have corresponded with variations in particle composition or toxicity. However, the authors concluded that airborne PM, measured by gravimetric methods, and in particular the coarse fraction was associated with daily mortality and presented a health risk in Mexico City.

The effects of fine ($PM_{2.5}$) and coarse particles ($PM_{10-2.5}$), CO, SO_2, NO_2 and O_3 on daily mortality in Santiago, Chile from 1988 to 1996 have been reported (Cifuentes et al., 2000). Mean 24-h average concentrations for $PM_{2.5}$ and $PM_{10-2.5}$ were 58.3 and 46.4 $\mu g/m^3$, respectively, with $PM_{2.5}$ levels being higher in winter (82.4 $\mu g/m^3$) than in summer (32.8 $\mu g/m^3$), whereas levels of $PM_{10-2.5}$ were similar in the two seasons. $PM_{10-2.5}$ was significantly associated with total daily mortality and the increase in mortality was higher for summer (2% per 10 $\mu g/m^3$ increase in $PM_{10-2.5}$) than for winter (0.8%). However, over the whole year and in winter, fine particles were more strongly associated with daily mortality than coarse particles. NO_2 and CO were also significantly associated with daily mortality, as was O_3 during the warmer months. These results suggested that in Santiago, combustion-generated pollutants, especially those generated by motor vehicles, may be associated with increased mortality.

A study from Sao Paulo, Brazil suggests that socioeconomic status may influence the association between daily respiratory mortality in the elderly and air pollution (Martins et al., 2004). Percentage increases in daily respiratory deaths attributable to a 10 $\mu g/m^3$ increase in three-day moving average PM_{10} concentration ranged from 1.4% to 14.2% depending on the region of the city sampled. There were negative correlations between the effect of PM_{10} and both family income and the percentage of the population with a college education, whereas there was a positive correlation with the percentage living in slums. A similar effect modification by socioeconomic status was observed in a study of the association between mortality and indicators of traffic-related air pollution in the Netherlands (Hoek et al., 2002).

(iii) European studies

There were no significant short-term associations between any pollutant and all-cause mortality in London during 1992-4. However, PM_{10} levels were associated with a 4% increase in respiratory mortality and NO_2, black smoke and ozone were associated with cardiovascular deaths (Bremner et al., 1999). Associations of daily mortality with fine and coarse particles, black smoke and sulphate have been investigated in the West Midlands urban area of the UK, using data collected in 1994-6 (Anderson et al., 2001). Mean 24-h average concentrations for PM_{10}, $PM_{10-2.5}$ and $PM_{2.5}$ were 23, 9 and 14.5 $\mu g/m^3$, respectively. Daily all-cause mortality was not associated with any PM fraction, although there were positive, non-significant associations for cardiovascular mortality

with $PM_{2.5}$ and PM_{10}. For respiratory mortality, positive associations with $PM_{2.5}$ and PM_{10} were only observed in the summer. Few statistically significant associations were identified in this study, possibly due to the relatively small size of the data set, but there were strong seasonal interactions in the mortality analysis. Although $PM_{2.5}$ appeared to be more consistently associated with health effects, a contribution of the coarse PM fraction could not be excluded.

Associations of daily mortality with variations in the ambient-air concentrations of O_3, NO_2, CO, SO_2 and PM_{10} in Helsinki, Finland during 1988-96 have been investigated (Penttinen et al., 2004). Spring and summer O_3 concentrations were significantly associated with total mortality (2.4% increase per 10 $\mu g/m^3$ increment in the 4-day mean O_3 level) and respiratory mortality (4.3% increase per 10 $\mu g/m^3$). PM_{10} concentrations were also consistently associated with respiratory mortality (4% increase per 10 $\mu g/m^3$ increment in PM_{10}). This study supported previous findings that coarse mineral particles were not as strongly associated with mortality as fine, combustion-derived particles.

In a study of all-cause mortality from 1983-1991 in Rotterdam, the Netherlands, daily mortality was most consistently associated with previous-day concentrations of total suspended particulates (TSP) and ozone (Hoek et al., 1997). Total iron concentration was less consistently associated with mortality than TSP mass. The relationship between mortality and TSP was linear below 100 $\mu g/m^3$ and the threshold, if it existed, was at very low levels. In another study from the Netherlands (Fischer et al., 2003), associations between daily mortality and short-term variations in ambient ozone, black smoke, SO_2, NO_2, CO and PM were studied. Statistically significant associations between daily mortality and PM_{10}, black smoke, SO_2, NO_2 and CO were identified in the elderly (> 65 years of age). Significant associations in those < 65 years were found for total and COPD mortality with O_3, and for pneumonia mortality with PM_{10}, NO_2 and CO.

A study of the short-term effects of air pollution on mortality in the French cities of Rouen and Le Havre in 1990-95 showed that in Rouen increases in ozone were associated with total mortality, while variations in SO_2 and NO_2 were associated with increases in respiratory and cardiovascular mortality, respectively. In Le Havre,

increases in SO_2 and PM_{13} were associated with cardiovascular mortality (Zeghnoun et al., 2001). In a multi-centre study of air pollution and mortality in Spain, associations with particulates and SO_2 were assessed in 13 Spanish cities for the period 1990-6 (Ballester et al., 2002). Increases in PM_{10} and SO_2 were associated with small increases (~0.5%) in daily deaths, while peak SO_2 concentrations showed significant associations with all-cause, cardiovascular and respiratory mortality. PM_{10}, rather than gaseous pollutants, were associated with mortality among COPD patients in Barcelona (Sunyer and Basagana, 2001). These results suggested that the populations of Spanish cities are exposed to health risks related to air pollution.

The association between air pollution and mortality was investigated for a highly polluted region of the Czech Republic and a rural region of Germany (Peters et al., 2000). In the Czech Republic there was a 3.8% increase in mortality associated with a TSP (68% of which was PM_{10}) concentration of 100 $\mu g/m^3$ for the period 1982-94. There was no evidence for an association between mortality and PM in the rural area of Germany near the Czech border. Deaths due to respiratory infections and heart failure were significantly associated with TSP in an analysis of data from 1980-89 for Milan, Italy (Rossi et al., 1999).

In an attempt to overcome the limitations of using data from one or a few locations, the Air Pollution and Health – A European Approach (APHEA) project was established to examine exposure-response relationships between daily mortality and airborne particles, using an extensive database from 30 European cities (Samoli et al., 2005). For PM_{10} concentrations between 36 and 83$\mu g/m^3$ there were approximately linear associations with total, cardiovascular and respiratory mortality. An increase in PM_{10} from 50 to 60 $\mu g/m^3$ was associated with an increase of about 0.4% in total deaths and increases of about 5% in both cardiovascular and respiratory deaths. The curve for respiratory, but not cardiovascular mortality, showed some deviation from linearity at the lowest concentrations of PM_{10}, suggesting a difference in the mechanisms underlying the association of particulate pollution with these mortality outcomes. Cardiovascular mortality has been reported to increase within the first few days of exposure to PM, whereas increases in respiratory mortality peaked up to 2 weeks later (Goodman et al., 2004). Furthermore, increases in ambient temperature were found to magnify the effect of particulate pollution on mortality.

In Italy, a meta-analysis has been conducted of short-term effects of air pollution for the period 1996-2002 in 15 cities, constituting a population of over 9 million inhabitants (MISA-2) (Biggeri et al., 2004). Overall mortality from all natural causes associated with a PM_{10} increase of 10 μg/m^3 was marginal (0.31%) and did not attain statistical significance. However effect-modification by temperature was demonstrated, and during the warm season an increase of 1.95% in all natural-cause mortality was associated with a PM_{10} increase of 10 μg/m^3. Greater effect size was evident in extreme age groups (Billings and Howard, 1993).

(iv) Australian studies

A similar protocol to that of the APHEA study was used to examine the short-term effects of air pollution on daily mortality in four Australian cities (Sydney, Melbourne, Brisbane and Perth). There were significant effects for PM and NO_2 on total mortality and for O_3 on respiratory mortality. Meta-analyses for three cities gave estimates for the increase in total daily deaths of 0.2% per 10 μg/m^3 increase in PM_{10}, and 0.9% per 10 μg/m^3 increase in $PM_{2.5}$ (Simpson et al., 2005b). A previous study of air pollution and daily mortality in Brisbane for the period 1987-93 had shown that associations between total daily mortality and the levels of particulate matter identified in studies from the US and other countries may be also be applicable in Brisbane (Simpson et al., 1997). The associations of PM and ozone with daily mortality were significant only for individuals > 65 years of age. A similar study examined the effects of air pollution on daily mortality in Melbourne between 1991 and 1996 (Simpson et al., 2000). The 24-h mean PM_{10} and $PM_{2.5}$ concentrations were 19 and 9.4μg/m^3, respectively. During the summer, a 1 μg/m^3 increase in 24-h $PM_{2.5}$ was associated with a 0.4% increased risk of all-cause mortality and a 1.2% increase in the risk of respiratory mortality. For a 1 μg/m^3 increase in PM_{10} the increases in risk were approximately half as much, but still significant.

(v) Asian studies

The short-term effects of suspended particulate matter (SPM) on daily mortality of those aged > 65 years was investigated using data collected in the 13 largest Japanese cities from 1990 through 1994. A 10 μg/m^3 increase in SPM concentrations (adjusted for SO_2, NO_2, CO, O_3, temperature, and humidity) was associated with a 0.8% increased risk of all-cause mortality, a 1.1% increased risk of respiratory mortality, and

a 0.9% increased risk of cardiovascular mortality, suggesting a positive relationship between particulate matter concentrations and daily mortality in Japan (Omori et al., 2003).

Air pollution and daily mortality data from 2000-01 for Shanghai, China showed that increases of 10 μg/m^3 in PM_{10}, SO_2, and NO_2 were associated with relative risks of all-cause mortality of 1.003, 1.014 and 1.015, respectively, and although these risks were small they were all statistically significant. However, these results suggested that gaseous pollutants may have a greater impact on daily mortality in Shanghai than particulate matter (Kan and Chen, 2003). Some confirmation of this is provided by a study of daily mortality in Chongqing in 1995, which showed that the relative risks of all-cause, respiratory and cardiovascular mortality for a 100 μg/m^3 increase in mean daily SO_2 were 1.04, 1.11 and 1.10, respectively. In contrast, associations between daily mortality and mean daily $PM_{2.5}$ were negative and statistically insignificant on all days (Venners et al., 2003).

The association between daily mortality and air pollution was investigated during a 20-month period (1995-6) in Inchon, South Korea. PM_{10} concentration was significantly associated with total, cardiovascular and respiratory mortality, while SO_2 and CO were associated with respiratory mortality. However, the exposure-response relationship with total mortality was best explained by a combined index of PM_{10}, NO_2, SO_2, and CO (Hong et al., 1999). The impact of windblown dust from the deserts of Mongolia and China on daily mortality in Seoul, Korea has also been investigated. Between 1995 and 1998, 28 of these Asian dust events were recorded in Seoul, and the data provided weak evidence of an association between these events and deaths from all causes, but stronger evidence of associations with cardiovascular and respiratory mortality (Kwon et al., 2002).

Two studies performed in Taiwan have raised questions about the wider applicability of associations between daily mortality and air pollution, based on US and European studies. Using daily mortality data for the period 1994-2000 in Kaohsiung, Taiwan, a large industrial city with a tropical climate, no significant associations were found between PM_{10} or SO_2 exposure levels and respiratory mortality (Tsai et al., 2003b). Similarly, in a study of daily mortality for the period 1994-8 in Taipei, the largest

observed effect, which was not statistically significant, was for NO_2 and CO levels on respiratory mortality (OR 1.013 and 1.014, respectively) (Yang et al., 2004a). The authors suggested that the established link between air pollution and daily mortality may not be as strong in subtropical cities, although other factors such as differences in the pollutant mix or underlying health of the population may also play a role.

The importance of seasonal factors and of gaseous oxidant pollutants (SO_2, NO_2, O_3) compared to particulate matter has also been highlighted by studies from Hong Kong. In this subtropical city, daily mortality was significantly associated with the levels of gaseous pollutants during the cool season, while associations with PM_{10} did not reach significance (Wong et al., 2001a). In another study that examined respiratory and cardiovascular mortality in Hong Kong from 1995 to 1998, the relative risk of respiratory mortality for a 10 $\mu g/m^3$ increase in PM_{10} was 1.008, and for SO_2 it was 1.015. The relative risks of ischaemic heart disease mortality ranged from 1.009 for O_3 to 1.028 for SO_2. Therefore in this study the associations with the particulates and gaseous pollutants, when analysed singly, were consistent with those reported from temperate countries (Wong et al., 2002b).

Associations between ambient PM_{10} and daily mortality from 1992 to 1995 have been analysed in Bangkok, Thailand, a tropical metropolitan area in which the main sources of PM_{10} are fine particles generated by diesel- and petrol-powered vehicles. There were statistically significant associations between PM_{10} and all measures of mortality, with a 10 $\mu g/m^3$ increase in daily PM_{10} being associated with a 1-2% increase in cardiovascular mortality and a 3-6% increase in respiratory mortality (Ostro et al., 1999a). An investigation of daily mortality in Bangkok, Thailand from 1992-97, showed that increased PM_{10} concentrations were independently associated with increases in daily all-cause, respiratory and cardiovascular mortality, with the associations being stronger for persons aged $\geq$ 65 years (Vajanapoom et al., 2002). These observations on the relative risks of PM_{10} exposure on mortality were therefore consistent with previous observations from US studies.

In order to gain some understanding of the factors underlying the variability in time-series studies of mortality due to the effects of PM_{10}, Levy et al. (2000) performed an empirical Bayes meta-analysis. For this analysis 29 estimates of the association between

PM and mortality were selected from 21 published studies. The meta-analysis suggested that, on average, mortality rates increased by 0.7% per 10 μg/m^3 increase in PM_{10} concentration and the effect was greater at locations where the $PM_{2.5}/PM_{10}$ ratio was higher. When potential confounders and effect modifiers were included this estimate did not change, although there was some evidence that the effects of PM on mortality were influenced by climate, demographic factors, SO_2 and ozone. There also appeared to be some evidence of publication bias among single-city studies of daily mortality. However, after statistical correction, which reduced the relative risk of mortality for a 10 μg/m^3 increase in PM_{10} concentration from 1.006 to 1.005, the effect was positive and could be precisely estimated (Anderson et al., 2005).

b) Effects of long-term exposure to PM on mortality

In comparison to the large number of studies on the associations between daily variations in air pollutants and short-term mortality outcomes, relatively few studies have examined the effects on mortality of long-term or chronic exposure to air pollutants.

In a study of 6,338 non-smoking Seventh-day Adventists in California, long-term concentrations of PM_{10} and other air pollutants (1973-1992) were related to 15-year mortality data (Abbey et al., 1999). In both sexes, there were strong associations between PM_{10} and mortality due to any non-malignant respiratory disease, and between SO_2 and lung cancer mortality. In males PM_{10} and ozone also showed strong associations with lung cancer deaths. In a subsequent study, data from a subset of this cohort (3,769 individuals) was used to evaluate the relative importance of the fine or coarse fraction of long-term PM_{10} concentrations on mortality outcomes (McDonnell et al., 2000). In single-pollutant models, an interquartile range (IQR) increase in $PM_{2.5}$ (24.3 μg/m^3) was associated with relative risks of mortality in males due to all natural causes, non-malignant respiratory disease and lung cancer of 1.22, 1.64 and 2.23, respectively. For an IQR increase in $PM_{10\text{-}2.5}$ (9.7 μg/m^3), the corresponding relative risks were 1.05, 1.19 and 1.25, respectively. When the $PM_{2.5}$ and $PM_{10\text{-}2.5}$ estimates were both entered into the model, the latter decreased while the former remained stable, leading the authors to conclude that associations of long-term PM_{10} concentrations with mortality in males was best explained by a relationship with the fine rather than the coarse fraction.

Further confirmation of this was provided by the largest cohort study conducted in the US, which found no association between coarse PM and mortality over long follow-up periods (Pope et al., 2002; Brunekreef and Forsberg, 2005). Approximately 1.2 million adults were enrolled in this American Cancer Society study in 1982, and risk factor and mortality data for ~0.5 million adults were linked with air pollution data for metropolitan areas throughout the US up to 1998. Each 10 $\mu g/m^3$ increase in fine PM was associated with approximately 4%, 6% and 8% increased risks of all-cause, cardiopulmonary and lung cancer mortality, respectively, whereas coarse PM and TSP were not consistently associated with mortality. The authors concluded that long-term exposure to combustion-related fine PM is an important risk factor for cardiopulmonary and lung cancer mortality (Pope et al., 2002). Ongoing analysis of data from this cohort study will be used to provide information on the ecological, economic and demographic influences on the association between fine PM and mortality. These analyses will also investigate the role of spatial autocorrelation at multiple geographic scales, and assess the importance of critical episodes of fine PM exposure on the risk of mortality from cardiopulmonary and lung cancer (Krewski et al., 2005). In contrast to these findings, however, a prospective mortality study of ~50,000 US veterans (81% with a smoking history), who were diagnosed as hypertensive in the mid 1970s and were followed-up for 21 years, found no evidence for an association between fine PM and excess mortality risk (Lipfert et al., 2000b).

Long-term exposure to inorganic dust (asbestos, mineral fibres, cement, concrete, quartz) was assessed in a large cohort of Swedish male construction workers, who were followed from 1971 to 1999. There was an increased mortality due to COPD among construction workers exposed to inorganic dust as compared to unexposed construction workers, and the risk was higher among never-smokers (Bergdahl et al., 2004). The results of this study suggested that, regardless of other factors such as smoking, 1 out of 10 COPD deaths could be prevented by reduction of workplace exposures to airborne dust, gases and fumes.

c) Summary – effects of PM on mortality

A number of studies conducted at different locations worldwide indicate that there is an association between ambient PM and mortality. In some locations there is evidence for

an independent effect of coarse PM on short-term mortality, although many studies that have examined the effects of crustal or windblown particles have not identified a significant association with mortality. There is also little or no evidence that long-term exposure to coarse PM is related to increased mortality. In urban areas, there is stronger evidence that exposure to fine PM derived from anthropogenic sources, including fossil fuel combustion and industrial sources, might be associated with increased short-term mortality. Long-term exposure to fine PM also appears to be associated with increased mortality.

It has been estimated that in Europe, a reduction in long-term exposure to PM_{10} concentrations of 5 $\mu g/m^3$ would prevent between 3,300 and 7,700 early deaths per year (Medina et al., 2004). The WHO Global Burden of Disease Comparative Risk Assessment has also estimated the global impact of urban ambient air pollution in terms of mortality. It was estimated that $PM_{2.5}$ air pollution causes ~3% of mortality from cardiopulmonary disease, ~5% of mortality from cancer of the trachea, bronchus and lung, and ~1% of mortality from respiratory infections in children < 5 years of age (Cohen et al., 2005). This burden of 6.4 million years of life lost occurs mainly in developing countries with deaths in Asian countries accounting for 65% of the total.

d) Effects of urban PM on short-term morbidity – hospital admissions

(i) North American studies

A number of studies, mainly from Canada and the US, have examined the effects of air pollutants on cardiorespiratory-related hospital admissions and emergency department visits (Brunekreef and Forsberg, 2005). The roles of particle size and chemistry in cardiorespiratory hospital admissions in Toronto, Canada during the summers of 1992-4 have been investigated. Although significant positive associations were observed for both cardiovascular and respiratory admissions with coarse and fine PM concentrations, particle mass and chemistry were not independent risk factors for cardiorespiratory exacerbations beyond those attributable to gaseous pollutants (O_3, NO_2, SO_2, CO) (Burnett et al., 1997). In a previous study in which air pollution was monitored in Toronto during the summers of 1986-88, $PM_{2.5}$ and PM_{10} were positively associated with respiratory hospital admissions (Thurston et al., 1994). Another study linked respiratory, cardiac, cerebro-vascular, and peripheral vascular disease hospital

admissions in Toronto, from 1980 to 1994, to daily measures of PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, ozone, CO, NO_2, and SO_2 (Burnett et al., 1999). After controlling for climatic factors and gaseous pollutants, 10 $\mu g/m^3$ increases in PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ were associated with 0.50%, 0.75%, and 0.77% increases, respectively, in respiratory and cardiac hospital admissions. It was estimated that of the 7.7 excess daily hospital admissions attributable to air pollution, 11.8% were due to $PM_{2.5}$, 8.2% to $PM_{10-2.5}$, 17% to CO, 40.4% to NO_2, 2.8% to SO_2, and 19.8% to ozone. Summertime ambient ozone levels in Toronto have also been shown to be a risk factor for respiratory problems in children < 2 years of age (Burnett et al., 2001).

Recent studies have investigated the health impacts of air pollution in Windsor, Ontario, a heavily industrialized and polluted city, with major industries including automobile assembly plants, an engine plant, a foundry, and a scrap metal recycling plant. In addition, it is affected by cross-border air and water pollution from the US states of Ohio, Illinois, and Michigan. Associations were identified between NO_2, SO_2, CO, and PM_{10} and daily hospital admissions for respiratory diseases, especially among females in the 0-14 years age group. In addition PM_{10} levels were associated with respiratory admissions among adult males (Luginaah et al., 2005). Short-term SO_2 levels were significantly associated with daily cardiac hospital admissions for people ≥ 65 years of age living in Windsor (Fung et al., 2005). The authors concluded that although the risks of respiratory disease due to ambient air pollution appear to be low in the general population, it is reasonable to assume that the risks would be much higher among susceptible groups.

Hospital admissions in the Utah Valley were investigated during 1985-88, a period that included the closure and re-opening of the local steel mill, which was the main source of PM_{10}. Elevated ambient PM_{10} levels were associated with hospital admissions for pneumonia, pleurisy, bronchitis and asthma. PM_{10} levels were nearly two-fold greater during the winter months when the steel mill was open, compared to the winter months when the mill was closed. Hospital admissions among children were two to three times higher during these periods of high ambient PM levels (Pope, 1989).

A superstation monitored the chemical composition of ambient particles by size, as well as a number of other pollutants, at a site in downtown Atlanta, Georgia during 1998-

2000 and these data have been utilized in a number of morbidity studies, collectively referred to as the "Study of Particulates and Health in Atlanta" (SOPHIA). In the period prior to operation of the superstation (1993-98), emergency department (ED) visits for adult asthma were significantly associated with ozone, and ED visits for COPD with ozone, NO_2 and PM_{10}. During the period of superstation operation, ED visits for all cardiovascular diseases were associated with CO, $PM_{2.5}$ elemental carbon and $PM_{2.5}$ organic matter (Tolbert et al., 2000a). Paediatric ED visits for asthma were related to PM_{10} and ozone concentrations (Tolbert et al., 2000b), while adult ED visits for cardiovascular disease were associated with NO_2, CO, $PM_{2.5}$, organic carbon, elemental carbon, and oxygenated hydrocarbons (Metzger et al., 2004). Subsequent analyses have shown that increases in ozone, NO_2, CO, and PM_{10} were associated with 1-3% increases in ED visits for upper respiratory tract infections, increases in $PM_{2.5}$ organic carbon with a 3% increase in pneumonia visits; and increases in NO_2 and CO with 2-3% increases in COPD-related ED visits (Peel et al., 2005).

The association of PM components with daily morbidity was investigated in elderly residents (≥ 65 years of age) of Detroit, Michigan (Lippmann et al., 2000). Significant associations were observed between $PM_{10-2.5}$ and hospital admissions for pneumonia and ischaemic heart disease among the elderly. Similarly, a study of the association between PM_{10} and hospital admissions for heart and lung disease in ten US cities showed that a 10 $\mu g/m^3$ increase in PM_{10} was associated with a 2.5% increase in COPD admissions, a 2% increase in pneumonia admissions, and a 1.3% increase in admissions for cardiovascular disease. The effects were not modified by poverty or minority status and were stable after controlling for confounding by SO_2, ozone, and CO (Zanobetti et al., 2000).

A study of the relationship between ambient air pollutants in Seattle, Washington and hospital admissions among the non-elderly, for which the principal diagnosis was asthma, found 4-5% increases in hospital admissions associated with 19, 11.8 and 9.3 $\mu g/m^3$ increases in PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$, respectively. PM and CO appeared to be jointly associated with asthma admissions, with the greatest increase in risk occurring during spring and autumn. The authors suggested that the association between the coarse PM fraction and asthma hospital admissions might reflect irritation of the airways by larger particles (Sheppard et al., 1999). However, a more recent study from

Spokane, Washington found no association between respiratory or cardiac emergency department visits and any of the four PM size fractions measured (1, 2.5, 10, 10-2.5) (Slaughter et al., 2005). Another study investigated emergency department visits for asthma between 1991-96 among Medicaid recipients in Cleveland, Cincinnati and Columbus, Ohio. In Cleveland there was a 12% increased likelihood of an asthma ED visit per 50 $\mu g/m^3$ increase in PM_{10} and in Cincinnati, a 35% increase per 50 $\mu g/m^3$ increase in SO_2. For all three cities combined, the risk of an ED visit increased with increases in all pollutants, and specifically by 12% per 50 $\mu g/m^3$ increase in SO_2 (Jaffe et al., 2003).

Associations between air pollution and daily hospital admissions for COPD have been investigated in three metropolitan areas (Chicago, Los Angeles, Phoenix) of the US (Moolgavkar, 2000b). In Chicago and Los Angeles, ozone was associated with COPD admissions only during the summer, while in Los Angeles CO, SO_2 and NO_2 were strongly associated with COPD admissions throughout the year. The results indicated that gaseous pollutants were more strongly associated with COPD admissions than PM, but there was considerable heterogeneity in the effects of individual pollutants in different geographic areas.

(ii) South American studies

A study of the effects of daily air pollution levels in Sao Paulo, Brazil, from 1996 to 1998 on the number of emergency room visits due to chronic lower respiratory disease in people > 64 years of age showed associations with the levels of SO_2 and ozone, but not PM_{10} (Martins et al., 2002). Meanwhile NO_2 levels measured in Sao Paulo in 1996-7 were found to be significantly associated with paediatric emergency room visits for lower respiratory tract diseases, as well as paediatric hospital admissions for pneumonia, asthma or bronchiolitis (Farhat et al., 2005). In another study, of the air pollutants measured in Sao Paulo in 1994-5, only CO levels were significantly associated with daily emergency department visits for ischaemic heart diseases (Lin et al., 2003).

(iii) European studies

The APHEA-2 study investigated short-term effects of particulate air pollution on hospital admissions in eight European cities (Barcelona, Birmingham, London, Milan,

The Netherlands, Paris, Rome, and Stockholm), covering a population of 38 million (Atkinson et al., 2001). Hospital admissions for asthma and COPD among those > 65 years of age were increased by 1%, and admissions for asthma among children (0-14 years) were increased by 1.2% per 10 $\mu g/m^3$ increase in PM_{10}. In the 65+ age groups the effects of PM_{10} were positively associated with annual mean ozone concentrations in the cities, whereas estimates of the effects of PM_{10} on asthma admissions among children were confounded by day-to-day variations in NO_2, suggesting that the traffic-related component of the PM_{10} fraction could have been relevant.

The APHEA-2 data from the same eight European cities were used to specifically examine the association between particulate air pollution and hospital admissions for cardiovascular diseases (Le Tertre et al., 2002). Increases of 10 $\mu g/m^3$ in PM_{10} and black smoke were respectively associated with 0.5% and 1% increases in cardiac admissions for all age groups. The effect of PM_{10} was unchanged by controlling for ozone or SO_2, but was substantially reduced by controlling for the other traffic-related pollutants, CO and NO_2. These results suggested that the primary effect of particulate air pollution on cardiac admissions was likely to be mainly attributable to diesel exhaust.

Older data from phase 1 of the APHEA study provided the basis of a quantitative summary of the short-term effects of air pollution on hospital admissions for respiratory diseases in five European cities (London, Amsterdam, Rotterdam, Paris, Milan) (Spix et al., 1998). The age groups studied were adults (15-64 years) and the elderly (>65 years), and the effects of SO_2, total suspended particles, ozone and NO_2 were investigated. The most consistent finding was a significant increase in daily admissions for respiratory diseases, in both adults and the elderly, with an increase in the levels of ozone.

In contrast a study of the associations between hospital admissions and air pollution in London from 1992 to 1994 found no association between ozone levels and respiratory admissions. However, admissions for respiratory diseases were significantly associated with PM_{10} and SO_2, while admissions for cardiovascular diseases were associated with CO and black smoke (Atkinson et al., 1999). Another case-control study of children aged 5-14 living in north-west London showed no association between risk of hospital admission for asthma or respiratory illness and proxy markers of road traffic pollution

(Wilkinson et al., 1999). However, an investigation of the association between air pollution and cardiovascular hospital admissions in London from 1987-94 found significant associations between admissions for acute myocardial infarction and black smoke, NO_2, CO and SO_2. The population data were consistent with 1 in 50 cases of acute myocardial infarction presenting at London hospitals during this period being triggered by outdoor air pollution (Poloniecki et al., 1997).

Hospital admissions in the West Midlands urban area of the UK were analysed in relation to fine and coarse particles, black smoke and sulphate. For all ages, neither respiratory nor cardiovascular admissions were associated with any air pollutant. However, there was evidence for associations between PM_{10}, $PM_{2.5}$, black smoke and SO_2 and respiratory admissions in the 0-14 age group. PM-related effects were attributed primarily to the fine fraction (Anderson et al., 2001). A recent comparison of the health implications of fine and coarse particles at roadside, urban background and rural sites in the UK concluded that PM_{10} emissions due to anthropogenic activities in London contributed to an additional 410 respiratory hospital admissions per year, while NO_2 accounted for an additional 1,205 respiratory hospital admissions (Namdeo and Bell, 2005).

The association of suspended particles and gaseous pollutants with hospital admissions for respiratory diseases was investigated in the metropolitan area of Rome during 1995-97. Total respiratory admissions were significantly associated with the same-day levels of NO_2 and CO, but not with PM or SO_2. Ozone was only associated with respiratory admissions among children (Fusco et al., 2001). Similar data were used to investigate the association of air pollutants with hospital admissions for acute myocardial infarction in Rome. Positive associations were found for total suspended particulates, NO_2 and CO. The strongest effect was for total suspended particulates and the association tended to be stronger among the elderly (> 74 years of age) and during the warm period of the year (D'Ippoliti et al., 2003). The risk of emergency hospital admissions for asthma among residents of Turin, Italy during 1997-99 increased significantly with increasing exposure to NO_2 and total suspended particulates, which were considered to be indicators of traffic-related air pollution (Migliaretti et al., 2005). The association was particularly evident among young (0-14 years) and elderly (> 64 years) patients. Seven traffic-related air pollutants (NO_2, SO_2, ozone, benzene, formaldehyde, toluene and

PM_{10}) were also found to be significantly associated with acute hospital admissions for respiratory diseases from 1995-2000 in Drammen, Norway (Oftedal et al., 2003). Another study from Oslo, Norway found that although an interquartile increase in PM_{10} was associated with a 1.04 relative risk of hospitalization for respiratory disease, the risk associated with benzene was 1.1, suggesting that pollutants other than PM_{10} were more strongly associated with respiratory admissions (Hagen et al., 2000).

Emergency room admissions due to ischaemic heart disease in Helsinki, Finland from 1987 through 1989 were significantly associated with the ambient levels of NO and ozone, while admissions due to cerebrovascular disease were associated with NO_2, although the levels of these gaseous pollutants were described as being only moderate. PM concentrations were related to long-term transient myocardial ischaemic attacks (Ponka and Virtanen, 1996).

Emergency department visits for cardiac and respiratory diseases in Athens, Greece during 1988 were positively related with the levels of air pollution, while admissions for cardiac and respiratory causes were significantly associated with all indices of air pollution during winter (Pantazopoulou et al., 1995).

Daily emergency department visits for asthma in Barcelona, Spain during 1985-9 were analysed in relation to air pollution levels in summer and winter (Castellsague et al., 1995). Black smoke (TSP) levels were associated with asthma visits in summer but not in winter, with a 25 $\mu g/m^3$ increase in black smoke resulting in a relative risk of 1.082 for asthma visits. NO_2 levels were associated with asthma visits in both summer and winter. The unloading of soybeans into a silo in Barcelona between 1981 and 1987 was associated with large increases in the numbers of patients presenting at emergency departments with acute exacerbations of asthma. Filters were subsequently installed on the silos to prevent the airborne dispersion of allergenic soybean dust and this eliminated the outbreaks of asthma (Anto et al., 1993). A subsequent study investigated the association between asthma admissions for the period 1993-95 and unloading of soybeans at two other Spanish ports, Valencia and A Coruna. A significant association was observed for the unloading of soya husk (relative risk 1.5) and soybeans (relative risk 1.3) in A Coruna, whereas in Valencia a significant association was only identified for the unloading of soybeans at two particular docks. The authors recommended that

appropriate control measures be implemented to reduce soybean dust emissions at harbours with adjacent residential populations (Ballester et al., 1999). Another study conducted in Valencia between 1994-96 found significant associations between hospital admissions for cardiovascular disease and the concentrations of black smoke (particulates), SO_2 and CO (Ballester et al., 2001), while increased levels of ozone and CO were associated with increased emergency department admissions for COPD (Tenias et al., 2002).

(iv) Asian studies

A study of ambient air pollution data for Taipei, Taiwan for the period 1997-2001 showed that on both warm (> 20°C) and cool (< 20°C) days the levels PM_{10}, NO_2 and CO were significantly associated with hospital admissions for cardiovascular disease (Chang et al., 2005) and similar results were obtained from a study in Kaohsiung, Taiwan (Yang et al., 2004b). The air pollution data for Kaohsiung were also used to demonstrate that on warm days the levels of PM_{10}, NO_2, SO_2, CO, and ozone were significantly associated with hospital admissions for both primary intracerebral haemorrhage and ischaemic stroke (Tsai et al., 2003a). In the spring, dust storms originating in the deserts of Mongolia and China are carried by the wind into Taipei, and 54 of these Asian dust storm events were identified during the period 1996-2001. The dust storms were shown to be responsible for a 3.6% increase in hospital admissions for cardiovascular disease one day after the event, although the association was not statistically significant (Chen and Yang, 2005). The Asian dust storm events were, however, significantly associated with daily admissions for primary intracerebral haemorrhagic stroke 3 days after the event (relative risk 1.15) and this was thought to be mainly due to the doubling of mean PM_{10} concentrations on dust storm days (112 $\mu g/m^3$) compared with comparison days (55 $\mu g/m^3$) (Yang et al., 2005a).

In a study of the respiratory morbidity of children in relation to air pollution in four Chinese cities (Lanzhou, Chongqing, Wuhan, Guangzhou), a 50 $\mu g/m^3$ increase in the $PM_{10-2.5}$ concentration was associated with an odds ratio of 1.58 for hospitalization for respiratory diseases suggesting that the effects of the coarse fraction on respiratory health should not be underestimated (Zhang et al., 2002). Daily concentrations of NO_2, SO_2, ozone and PM_{10} in Hong Kong in 1994-5 were associated with significant relative risks of hospital admission for all respiratory diseases, all cardiovascular diseases,

COPD and heart failure. Admissions for asthma, pneumonia and influenza were associated with NO_2, ozone and PM_{10} concentrations and those aged ≥ 65 years were at greater risk (Wong et al., 1999). Air pollution data from 1993-4 have also been used to investigate the relationship with hospital admissions for asthma among children in Hong Kong. The mean concentrations of PM_{10}, NO_2 and SO_2 during this period were 44.1 $\mu g/m^3$, 43.3 $\mu g/m^3$, and 12.2 $\mu g/m^3$, respectively, and the relative risks for asthma admission associated with 10 $\mu g/m^3$ increases in these pollutants were 1.08 for NO_2, 1.06 for SO_2 and 1.03 for PM_{10} (Wong et al., 2001b). In a review of the health outcomes of outdoor air pollution in China, Chen et al. (2004), noted that whereas conventional coal combustion used to be the main source of air pollution in the large cities, mixed coal combustion/motor vehicle emission type pollutants now predominate and are having acute and chronic effects on morbidity and hospital admissions.

Residual confounding, correlation between pollutants, and effect modification need to be taken into consideration when suggesting a causal association between air pollution and daily hospital admissions. However, if similar results are obtained at locations that differ with respect to climate, lifestyle and environmental factors, the argument for a causal association is strengthened. For this reason the effects of air pollution on hospital admissions in Hong Kong and London have been compared (Wong et al., 2002a). For respiratory admissions among those aged ≥ 65 years, the associations with PM_{10}, NO_2, SO_2 and ozone were significant in both cities, but tended to be stronger in the cool season in Hong Kong and in the warm season in London. For cardiac admissions in all age groups, significant associations with PM_{10}, NO_2, and SO_2 were observed in both cities and tended to be stronger in the cool season. These findings that the associations between air pollution and daily cardiorespiratory admissions were remarkably similar in both cities, in spite of differences in social, lifestyle, and environmental factors strengthen the argument that air pollution has detrimental short-term effects on health.

Increased levels of PM_{10}, ozone, NO_2, and CO were risk factors for hospital admissions for ischaemic cardiovascular diseases among the elderly population (> 64 years) of Seoul, Korea, and SO_2 was an additional risk factor during the summer months (Lee et al., 2003). In contrast daily hospital admissions due to angina pectoris in Tehran, Iran were only significantly correlated with the CO level, after controlling for confounding effects (Hosseinpoor et al., 2005). The levels of PM_{10}, SO_2, NO_2, ozone and CO in

Seoul were also significantly associated with the risk of hospital admissions for asthma among children under 15 years of age (Lee et al., 2002). Similarly in Ankara, Turkey average weekly exposures to SO_2 and PM_{10} in the previous week were significantly correlated with emergency department visits for asthma (Berktas and Bircan, 2003), and in Singapore the levels of SO_2 and TSP were positively correlated with daily emergency department visits for asthma in children aged 3-12 years (Chew et al., 1999). These findings suggested that asthmatic children were susceptible to increased levels of SO_2 and TSP, despite the overall ambient levels of these pollutants being generally within the guidelines established by WHO.

An analysis of hospitalizations in Kuching, Malaysia, associated with exposure to PM in smoke from the 1997 Southeast Asian forest fires indicated significant fire-related increases in hospitalizations for COPD and asthma (Mott et al., 2005). There is also some evidence that Gulf War veterans exposed to PM from Kuwaiti oil well fires had an increased risk of hospitalization, although there was no evidence of a dose-response relationship (Smith et al., 2002).

(v) Australian and New Zealand studies

Air pollution in Australia and New Zealand is primarily the consequence of motor vehicle emissions, electricity generation from fossil fuels, heavy industry, and use of wood and coal for home heating (Kjellstrom et al., 2002). An analysis of hospital admissions for all causes and respiratory causes in the Newcastle region from 1979-88, showed that admission rates to Newcastle hospitals were significantly lower than those for the rest of New South Wales. However, residence in the industrial part of the city was positively correlated and mean disposable family income was negatively correlated with all-cause and respiratory admissions (Christie et al., 1992). Another study conducted in the Latrobe Valley, Victoria in 1988 showed that the levels of PM and NO_2 were associated with hospital admissions for COPD, whereas none of the pollutants measured were significantly associated with asthma admissions (Voigt et al., 1998).

In contrast, a study of the relationship between daily hospital admissions and air pollutants in Sydney between 1990 and 1994 showed that an increase in the daily maximum 1-h concentration of NO_2 from the 10th to the 90th percentile was associated

with a 5.3% increase in childhood asthma admissions and a 4.6% increase in COPD admissions. A similar increase in PM concentrations was associated with a 3% increase in COPD admissions, while admissions for heart disease among those > 65 years of age were associated with increased levels of NO_2, ozone and PM. The authors concluded that air pollution in Sydney during this period was associated with increased hospitalization for respiratory and cardiac disease (Morgan et al., 1998). Another study that used the APHEA protocol to investigate daily hospital admissions in Brisbane during the period 1987-94 found that ozone levels were consistently associated with admissions for asthma and respiratory diseases and there was little evidence of a threshold. PM was positively associated with asthma and respiratory admissions in summer, while NO_2 levels did not appear to make a significant contribution (Petroeschevsky et al., 2001). A more recent study examined the effects of air pollution on daily hospital admissions in Brisbane, Melbourne, Perth and Sydney for the period 1996-99. In all cities, fine PM and NO_2 were positively associated with cardiovascular admissions, while in three of the four cities fine PM, NO_2 and ozone had significant impacts on respiratory admissions. In all the analyses there appeared to be a relationship between the effects of PM and NO_2 (Simpson et al., 2005a). A study from Christchurch, New Zealand also found a significant association between ambient PM_{10} levels and cardio-respiratory hospital admissions among both adults and children (McGowan et al., 2002).

In 2000-01 a specific investigation was conducted into the effects of air pollutants on emergency hospital admissions for asthma among children living in four regions of Melbourne. The regional PM_{10} concentration was consistently associated with emergency department asthma presentations, with the central district of Melbourne showing the strongest association (relative risk 1.17). On the other hand, NO_2 and ozone were associated with increased asthma presentations among children living in the Western districts of Melbourne (Erbas et al., 2005). A recent study used data from 1998-2001 to investigate the impact of outdoor air pollution on respiratory hospital admissions among children living in five Australian cities (Brisbane, Canberra, Melbourne, Perth, Sydney) and two New Zealand cities (Auckland, Christchurch) (Barnett et al., 2005). Across all the cities, increases in hospital admissions for pneumonia and bronchitis among children aged 0-4 years, respiratory diseases among those aged 0-14 years and asthma among those aged 5-14 years were associated with

concentrations of $PM_{2.5}$, PM_{10}, NO_2 and SO_2. The largest association for all cities was a 6.0% increase in asthma admissions among children aged 5-14 years related to a 5.1-ppb increase in 24-h NO_2 concentrations. A 7.5 $\mu g/m^3$ increase in PM_{10} was associated with a 1.9% increase in respiratory admissions among the 5-14 year age group, and a meta-analysis of data for Brisbane, Melbourne, Perth and Sydney showed a significant association between 24-h $PM_{2.5}$ concentrations and increases in respiratory admissions for the 0-1 year age group. Meta-analyses for all seven cities also showed significant associations between 1-h maximum NO_2 concentrations and increased respiratory admissions among the 1-4 and 5-14 year age groups.

e) Summary – effects of urban PM on hospital admissions

Numerous studies worldwide have investigated the relationship between exposure to ambient PM and hospital admissions for respiratory or cardiovascular disease. In many studies of admissions for respiratory diseases, including asthma and COPD, the effect of exposure to coarse PM has been found to be as strong or stronger than that of fine PM. Coarse PM may induce adverse lung responses and trigger processes that result in an increased likelihood of hospitalization. Ambient PM levels appear to be consistently associated with increased asthma admissions among children, and with COPD admissions among the elderly. There is also a considerable body of evidence supporting an association between ambient coarse PM and cardiovascular hospital admissions.

f) Effects of urban PM on respiratory function and symptoms in healthy subjects

The effects of ambient PM_{10}, $PM_{2.5}$, SO_4^{2-}, strong acid and ozone on peak expiratory flow (PEF) were studied in 473 non-smoking healthy women (age 19-43 years) living in Virginia during the summers of 1995-96. Morning PEF was significantly associated with strong acid and $PM_{2.5}$ concentrations, with a 10 $\mu g/m^3$ increase in $PM_{2.5}$ being associated with a 0.73 L/min decrease in morning PEF. Ozone was the only pollutant related to evening PEF, with a 7.65 L/min decrease being associated with a 30 ppb increase in the 5-day cumulative lag exposure to ozone. These results did not appear to be influenced by potential confounding factors including environmental tobacco smoke, indoor air quality, pollen or other biogenic factors (Naeher et al., 1999).

Following the collapse of the World Trade Center in New York as a result of the terrorist attacks of September 11, 2001, large amounts of airborne $PM_{2.5}$ were released into the local environment. Calcium sulphate and calcium carbonate were the major components of this $PM_{2.5}$, with much lower levels of transition metals and other elements (McGee et al., 2003). Previously healthy residents living in the exposed area and workers involved in the clean-up have reported increased respiratory symptoms including cough, wheeze, dyspnoea and phlegm resulting from this exposure (Herbstman et al., 2005; Reibman et al., 2005).

The association between PM_{10} pollution and the respiratory health of asymptomatic and symptomatic children was investigated in the Utah Valley in the winter of 1990-91. In both groups of children there were significant associations between PM_{10} concentrations, reductions in PEF and respiratory symptoms such as cough, with the effects of PM_{10} being greater for the symptomatic children (Pope and Dockery, 1992). PM_{10} pollution in the Utah Valley was also significantly associated with absenteeism from primary school (Ransom and Pope, 1992).

The impact of summertime haze episodes on PEF of children attending camps in Philadelphia has been investigated. A decrease in morning PEF was significantly associated with increased PM_{10} concentration (-2.9 L/min per 18 $\mu g/m^3$ increase) and most of the effect was attributable to the fine fraction (Neas et al., 1999). A study of air pollution in southern California and lung function growth in children indicated that exposure to PM_{10}, $PM_{2.5}$, $PM_{10\text{-}2.5}$ and NO_2 was associated with significant deficits in the growth of lung function. It was estimated that for children living in communities exposed to the highest as compared to the lowest levels of these air pollutants there was likely to be cumulative reduction of 3.4% in the growth rate for FEV_1 over the 4-year study period (Gauderman et al., 2000). A more recent study assessed the effect of exposure to air pollution on growth of lung function between the ages of 10 and 18 years, when rapid lung development occurs. In that study, lung function and ambient exposures to ozone, acid vapour, NO_2 and PM were monitored annually for 8 years in 1,759 children from 12 southern California communities. Over the 8-year period, deficits in growth of FEV_1 were significantly associated with exposure to NO_2, acid vapour, $PM_{2.5}$ and elemental carbon, even after adjustment for potential confounders and effect modifiers. Among 18 year-old subjects exposed to the highest levels of

$PM_{2.5}$, 7.9% had a low FEV_1 (< 80% of predicted) compared with 1.6% of those exposed to the lowest levels of $PM_{2.5}$. The authors concluded that current levels of air pollution adversely affect lung development in teenagers and may lead to clinically significant deficits in the FEV_1 attained in adulthood (Gauderman et al., 2004). It has been suggested that a 10 $\mu g/m^3$ increase in PM_{10} concentration may significantly increase the number of subjects with clinically relevant reductions in lung function and this may have important public health implications (Künzli et al., 2000).

The relationship between immune biomarkers and exposure to PM was investigated in a cross-sectional survey of 366 children living in 17 Central European cities with wide variation in levels of air pollution. The numbers of B, CD4+, CD8+ and NK lymphocytes in the blood of these children increased with increasing concentrations of PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ after adjustment for age, gender, parental smoking, laboratory of analysis, and recent respiratory illness. Differences in lymphocyte numbers were more marked for exposure to $PM_{2.5}$ than $PM_{10-2.5}$. It was suggested that long-term exposure to PM, and particularly $PM_{2.5}$ may lead to inflammation of the airways and activation of the innate and adaptive immune systems (Leonardi et al., 2000).

Three longitudinal diary studies were re-analysed to investigate the relative effects of fine and coarse PM on PEF and respiratory symptoms in school children. Data from the Harvard Six Cities Study showed an odds ratio of 1.14 for the association of lower respiratory symptoms with an increase of 8 $\mu g/m^3$ in the coarse fraction, while for an increase of 15 $\mu g/m^3$ in the fine fraction the odds ratio was 1.33. Data from the study performed in Uniontown, Pennsylvania showed that a 15 $\mu g/m^3$ increase in fine particles ($PM_{2.1}$) was associated with a 0.9 L/min reduction in PEF, but there was no association with coarse particles ($PM_{10-2.1}$). Similar results were obtained from the study performed on children living in State College, Pennsylvania, with both fine PM, and especially fine sulphate particles, being associated with reductions in PEF, whereas coarse PM had little effect (Schwartz and Neas, 2000). In contrast, in a study of 7,621 children living in four cities in China, respiratory morbidity was most strongly associated with $PM_{10-2.5}$, although PM_{10} and $PM_{2.5}$ were also positively associated (Zhang et al., 2002). Similarly, coarse PM was the only pollutant significantly associated with new episodes of rhinitis in a study of mothers living in Virginia during the summer of 1995. Other factors that were related to an increased likelihood of new

episodes of rhinitis included the presence of pets in the home, a history of allergies and having children in day care. The authors noted that the independent association of coarse PM with new episodes of rhinitis may relate to the initiation of a local inflammatory response when these particles impact on the upper airways (Zhang et al., 2000).

In order to study the impact of $PM_{2.5}$ on infant respiratory illnesses, 504 infants in Santiago, Chile were followed through the first year of life. After adjusting for gender, socioeconomic level, family history of asthma, minimum temperature, and number of older siblings, an increase of 10 $\mu g/m^3$ in the 24-h average $PM_{2.5}$ concentration was associated with a 5% increase in the risk for wheezing bronchitis, whereas there was no association with ambient NO_2 or SO_2 levels. Therefore fine PM, mostly derived from vehicle exhausts, may adversely affect the respiratory health of infants and potentially contribute to chronic effects later in life (Pino et al., 2004).

g) Effects of urban PM on respiratory health of asthmatic children

In a review of the link between particulate air pollution and asthma, Goldsmith and Kobzik (1999) found that the epidemiological studies up to that time generally showed a positive correlation between PM and increased morbidity among asthmatic patients. Thus studies of asthmatic children living in Mexico City (Romieu et al., 1996), the Czech Republic (Peters et al., 1997a), Finland (Timonen and Pekkanen, 1997) and the Netherlands (Gielen et al., 1997) have all shown that the respiratory health of these children was adversely affected by ambient concentrations of PM. The association of particulate air pollution with PEF and respiratory symptoms was investigated in 49 children (age 8-13 years) with chronic respiratory symptoms living in Kuopio, Finland. These children were followed daily for 6 weeks in spring 1995 when PM_{10} was mainly derived from resuspended soil and street dust. The associations varied with the lag time and only $PM_{2.5}$ with a 1-day lag was significantly associated with morning PEF. However 1-day lagged PM_{10}, $PM_{10-2.5}$, and $PM_{2.5}$ were significantly associated with increased risk of cough (Tiittanen et al., 1999).

Among children with asthma in Southern California, increases in PM_{10} concentration were significantly associated with the risk of bronchitis (odds ratio [OR] 1.4 per 19 $\mu g/m^3$ increase) (McConnell et al., 1999). In a subsequent prospective study,

McConnell et al. (2003) showed that among a cohort of asthmatic children bronchitic symptoms were associated with annual variability in $PM_{2.5}$ (OR 1.09 per $\mu g/m^3$), organic carbon (OR 1.4 per $\mu g/m^3$), NO_2 (OR 1.07 per ppb), and ozone (OR 1.06 per ppb). The authors suggested that organic carbon and NO_2 deserve greater attention as potential causes of chronic bronchitis symptoms in children with asthma. Among asthmatic children from a low-income population in San Diego County, those living near high traffic flows were more likely to make two or more medical care visits for their asthma during the year rather than only one. Therefore repeated exposure to PM from vehicle exhausts may aggravate asthma symptoms in children already diagnosed as having asthma (English et al., 1999). In a study of 133 asthmatic children residing in Seattle, it was estimated that the odds of asthma symptoms occurring were increased by 25% for a 1-ppm increment in CO, 14% for a 10 $\mu g/m^3$ increment in $PM_{1.0}$ and 10% for a 10 $\mu g/m^3$ increment in PM_{10}. It was suggested that CO might be a marker for vehicle exhaust emissions and combustion-derived pollutants that aggravate asthma (Yu et al., 2000).

The effect of air pollutants on respiratory symptoms was specifically investigated in 138 African-American asthmatic children living in Los Angeles. New episodes of cough were associated with exposure to PM_{10}, $PM_{2.5}$, NO_2 and moulds (Cladosporium and Alternaria), while PM_{10} and ozone levels were also associated with use of extra asthma medication (Ostro et al., 2001). In a study of Hispanic children (10-16 years old) with asthma living in a Los Angeles community with a high traffic density, asthma symptoms were positively associated with 24-h concentrations of ozone, NO_2, SO_2, PM_{10}, elemental and organic carbon, and volatile organic compounds, suggesting that air pollutants from traffic and industrial sources were having adverse effects on these asthmatic children (Delfino et al., 2003). The association between traffic-related air pollution and respiratory symptoms in children was supported by a school-based, cross-sectional study conducted in the San Francisco Bay Area in 2001 (Kim et al., 2004). The simultaneous effects of ozone and $PM_{2.5}$ on daily respiratory symptoms and medication use were examined prospectively in 271 asthmatic children (< 12 years old) living in southern New England. The levels of ozone, but not $PM_{2.5}$, were significantly associated with respiratory symptoms and rescue medication use among children using maintenance medication, suggesting that these children were particularly vulnerable to the effects of ozone (Gent et al., 2003).

Some recent studies have specifically examined the effects of exposure to airborne PM on lung function in asthmatic children. Inverse associations were identified between FEV_1 and increasing exposure to PM_{10} and $PM_{2.5}$ measured both within the home and at central sites (Delfino et al., 2004). In a longitudinal study of primary school children with asthma in Detroit there were associations between higher exposures to PM_{10}, $PM_{2.5}$ and ozone, and increased diurnal variability and decreased minimum FEV_1 values among children using inhaled corticosteroids (Lewis et al., 2005). Measurements of exhaled nitric oxide, a marker of airway inflammation, have been used to compare the effects of exposure to indoor-generated $PM_{2.5}$ and ambient-generated $PM_{2.5}$ in asthmatic children. Only ambient-generated $PM_{2.5}$ was significantly associated with an increase in exhaled nitric oxide (5 ppb per 10 $\mu g/m^3$ increase in $PM_{2.5}$), and this effect was only observed in children who were not using corticosteroid therapy (Koenig et al., 2005).

In the Pollution Effects on Asthmatic Children in Europe (PEACE) study, the relationship of short-term changes in air pollution with lung function, respiratory symptoms and medication use was investigated in 14 European centres in the winter of 1993-94 (Roemer et al., 2000a). PM_{10}, black smoke, NO_2 and SO_2 concentrations measured at urban locations were generally higher than those at rural locations. There was no clear association between these pollutants and PEF, the prevalence of symptoms or medication use in asthmatic children. PM_{10} elemental compositions were also measured at some centres, and median iron concentrations ranged from 105 ng/m^3 in urban Oslo to 1,110 ng/m^3 in urban Athens, and from 32 to 517 ng/m^3 at suburban locations in Kuopio and Pszczyna, respectively. Daily concentrations of most elements were not associated with variation in PEF, prevalence of respiratory symptoms or bronchodilator use. However, iron and silica concentrations tended to be negatively associated with PEF and positively associated with the prevalence of phlegm. Thus in this European study, there was only weak evidence that differences in the elemental composition of PM_{10} contributed to variations in acute respiratory health effects among asthmatic children (Roemer et al., 2000b).

In a study from the Netherlands, PEF and respiratory symptoms were investigated in 7-11 year old asthmatic children living in urban areas with high traffic densities and in non-urban areas. In children from both areas, decrements in PEF were significantly associated with PM_{10} and black smoke, but children with symptoms appeared to be

more susceptible to the effects of PM than children without symptoms (van der Zee et al., 1999). In a study that assessed the short-term effects of photo-oxidant and particulate air pollution on childhood asthma in Paris during the spring, black smoke and NO_2 were associated with increased rates of nocturnal cough and respiratory infections. Ozone was associated with an increase in asthma attacks and respiratory infections and with decreases in PEF and increased PEF variability (Just et al., 2002).

A study of the association between air pollution and lung function in asthmatic children living near two power plants in Israel showed a significant direct effect of $PM_{2.5}$ on PEF at one location (Peled et al., 2005). Among children exposed to SO_2 and PM from a power plant in Thailand, SO_2 was not associated with respiratory symptoms in either asthmatics or non-asthmatics, whereas increases in PM_{10} were associated with increases in the incidence of lower respiratory symptoms and cough among asthmatic children (Aekplakorn et al., 2003).

In a large cross-sectional study of high school students in Taiwan, conducted in 1995-96, the prevalence of asthma was independently associated with total suspended particulates, NO_2, CO, ozone, and airborne dust particles (Wang et al., 1999). In a larger nationwide survey of Taiwanese middle-school students, asthma prevalence was associated with traffic-related air pollution, especially NO_2 and CO (Guo et al., 1999). NO_2 concentrations were also significantly associated with the incidence of asthma in a large prospective study of school children from eight different communities in Japan. PM_{10} concentrations were also associated with a higher incidence of asthma, although not significantly (Shima et al., 2002). In a group of primary school children with a history of wheeze, living in Sydney, Australia, there were associations between PM_{10} concentrations and doctor visits for asthma and between NO_2 concentrations and wet cough. However, the authors were unable to demonstrate that current levels of air pollution in western Sydney had consistent adverse effects on children with a history of wheezing (Jalaludin et al., 2004). A previous study also showed that high levels of PM_{10} (up to 210 $\mu g/m^3$) resulting from the January 1994 Sydney bushfires did not lead to any clinically significant reductions in PEF in children with a history of wheeze (Jalaludin et al., 2000).

h) Effects of urban PM on respiratory health of adults with lung disease

(i) Asthma

Epidemiological studies suggest that although increases in the levels of PM_{10} do not enhance the likelihood of initial sensitisation and induction of asthma, symptoms may be worsened in patients with the disease (Donaldson et al., 2000), and PM may interact with allergens to enhance allergic airway responses (Polosa, 2001; D'Amato et al., 2001).

A study was performed in Helsinki, Finland to assess the effects of particulate air pollutants on respiratory health in adult asthmatics, and in particular whether the effects of PM_{10} from resuspended road dust differed from those of traffic-related PM_{10}. The daily mean particle number concentration, but not particle mass, was significantly associated with reductions in daily PEF, with the strongest effects being observed for ultrafine particles ($PM_{0.1}$). There were no associations with respiratory symptoms or medication use (Penttinen et al., 2001b). Another study showed that the number of accumulation mode particles ($PM_{1.0\text{-}0.1}$) was consistently inversely associated with spirometric PEFR (Penttinen et al., 2001a). However, a recent review concluded that the evidence that traffic-related pollutants contributed to an increased risk of asthma and hay fever was still weak (Heinrich and Wichmann, 2004).

The association between respiratory symptoms in asthmatic subjects and daily air pollution was analysed in Spokane, Washington, a semi-arid city with diverse sources of PM, including motor vehicles, woodstoves, agricultural burning, resuspended road dust, and dust storms. In children there was a strong association between cough and PM_{10}, $PM_{2.5}$, $PM_{10\text{-}2.5}$, and $PM_{1.0}$, whereas in the adult subjects no respiratory symptom was associated with PM measurements. This suggested that children with asthma may be more sensitive than adult asthmatics to the effects of increased levels of PM (Mar et al., 2004). Controlled exposures of healthy and asthmatic adults to fine ($PM_{2.5}$) and ultrafine ($PM_{0.1}$) particles did not show any effects on respiratory symptoms or lung function (Gong et al., 2003; Frampton et al., 2004).

Subjects with a history of asthma were particularly susceptible to respiratory problems associated with “hazardous” levels of PM_{10} resulting from the 1997 forest fires in

Indonesia (Kunii et al., 2002). In Australia, wind erosion in the arid inland results in the formation of wind-blown dust plumes that may impact on coastal cities such as Brisbane. These dust events may be characterised by high levels of PM with high ratios of fine to coarse particulates. An analysis of 11 dust events in Brisbane between 1992-94 indicated significant associations with changes in asthma severity, but general relationships were not apparent (Rutherford et al., 1999). Similarly, increased levels of PM_{10} are experienced in Korea during Asian dust events, 14 of which were recorded in 2002 (Park et al., 2005a). Increased PM_{10} concentrations during these events were associated with increases in PEF variability, night-time symptoms and a decrease in the mean PEF in asthmatic subjects.

(ii) COPD

The effects of urban air pollution on patients with COPD have been reviewed by Sunyer (2001). A study of 624 smokers with mild-to-moderate COPD living in Salt Lake City, USA, between 1987-89 showed that FEV_1 on the day of testing was associated with the ambient level of PM_{10}, although this association only explained 2-3% of the variation in FEV_1 (Pope and Kanner, 1993). In Christchurch, New Zealand a 3-month study of 55 COPD patients showed that a 35 $\mu g/m^3$ increase in PM_{10} was associated with an increased risk of night-time chest symptoms (relative risk 1.38), while increased NO_2 concentrations were associated with increased use of reliever medication (relative risk 1.42) (Harre et al., 1997). The risk of COPD symptoms as a result of long-term exposure to ambient levels of total suspended particulates (TSP) was assessed in non-smoking Seventh-Day Adventists living in California. Chronic symptoms were significantly associated with TSP concentrations above 200 $\mu g/m^3$, with a relative risk of 1.22 for 750 h of exposure per year (Euler et al., 1987). In a cross-sectional study of long-term exposure to ambient air pollution and respiratory symptoms in a random population of adults in Switzerland, concentrations of TSP and PM_{10} were positively associated with COPD symptoms such as chronic phlegm production, chronic cough, and dyspnoea at rest during the day or at night, or on exertion (Zemp et al., 1999). Changes in lung function over time were assessed in several smoking and non-smoking cohorts living in Southern California, and who were exposed different levels of air pollution. The results suggested that chronic exposure to high levels of PM, sulphates and NO_2 may lead to the development of COPD (Tashkin et al., 1994; Sunyer, 2001).

In a study of day-to-day PM exposure and health changes in 30 Los Angeles residents with severe COPD, only blood pressure showed a consistent unfavourable longitudinal association with PM (Linn et al., 1999). However, in a study of 16 non-smoking COPD patients living in Vancouver, it was estimated that each 10 $\mu g/m^3$ increase in PM_{10} and $PM_{2.5}$ was associated with declines in daily FEV_1 of 3% and 1%, respectively, although these changes were not statistically significant. There was also a weak association between PM concentrations and decreased systolic blood pressure. Ambient PM_{10} concentration was most consistently associated with health effects, but the strength of associations and effect estimates were not increased by using personal exposure data (Brauer et al., 2001).

Personal exposures to PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ were investigated in 18 subjects with COPD living in Boston (Rojas-Bracho et al., 2004). Time-weighted indoor concentrations and time spent cleaning were significant predictors of personal PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ exposures. Time-weighted outdoor concentrations, time spent near smokers, and time spent during transportation were important predictors for $PM_{2.5}$ exposure, while cooking affected personal $PM_{10-2.5}$ exposure.

The effects of air pollution during two winters were studied in patients with advanced COPD living in Denver, Colorado. During the first winter no adverse associations were identified. However during the second winter, which was colder and more humid, increased levels of PM_{10}, CO and NO_2 were associated with worse lung function and increased use of rescue medication (Silkoff et al., 2005). However, ozone was the only air pollutant associated with exacerbations of COPD in 39 residents of Paris with severe COPD, who were monitored for 14 months (Desqueyroux et al., 2002).

In a nested case-control study from Athens, Greece, personal exposures to air pollution were assessed on the basis of long-term residential and occupational history as well as geographical distribution of pollution. Participants reporting a history of COPD symptoms (n=168) and healthy matched control subjects were visited by a physician who assessed their spirometry and health status. Cases were found to have been more exposed to air pollution than controls, with the odds ratio indicating a 37% increase in the risk of medically confirmed chronic bronchitis, emphysema or COPD. The authors concluded that long-term exposure to air pollution contributes to the development of

chronic respiratory diseases (Karakatsani et al., 2003). This was supported by an Italian study showing that the prevalence of COPD was lower in a windy hill-country region with low humidity and low levels of chemical pollution, compared to that in a large city such as Rome (Avino et al., 2004), although neither of these studies identified the specific components of air pollution that might be responsible for adverse effects.

Although it is difficult to separate the effects of smoking from occupational exposures, there is now a large body of evidence indicating that occupational exposure to various inorganic (mineral) dusts contributes to the development and morbidity of COPD (reviewed in American Thoracic Society Statement, 2003). Longitudinal studies have identified associations between COPD and occupational exposures among coal miners (Coggon and Newman Taylor, 1998), hard-rock miners (Holman et al., 1987), tunnel workers (Ulvestad et al., 2001), concrete-manufacturing workers (Meijer et al., 2001), non-mining industrial workers (Kauffmann et al., 1982), and in community-based populations (Xu et al., 1992). A population-based survey in the US found that after adjustment for age, smoking and other factors odds ratios for COPD were increased for a number of occupations, including freight, stock, and material handlers; records processing and distribution clerks; sales; transportation-related occupations; machine operators; construction trades; and waitresses. It was estimated that the fraction of COPD attributable to work was 19.2% overall and 31.1% among never smokers (Hnizdo et al., 2002). Among gold miners in Kalgoorlie, Western Australia, the prevalence of chronic bronchitis was 14%. After controlling for the effects of age and smoking, it was estimated that 1-9 years of work as an underground gold miner was associated with an odds ratio of 1.8 for developing chronic bronchitis, while after 20 years or more the odds ratio increased to 5.1. (Holman et al., 1987).

The relationship between occupational exposure and changes in lung function and chronic bronchitis symptoms was investigated during a 9-year follow up of 20 to 45 year-old subjects from the general population, randomly selected from within the European Community Respiratory Health Survey in 1991-93. Lung function did not decline more steeply in individuals exposed to dust, gases and fumes, although the incidence of chronic phlegm was increased in men exposed to mineral dust (relative risk 1.9) and this was not modified by smoking (Sunyer et al., 2005). However, in a cross-sectional study of randomly selected 45 to 70 year-old residents of Melbourne,

Australia, exposure to mineral dust or gases and fumes did not confer a significantly increased risk for COPD, whereas exposure to biological dust in the workplace was associated with increased risks of chronic bronchitis, emphysema and COPD, with odds ratios of approximately 3 (Matheson et al., 2005).

In dry-climate farming regions, soil perturbation results in respirable dust exposures of 1,000-5,000 $\mu g/m^3$ among farmers and agricultural workers. These exposures reflect the soil composition, with crystalline silica and silicates being the major components. Exposure to high concentrations of inorganic (mineral) dust may partly explain the increased incidence or morbidity due to COPD and pulmonary fibrosis that have been identified in many studies of farmers and agricultural workers (Schenker, 2000). However, many of these exposures are to mixed organic and inorganic dusts and the independent contribution of mineral dusts beyond that of organic dusts remains uncertain.

Histological studies of lung sections from subjects living in Mexico City, a high PM location, compared with those living in Vancouver, Canada, a low PM region, indicate that PM penetrates the walls of the small airways and may contribute to small airway remodelling in subjects exposed to high levels of ambient PM over long periods of time. Airway remodelling may in turn lead to chronic airflow obstruction in these subjects (Churg et al., 2003).

i) The association between exposure to urban PM and the incidence of lung cancer

In a prospective cohort study of 8,000 adults in six US cities, air pollution was found to be positively associated with death from lung cancer, after adjustment for other risk factors such as smoking (Dockery et al., 1993). However, in a cohort study of the Danish population, besides smoking, occupational risk factors were identified as having an important role in lung cancer incidence, whereas air pollution had an identifiable influence only at high levels (Engholm et al., 1996).

The Adventist Health Study in California investigated the relationship between long-term concentrations of ambient air pollutants and the incidence of lung cancer in non-smoking adults aged 27-95 years. Incident lung cancer in males was associated with

increases in mean concentrations of PM_{10} (relative risk 5.2), ozone (relative risk 3.6) and SO_2 (relative risk 2.7). The relative risks were smaller in females, possibly due to gender differences in exposure (Beeson et al., 1998). A subsequent study used data from the same cohort of subjects to investigate the association of mortality with the fine ($PM_{2.5}$) and coarse ($PM_{10-2.5}$) fractions of PM_{10}. A 30 $\mu g/m^3$ increase in PM_{10} was associated with a relative risk of 1.8 for mortality due to lung cancer, while for 24 $\mu g/m^3$ increases in $PM_{2.5}$ and 10 $\mu g/m^3$ increases in $PM_{10-2.5}$ the relative risks were 2.2 and 1.3, respectively. The authors concluded that associations of long-term ambient PM_{10} concentration with lung cancer mortality among males was best explained by a relationship with the fine rather than the coarse fraction of PM_{10} (McDonnell et al., 2000).

The relationship between long-term exposure to fine particulate air pollution and lung cancer mortality was assessed in the American Cancer Society study, which analysed risk factor data for ~0.5 million adults living in metropolitan areas throughout the US (Pope et al., 2002). Each 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration was associated with approximately an 8% increased risk of mortality due to lung cancer, whereas there was no consistent association with the coarse particle fraction. In addition, long-term exposure to NO_2 was found to increase the risk for developing lung cancer (relative risk 1.08 per 10 $\mu g/m^3$ increase in NO_2), after adjustment for age and smoking habit, among ~16,200 men living in Oslo, Norway (Nafstad et al., 2003). Similarly, in a population-based case-control study of men aged 40-75 years living in Stockholm, Sweden, the top decile of average traffic-related NO_2 exposure over 30 years was associated with a relative risk of 1.2 for lung cancer (Nyberg et al., 2000).

In a case-control study from an industrialized area in Italy, smoking habit and occupational exposure to asbestos were significantly associated with an increased risk of lung cancer, whereas reported traffic levels in the area of residence and residence near a cement factory, power plants or a harbour were not (Fano et al., 2004).

Exposure to air pollution has been estimated to contribute to 62,000 lung cancer deaths per year worldwide. It is thought that the major burden of mortality is borne by developing countries such as China where air pollution is much worse than it is in the developed West (Cohen, 2003).

Although the evidence now seems reasonably strong that air pollution does cause some cases of lung cancer, questions remain as to how many excess cases are likely to be caused by pollutants, which pollutants are responsible, and how many cases are due to the combined effects of smoking and air pollution (Cohen, 2003). Based on a review of the data from the American Cancer Society cohort study (Pope et al., 2002), Harrison et al. (2004) concluded that, assuming a latency period of 20 years or more, known airborne chemical carcinogens such as polycyclic aromatic hydrocarbons (PAH), Cr, Ni and As may account for the carcinogenic effects of $PM_{2.5}$. However, historic data cannot be used to demonstrate a correlation between concentrations of chemical carcinogens and $PM_{2.5}$ concentrations at the time of exposure and it is plausible that PM might be carcinogenic in its own right, irrespective of its content of chemical carcinogens. The question of whether a specific component of PM causes lung cancer is not easily addressed, because the constituents of PM vary with the source of pollution and time of exposure (Forastiere, 2004). For example, exhaust fumes from diesel engines contain thousands of combustion products adsorbed onto small solid carbon particles, including mutagenic PAH derivatives that induce oxidative damage to DNA (Vineis and Husgafvel-Pursiainen, 2005).

In addition to carrying adsorbed carcinogenic components into the lung, particles are also thought to exert genotoxic effects through their capacity to generate reactive oxygen and nitrogen species (Knaapen et al., 2004). The soluble metal constituents of PM have been proposed to play a role in the oxidative stress and DNA damage caused by particulate matter (Knaapen et al., 2002). In a study of personal exposures among students in Copenhagen, the concentrations of vanadium and chromium in $PM_{2.5}$ samples, but not iron, nickel, copper or platinum, were significantly associated with a marker of oxidative DNA damage in peripheral blood lymphocytes (Sorensen et al., 2005). Leukocyte DNA adducts were found to be associated with the subsequent risk of lung cancer in a case-control study nested within the European Prospective Investigation into Cancer and Nutrition (EPIC), suggesting these DNA adducts may predict lung cancer risk among never-smokers (Peluso et al., 2005).

j) Summary – effects of urban PM on respiratory health

Fine PM appears to have a stronger and more consistent association with reduction in lung function, as measured by PEF, than coarse PM. Long-term exposure to $PM_{2.5}$

appears to adversely affect the development of lung function among teenagers. In contrast, many studies have shown significant associations between exposure to coarse PM and the incidence of respiratory symptoms in both healthy subjects and those with lung disease. A number of studies have examined the effects of PM on the respiratory health of asthmatic children. Many of these studies have shown that exposure to PM, both coarse and fine, increases the likelihood of asthma symptoms, chronic bronchitis and cough among asthmatic children. However, the available evidence suggests that adults with asthma are much less sensitive to the effects of increased levels of PM than children. The evidence that traffic-related pollutants contribute to the risk of asthma symptoms among adults is weak. Cross-sectional studies have shown that increased levels of PM are associated with higher rates of diagnosis of chronic bronchitis and emphysema. High levels of PM are associated with increased breathlessness and mucus production, and adverse effects on lung function in patients with COPD. Occupational exposure to various inorganic dusts is associated with the development and morbidity of COPD, independent of the effects of smoking. Long-term exposure to combustion-derived fine PM is an important risk factor for mortality from lung cancer. $PM_{2.5}$ or carcinogenic substances associated with fine PM may be causally related to the development of some lung cancers, either alone or in combination with tobacco smoke.

k) Effects of urban particulate matter on cardiovascular disease

During the past 15 years there has been a substantial increase in the strength of evidence and number of studies linking air pollution and cardiovascular disease (Pope, 2000; Brook et al., 2004; Brunekreef and Holgate, 2002), with most of the evidence coming from studies that link exposure to ambient PM with cardiovascular disease (Peters, 2005; Mastin, 2005). The mechanisms by which exposure to fine particulate matter increases the specific risk for cardiovascular mortality include pulmonary and systemic inflammation, accelerated atherosclerosis, and altered cardiac autonomic function (Pope et al., 2004a). Apart from the studies on cardiovascular mortality and hospital admissions that have been previously referred to in this review, there are also studies that have investigated the associations between air pollution or exposure to PM and cardiovascular events or symptoms in various populations.

In a study of 772 patients in the greater Boston area, the risk of onset of acute myocardial infarction was increased with elevation in the concentrations of ambient

$PM_{2.5}$ over the previous 2-h period. The estimated odds ratio associated with a 25 $\mu g/m^3$ increase in $PM_{2.5}$ was 1.5, suggesting that exposure to increased concentrations of fine PM may transiently elevate the risk of myocardial infarction (Peters et al., 2001a). A recent study from Augsburg, Germany showed that exposure to traffic, whether as a result of time spent in cars, on public transport, motor cycles or bicycles, was consistently associated with an increased risk of myocardial infarction within 1 h afterwards (Peters et al., 2004). Among 22,000 myocardial infarction survivors in five European cities, cardiac readmissions increased in association with concentrations of PM_{10} and ultrafine particles (von Klot et al., 2005). However, a study from Seattle, Washington found no association between PM_{10} levels measured on the same day and increased risk of cardiac arrest among patients with pre-existing cardiac disease. There was also no consistent association between increased levels of PM and risk of primary cardiac arrest (Sullivan et al., 2003, 2005a).

A study of small numbers of healthy subjects before, during and after episodes of high particulate air pollution observed changes in mean heart rate and heart rate variability, possibly reflecting changes in cardiac autonomic function (Pope et al., 1999b). Observations on twenty-one 53-87 year-old active Boston residents indicated significantly less heart rate variability associated with elevated concentrations of ambient $PM_{2.5}$ (Gold et al., 2000). Similar results were obtained in a subsequent panel study of 28 elderly subjects, but it was noted that the stronger association was with black carbon, an indicator of traffic-derived particles (Schwartz et al., 2005). Continuous monitoring of PM exposure and heart rate in an occupational cohort during and away from work showed a decrease in heart rate variability with increases in the $PM_{2.5}$ concentration, suggesting that workers experienced alterations in cardiac autonomic control after exposure to occupational and environmental $PM_{2.5}$ (Magari et al., 2001). Exposure to $PM_{2.5}$ was also associated with decreased heart rate variability among elderly non-smoking residents of a retirement centre (Creason et al., 2001), and this finding has been confirmed by more recent panel studies of elderly subjects in Utah (Pope et al., 2004b) and Mexico City (Holguin et al., 2003). In a study of 30 patients with known ischaemic heart disease in Mexico City, heart rate variability decreased with increasing personal $PM_{2.5}$ exposure, suggesting that alteration of cardiac autonomic regulation in this high-risk population was associated with $PM_{2.5}$ exposure (Riojas-Rodriguez et al., 2006). In the VA normative aging study conducted in Boston

between 2000-03, the association between $PM_{2.5}$ and a reduction in heart rate variability was stronger in subjects with ischaemic heart disease, hypertension and diabetes (Park et al., 2005b). A recent population-based study also found evidence that higher ambient PM_{10} concentrations were associated with lower cardiac autonomic control, especially among persons with existing cardiovascular disease (Liao et al., 2004).

In contrast, a recent study of elderly individuals in Seattle found no association between increased residential levels of $PM_{2.5}$ and measures of heart rate variability (Sullivan et al., 2005b). Possible reasons for this conflicting result include a difference in the composition of the $PM_{2.5}$, particularly the content of reactive metals and sulphates, that there were too few periods of high exposure to cause detectable effects on heart rate variability, and the inclusion of patients with cardiovascular disease, many of whom would have been taking drugs that influence heart rate variability (Townend, 2005).

The ambient levels of $PM_{2.5}$ (odds ratio 2.8) and ultrafine particles (odds ratio 3.1) were found to be significantly associated with the risk of ST-segment depression during exercise in subjects with coronary heart disease, whereas there was no consistent association with coarse particles (Pekkanen et al., 2002). Based on these results it was suggested that particulate air pollution may influence cardiovascular morbidity, at least in part, by increasing susceptibility to myocardial ischaemia. In patients given implantable cardioverter defibrillators in Boston, there was a 19% increased risk of ventricular arrhythmia for an interquartile range increase in average $PM_{2.5}$ concentration (Rich et al., 2005).

Controlled inhalation of concentrated ambient fine particles plus ozone at environmentally relevant concentrations by healthy adults has been shown to cause acute arterial vasoconstriction (Brook et al., 2002) and rapid increases in diastolic blood pressure, which were associated with the $PM_{2.5}$ carbon content (Urch et al., 2005). Furthermore, a population-based study of blood pressure during a 1985 air pollution episode in Augsburg showed that systolic blood pressure increased by 1.8 mm Hg per 90 $\mu g/m^3$ of total suspended particulates. In subgroups with high plasma viscosity, systolic blood pressure increased by 6.9 mm Hg in association with total suspended particulates (Ibald-Mulli et al., 2001). Exposure to PM_{10}, CO and NO_2 was also significantly associated with hospital admissions among patients with congestive heart

failure, suggesting that traffic-related pollutants may trigger acute cardiac decompensation in these patients (Wellenius et al., 2005).

An association has also been identified between air pollution and increased plasma viscosity, which was measured in 3,256 randomly selected adults as part of the MONICA Augsburg survey during the winter of 1984-5. During a 13-day period, high concentrations of total suspended particulates (mean 98 $\mu g/m^3$) and SO_2 (mean 200 $\mu g/m^3$) were recorded. Comparison of measurements made during or outside this high pollution period revealed odds ratios of 3.6 among men and 2.3 among women for plasma viscosity above the 95^{th} percentile. The authors speculated that air pollutants may trigger inflammatory processes in the lung with induction of an acute-phase reaction that results in altered blood rheology (Peters et al., 1997b). This was supported by a prospective cohort study based on the MONICA Augsburg survey, which identified an association between the 1985 air pollution episode and increased serum concentrations of C-reactive protein (Peters et al., 2001b). Exposure to ambient PM and its effects on C-reactive protein may be risk factors for coronary artery disease (Sandhu et al., 2005).

The association between haematological variables and personal exposure to PM_{10} was investigated in 112 individuals > 60 years of age living in two cities in the UK. Increased personal exposures to PM_{10} were associated with lower haemoglobin concentrations, packed cell volumes and erythrocyte counts. Serum C-reactive protein levels were positively associated with city centre PM_{10} measurements. The authors suggested that inhalation of some component of PM_{10} affects lung endothelial cells or erythrocytes themselves, so as to increase cell adhesiveness and this may result in sequestration of erythrocytes in the circulation (Seaton et al., 1999).

Carotid intima-media thickness (CIMT), a measure of subclinical atherosclerosis, has recently been assessed in ~800 Los Angeles residents in relation to $PM_{2.5}$ exposure (Künzli et al., 2005). For an increase of 10 $\mu g/m^3$ in $PM_{2.5}$ concentration, CIMT increased by 5.9% and the association was strongest among those ≥ 60 years of age, women, never-smokers and those taking lipid-lowering treatment. This study provides the first epidemiological evidence of an association between atherosclerosis and exposure to ambient $PM_{2.5}$. Exposure to $PM_{2.5}$, black carbon and sulphates was also

associated with decreased endothelial dilation in subjects with diabetes mellitus (Rajagopalan et al., 2005; O'Neill et al., 2005). Potential mechanisms for the interaction between the ultrafine component of PM_{10}, the acute phase response and cardiovascular disease have been reviewed (Donaldson et al., 2001).

l) Summary – effects of urban PM on cardiovascular disease

There is now a substantial body of evidence linking exposure to ambient PM with cardiovascular disease. Exposure to fine PM increases the risk for cardiovascular mortality by inducing pulmonary and systemic inflammation, accelerating atherosclerosis and altering cardiac autonomic function. Acute exposure to increased concentrations of fine PM may transiently elevate the risk of myocardial infarction. Exposure to $PM_{2.5}$ is also associated with a reduction in heart rate variability among the elderly and subjects with ischaemic heart disease, hypertension and diabetes. Increases in the levels of ambient PM have also been found to be associated with increases in blood pressure, plasma viscosity and serum C-reactive protein among healthy subjects, and with decreased endothelial dilation in subjects with diabetes.

m) Health effects of urban ultrafine particles ($PM_{0.1}$, UFP)

Data on exposures to UFP and their health effects are still limited (Englert, 2004). However almost ten years ago, it was observed that the effects of the number of UFP on PEF and respiratory symptoms of adult asthmatic subjects were greater than those of the mass of $PM_{2.5}$ or PM_{10} (Peters et al., 1997c). In a study of asthmatic children, however, the ambient concentration of UFP was no more strongly associated with variations in PEF than PM_{10} concentration (Pekkanen et al., 1997).

It has been suggested that inhalation of UFP could lead to exacerbation of underlying cardio-respiratory disease by inducing airway inflammation, and by altering leukocyte and endothelial adhesion molecule expression, blood coagulability and cardiac electrical activity (Utell and Frampton, 2000). The toxic effects of UFP appear not to be mediated by transition metals, but rather by generation of free radicals that cause oxidative stress and alterations in calcium signalling with consequent induction of inflammatory processes, promotion of atherosclerosis and precipitation of acute cardiovascular responses (Delfino et al., 2005; MacNee and Donaldson, 2003). It has also been postulated that because there is a strong correlation between UFP numbers and NO_2

concentrations in urban air, epidemiological associations between respiratory illness and NO_2 may be the result of confounding by particle numbers (Seaton and Dennekamp, 2003).

Evidence supporting the adverse health effects of UFP came from a study of asthmatic subjects in Finland, which found that the daily mean number concentration of particles, and particularly UFP, in urban air was inversely associated with PEF variations (Penttinen et al., 2001b). However, the effect of UFP could not be separated from that of the other traffic-related pollutants, NO_2 and CO. Epidemiological evidence for the health effects of UFP has also come from panel studies of patients with chronic pulmonary disease in Germany, Finland and the UK (Ibald-Mulli et al., 2002). Overall, decreased PEF and an increase in symptoms and medication use were found with elevation of both fine and ultrafine particles. However, fine particles showed more immediate effects while UFP showed more delayed effects on mortality.

In a panel study of elderly subjects with coronary heart disease living in Amsterdam, Erfurt and Helsinki, the number concentration of ambient UFP was associated only with avoidance of activity, whereas $PM_{2.5}$ levels were more strongly related to cardiorespiratory symptoms (de Hartog et al., 2003). In a recent analysis of 5,144 out-of-hospital fatalities among residents of Rome, particle number concentration, a measure of exposure to UFP, was significantly associated with fatal coronary events (Forastiere et al., 2005).

n) Relevance to Port Hedland of urban epidemiological studies of the health effects of particulate matter

Almost all epidemiological studies of the health effects of particulate matter have been conducted in urban areas, which are impacted by PM that is likely to be quite different in size distribution and composition to the PM impacting a semi-arid rural environment such as Port Hedland. Therefore the extent to which this epidemiological data can be extrapolated from urban environments to Port Hedland is uncertain, and should be the subject of future studies.

6.3 Toxicology of airborne particulates

The epidemiological evidence on the relationship between airborne PM and health is complemented by an extensive body of toxicological studies. Design of appropriate toxicological studies and interpretation of the experimental data are challenging, by virtue of the physicochemical complexity of PM in any given setting and the considerable heterogeneity of PM in different geographical and seasonal contexts. Additionally, the epidemiological applicability of toxicological data is often compromised by extrapolation from comparatively very high doses delivered acutely in experimental contexts, and uncertainties surrounding inter-species equivalence in susceptibilities to toxic effects.

Inhaled PM appears to generate adverse effects by inciting an inflammatory response. Data from inhalation experiments in rats suggest that an inflammatory response occurs when the lung is exposed even to dusts (titanium dioxide and barium sulphate) considered to be of relatively low toxicity in *in vitro* models (Cullen et al., 2000).

The results of several studies involving cellular assays suggest that the toxicity of urban PM_{10} is greater than that of titanium dioxide. The results of cellular assays reported by Seaton et al. (2005) suggest that the high dose toxicity of PM_{10} is intermediate between that of titanium dioxide and quartz. In experiments with mouse cell lines, Chauhan et al. (2004) established that urban particles decreased nitric oxide (NO) production whereas $PM_{2.5}$ or cristobalite (crystalline silica with a toxic potential similar to or greater than that of quartz) increased the production of nitric oxide. Titanium dioxide had no effects on NO production. However, the consequences of altered pulmonary NO production in this context remain unclear, as NO synthesis potentially results in a range of both protective and deleterious effects on lung tissue (Ricciardolo, 2003; Ricciardolo et al., 2004). The pro-inflammatory effects of NO synthesis noted *in vitro* are not consistently reflected by substantial cytotoxicity *in vivo*. For example, NO is released in massive quantities in asthmatic airways without obviously causing cell injury. Diminished NO synthesis appears to lower bacterial clearance, potentially predisposing to infection.

Particle size is an important determinant of toxic effects. Until recently, nearly all toxicological studies focused on fine and ultrafine fractions of particulates, as these are considered by toxicologists to be most important in mediating the adverse effects of PM

(Donaldson and MacNee, 2001). In one experiment, the effects of ultrafine (diameter 20 nm) and fine (diameter 250 nm) particles were compared when inhaled at similar mass quantities into the lower respiratory tract of rats (Oberdörster et al., 1994). The ultrafine particles elicited a greater acute and persistent inflammatory response. Burden of particle surface area, which is intimately related to particle size, appeared to correlate with toxicity (Tran et al., 2000).

There has been extensive study of the various chemical constituents of PM and their relationship to toxicity of particles, including organic compounds and inorganic components such as acids and transition metals.

Methanol extraction of diesel exhaust particles (DEP) was found to reduce markedly the mortality of healthy mice exposed to DEP, suggesting that organic compounds accounted for much of the toxicity (Sagai et al., 1993). The importance of free radical production in the toxicity of PM was highlighted in this study. DEP were shown to produce the free radicals •OH and $\bullet O_2^-$ *in vitro* in the absence of biological activating systems. Instilled DEP markedly reduced the activities of superoxide dismutase (SOD), glutathione peroxidase and glutathione S-transferase in mouse lungs. Additionally, lung injury and death were greatly diminished by pre-treatment of DEP with polyethylene glycol-conjugated SOD.

Although there has been a considerable quantity of epidemiological research published on the toxic effects of acid aerosols (Dockery et al., 1996; Dockery et al., 1992; Ito et al., 1993; Raizenne et al., 1996; Spengler et al., 1996), any potential toxic effects of PM specifically attributable to their acidity appear not to have been studied directly in experimental models. When administered to the airways in high concentrations, sulphuric acid aerosol caused acute lung injury and resulted in complex time- and dose-dependent ventilatory responses in guinea pigs (Roth et al., 1998). When rats were subjected to inhalation of sulphuric acid concomitantly with ozone, ultrafine (mass median diameter 60 nm) acid droplets but not fine (mass median diameter 300 nm) acid droplets, increased the percentage of severe lung injury in comparison with inhalation of ozone alone (Kimmel et al., 1997). The relevance of these data to human exposure to PM is highly uncertain, given the relatively low level of exposure in community settings and the likely neutralization of acidity in PM by compounds such as ammonia in

ambient air and within the respiratory tract (Dreher, 2000). Chronic (90 days) exposure of rats to sulphuric acid aerosol at a concentration of 20-140 parts per million resulted in no consistent effects on respiratory tract morphology (Last and Pinkerton, 1997).

PM from combustion sources contains bioavailable transition metals, and it is hypothesized that redox activity of these metals on the surface of PM may account for some of the observed toxicity. Iron is the most abundant metal in urban PM_{10}

Costa and Dreher (1997) administered PM samples from several ambient urban and combustion emission sources to rats by intratracheal instillation. The samples were characterized for constituents, including transition metal content. The effects of equimass or equimetal doses of PM were compared, to address directly the importance of metal content in the pathogenesis of lung inflammation and injury. Their results suggested that the lung dose of bioavailable transition metals was the primary determinant of lung toxicity. The potential importance of iron in stimulating an inflammatory response in cellular test systems has been established by several investigators. However in many studies, residual oil fly ash (ROFA) has been used as a source of bioavailable metals, and whereas the transition metal content of ROFA is very high, in ambient air these metals are found mainly in submicron sized particles.

There have been a number of *in vitro* studies assessing the cellular effects of transition metals in PM by comparing the effects of PM with and without prior treatment of samples with the chelator desferrioxamine (DFO). Pre-treatment with DFO has been shown to inhibit residual oil fly ash PM induced production of IL-6 by human epithelial BEAS-2B cells (Quay et al., 1998), production of IL-6, IL-8 and TNFα by normal human bronchial epithelial cells (Carter et al., 1997), and reactive oxygen species (ROS) by normal hamster alveolar macrophages (Goldsmith et al., 1998). Similarly, DFO was shown to inhibit induction of IL-8 in BEAS-2B cells (Ghio et al., 1999), and of ROS in alveolar macrophages (Goldsmith et al., 1998) by samples of ambient urban PM_{10} or TSP.

Hutchison et al. (2005) investigated the toxic potential of PM_{10} collected in the vicinity of a UK steel plant prior, during and subsequent to a period of shutdown for refurbishment of its blast furnace. Biological activity was assessed through intratracheal

instillation of PM_{10} samples into rats and assessment of the cellular and biochemical profile of the bronchioalveolar lavage fluid. The total metal content of PM_{10} collected before and during the closure period was similar, but on reopening there was a significant 3-fold increase compared with earlier samples. Iron was most abundant in the total sample and acid extract, while zinc was the most prevalent metal in the water-soluble fraction. Extracts of PM_{10} from the pre-closure and closure periods did not induce any significant alterations in inflammation or lung damage. Both soluble and insoluble extractable PM_{10} components from the re-opened period induced an inflammatory response as demonstrated by a significant increase in neutrophil cell number when compared to the control. In a cellular assay, PM_{10} from the re-opened period stimulated J774 macrophages to generate TNF-α, a pro-inflammatory cytokine. Chelation of the metal content of the PM_{10} prior to addition to the cells inhibited this reaction, confirming the importance of metals in mediating the inflammatory response.

In a review of experimental data derived for human lung epithelial cells, Aust et al. (2002) concluded that there was evidence to link the transition metal content, and particularly the iron content, of test dusts including urban $PM_{2.5}$ to toxic potential in cellular assays. In a study of the release of the pro-inflammatory cytokines, interleukin-6 (IL-6) and interleukin-8 (IL-8), from human epithelial lung cells induced by different stone dusts, Hetland et al. (2000) found that the samples with a relatively high content of transition metals, such as iron, had the greatest potency. Gilmour et al. (1996) determined that iron plays an important role in the potential of PM_{10} particles to generate hydroxyl radicals in aqueous solution. Hydroxyl radicals are extremely damaging to cells and could play a part in the pathogenicity of PM_{10} particles. Subsequently, Donaldson et al. (1997) demonstrated that the PM_{10} samples contained large amounts of iron that could be readily released as Fe(III) with lesser amounts of Fe(II), on leaching. When samples were centrifuged, the component of PM_{10} that was responsible for hydroxyl radical injury was found to be in the supernatant, suggesting that it was either soluble or contained within extremely fine particles.

Toxicological studies of PM samples collected during the period 1986-1988 in the Utah Valley, USA warrant specific discussion. Much of the ambient PM in this location can be accounted for by the operations of a steel mill. Closure due to a labour dispute during 1987 and subsequent reopening of the mill provided a unique opportunity to examine,

epidemiologically and toxicologically, the effects of PM emissions from the mill (Ghio, 2004). Aqueous extracts from PM_{10} sampling filters were prepared in equivalent masses for samples obtained in the year before, during and following mill closure. Dust from Years 1 and 3 contained higher concentrations of transition metals including iron, copper and zinc and, when added to cultures of human respiratory epithelial BEAS-2B cells, caused greater generation of oxidants than the Year 2 sample, and unlike the latter dust sample, resulted in induction of IL-6 and IL-8 (Frampton et al., 1999). Similarly, injury to cultured rodent airway epithelial cells, based on biochemical and light/electron microscopic changes, followed exposure to extracts from Years 1 and 3 but not Year 2 (Pagan et al., 2003). Greatest inhibition of human alveolar macrophage (AM) phagocytic function and most pronounced apoptosis of AM was effected with Year 1 samples (Soukup et al., 2000). These effects on AM were diminished by pre-treatment with the cation-chelating resin Chelex-100. Experiments *in vivo* have accompanied the foregoing findings. Equivalent-mass aqueous extracts of PM_{10} were intratracheally instilled into Sprague-Dawley rats. Rats given Year 1 and 3 extracts developed significant pulmonary neutrophilic inflammation, lung injury and increased airway responsiveness to acetylcholine compared with rats instilled with either Year 2 extract or saline control (Dye et al., 2001). 500 μg extracts from one of the three years were instilled bronchoscopically into the lungs of non-smoking human volunteers, and the instilled lung subsegment was lavaged after 24 hours. A greater inflammatory response was seen with extracts from Years 1 and 3 (Ghio and Devlin, 2001).

Under the European Union 5th Framework Programme, PM samples collected in cities with contrasting PM sources have been used for a variety of toxicological investigations, the results of which have been reviewed (Sandstrom et al., 2005). The HEPMEAP (health effects of particles from motor engine exhaust and ambient air pollution) project found that $PM_{10\text{-}2.5}$ and $PM_{2.5\text{-}0.1}$ samples collected at different locations throughout Europe varied considerably in their composition and biological activity (Pozzi et al., 2003), and regional differences in activity profiles partly related to traffic patterns (Sandstrom et al., 2005). Coarse and fine PM appeared to have similar toxic activities *in vitro* and *in vivo* on a per mass unit basis, highlighting the need to consider the toxicity of coarse as well as fine PM in risk assessment and when setting standards. The cytotoxicity of the coarse and fine PM was mainly due to oxidative stress resulting from the metal content of the PM (Mudway et al., 2004).

Controlled chamber exposure studies performed as part of the HEPMEAP project showed that asthmatic subjects were particularly sensitive to the effects of diesel engine exhaust particles, demonstrating clinically relevant increases in airway hyperresponsiveness (Nordenhall et al., 2001). Acute airway inflammatory responses to DEP, as assessed from induced sputum, BAL and bronchial biopsy samples, also differed in asthmatic compared with healthy subjects (Nordenhall et al., 2001). However, even non-allergic subjects demonstrated a Th-2 type immunological response in their airways on exposure to DEP (Pourazar et al., 2004).

In the RAIAP (respiratory allergy and inflammation due to ambient particles) project, PM_{10} and $PM_{2.5}$ were collected in Amsterdam, Lodz, Rome and Oslo during winter, spring and summer (Sandstrom et al., 2005). There were distinct differences in chemical composition between the fine and coarse fractions collected at the various locations. All ambient PM fractions showed allergy-enhancing (adjuvant) activity as assessed by cellular response in the lymph nodes and antigen-specific IgE antibody response in the serum of mice (Nygaard et al., 2005; Dybing et al., 2004). Site-specific and seasonal variations were observed in the potential of the PM fractions to induce cytokine release from different types of rat and human lung cells. Bacterial components of the particles did not appear to contribute to the observed effects, whereas the content of both crustal- and combustion-derived metals, was positively correlated with cytokine release (Sandstrom et al., 2005). In mice co-exposed to ovalbumin and PM there were increases in IgE and IgG1 as well as increased numbers of eosinophils and neutrophils in BAL. The adjuvant activity of the coarse and fine PM was ranked as Lodz > Rome = Amsterdam > Oslo, indicating that PM from Lodz had greater pro-inflammatory potential (Steerenberg et al., 2004).

The PAMCHAR project (chemical and biological characterisation of ambient air coarse, fine and ultrafine particles for human health risk assessment in Europe, www.pamchar.org) has the specific objectives of i) characterising the compositions of ambient air coarse, fine and ultrafine particulate fractions in contrasting pollution scenarios in Europe, ii) analysing associations between the physicochemical characteristics of PM_{10} subfractions and cytotoxic, pro-inflammatory and genotoxic effects on human and murine respiratory cells *in vitro*, and iii) testing significant *in*

vitro cytotoxic and pro-inflammatory effects in animal models, and assessing the relevance to human epidemiological data. The PAMCHAR project is currently in progress and will soon be providing important new information in this area (Sandstrom et al., 2005).

a) Toxicological studies of coarse PM

Recently, there have also been published toxicological analyses directly assessing the effects of the coarse fraction ($PM_{10-2.5}$). Many of the studies have combined comparison of the differential effects of the various size fractions with assessment of the specific constituents of PM responsible for their toxicological effects.

It appears from *in vitro* experiments using the human alveolar cell line A549 and primary rat type 2 cells exposed to size-fractionated ambient urban PM that $PM_{10-2.5}$ is equally effective or more effective than $PM_{2.5-0.1}$ or $PM_{0.1}$, on an equal mass basis, at inducing the releasing of the cytokines IL-6, IL-8 and macrophage inflammatory protein (MIP)-2, and inducing apoptosis. (Hetland et al., 2004).

An Italian study compared the *in vitro* toxicity of $PM_{2.5}$ and $PM_{10-2.5}$ collected from an urban area of Rome (Diociaiuti et al., 2001). Haemolytic potential of PM was comparable for similar quantities of the two fractions measured in terms of surface area per unit volume, suggesting that the cytotoxic oxidative stress induced by PM is mediated by interaction between the particle surfaces and cell membranes. However, the viability of cells from the murine RAW 264.7 macrophage line was reduced and their release of NO increased more effectively by $PM_{2.5}$ compared with the effects of coarse particles with a similar total surface area. The disparity in magnitude of effect induced by the two size fractions implies that specific physicochemical properties over and above surface area account for the difference in toxicity. The authors noted that the main obvious chemical difference between the $PM_{2.5}$ and $PM_{10-2.5}$ particle fraction was the relative abundance of carbon-rich particles with sulphur traces in the $PM_{2.5}$ fraction, and speculated that these particles may have accounted for the difference.

A subsequent study by this group, again using the murine macrophage RAW 264.7 cell line, assessed the ability of $PM_{10-2.5}$, $PM_{2.5}$ and carbon black, collected in urban Rome, to cause cell injury as measured by production of arachidonic acid (AA), tumour

necrosis factor alpha (TNFα) and IL-6 (Pozzi et al., 2003). Tested at the same mass concentrations, both urban PM fractions were more effective at inducing AA, TNFα, and IL-6 than was carbon black. It was concluded that the pro-inflammatory effects of PM must be at least partly accounted for by the chemical properties of contaminants adsorbed onto the particles.

Monn and Becker (1999) collected urban PM_{10}, size-fractionated using Andersen dichotomous samplers, in order to compare the cytotoxicity and pro-inflammatory properties of $PM_{10-2.5}$ and $PM_{2.5}$. The activity of water-soluble extracts of the PM was assessed *in vitro*. Cell death and cytokine (IL-6 and IL-8) production were induced in human monocytes by the extract from outdoor $PM_{10-2.5}$ but not by that from $PM_{2.5}$. Extract-induced cytotoxicity, but not cytokine production, was inhibited by DFO. Cytokine induction by $PM_{10-2.5}$ extract was completely inhibited by lipopolysaccharide (LPS) binding protein (LBP). It was concluded that bacterial endotoxin may account for the pro-inflammatory effects of $PM_{10-2.5}$, and that transition metals induce cytotoxic inflammatory pathways independent of macrophage cytokines.

The role of endotoxin in cytokine responses to particles has been further explored. Based on a hypothesis that bacteria in coarse PM as well as particle-bound LPS may be responsible for activation of macrophages by coarse PM, Becker et al. (2002) proposed that PM contaminated by bacteria is likely to activate receptors involved in macrophage recognition. Accordingly, experimental contamination of model pollution particles with environmental *Staphylococcus*, *Streptococcus* and *Pseudomonas* species was shown to confer cytokine-inducing activity on otherwise inactive particles. Cytokine responses to PM were inhibited by antibodies directed against CD14 [a high-affinity receptor for the complex of LPS and LBP (Triantafilou and Triantafilou, 2002)], and required the presence of LBP in serum. Furthermore, the involvement of Toll-like receptors (TLR) 2 and 4 was investigated in transfected Chinese hamster ovary cells expressing these molecules. TLR2 and TLR4 activation were both induced by $PM_{10-2.5}$. Taken together, Becker's results implicate microbial products as fundamentally important in alveolar macrophage-dependent inflammatory responses to PM.

These data are supported by evidence from Huang et al. (2002) in Taiwan. Cells from the murine monocyte-macrophage cell line RAW 264.7 were exposed to size-

fractionated PM samples from various sources. Coarse particles stimulated higher TNFα production than did fine particles. Furthermore, coarse particles had higher endotoxin content, and the TNFα production by both fractions of PM was considerably inhibited by polymyxin B.

Given that endotoxin is critical to the *in vitro* and *in vivo* pro-inflammatory effects and cytotoxicity of coarse particulates, it could be anticipated that in the context of PM in which endotoxin content is likely to be negligible, coarse PM would have limited inflammatory effects, and that smaller particulates would have comparatively greater effects in view of their markedly disproportionate surface area for a given mass. A study has been conducted in which fractionated particles ($PM_{0.2}$, $PM_{2.5-0.2}$ and $PM_{>2.5}$) were sampled from fly ash combustion products of pulverized coal (Gilmour et al., 2004). Saline and endotoxin were used as negative and positive controls respectively. Compared on an equal mass basis, the $PM_{0.2}$ induced the greatest degree of neutrophilic inflammation and cytokine levels in BAL.

However, the effects of coarse PM on macrophage function appear not to be exclusively mediated by endotoxin. In a study conducted in The Netherlands, the ability of fine and coarse PM to suppress macrophage free radical production was compared (Kleinman et al., 2003). Size-fractionated PM was collected from two sites in urban Utrecht with high exposure to vehicles and industry. Murine alveolar macrophages were incubated with the PM and dose-dependent production of superoxide radicals was measured by the chemiluminescent method. Diminution of free radical production by PM was dose-dependent for both coarse and fine particles. Coarse particles were significantly more active than fine particles in suppressing free radical production. Endotoxin was not directly detectable in the particle extracts. Bioavailable iron concentrations were similar in all the PM samples. The chemical basis for the differential biological activity could not be established.

A companion study using the Utrecht samples was performed to elucidate further the nature of the response of alveolar macrophages to PM (Becker et al., 2003). The effects of coarse, fine and ultrafine PM on human alveolar macrophages were compared. Coarse particles were substantially more active than fine particles, which in turn were more active than the ultrafine fraction in induction of IL-6. Cytokine induction by the

PM was inhibited by antibody to CD14 and required the presence of serum for optimal stimulation, suggesting that bacterial products such as endotoxin were implicated in the stimulatory activity of the PM. Phagocytic activity and the oxidative burst were inhibited more by the coarse fraction than by fine PM. The UF fraction did not influence these functions.

A study in which the effects of $PM_{10-2.5}$ and $PM_{2.5-0.1}$ from four European cities (Amsterdam, Lodz, Oslo and Rome) were examined has further highlighted the notion that the pro-inflammatory effects of coarse PM cannot be entirely attributed to the endotoxin content of particles (Hetland et al., 2005). In this study, the cytokine-inducing potential of PM of both fractions from each of the four cities sampled in each of three seasons (spring, winter and summer) was assessed on macrophages retrieved from rat lungs. On an equal mass basis, the coarse PM fraction from each city was more potent in inducing TNFα and IL-6 than the respective fine fraction. Coarse fractions contained higher levels of specific constituents including the transition metals, iron and copper, endotoxin, and some PAHs. However, differences in the content of these components did not appear to influence the variation in pro-inflammatory potency of coarse PM, nor did the addition of polymyxin B affect cytokine induction.

Given the presence of transition metals such as iron, chromium, vanadium, and copper in PM, both coarse and fine particles are able to generate the hydroxyl radical (•OH) via the Fenton reaction, and induce the •OH-specific DNA adduct 8-hydroxy-2′-deoxyguanosine (Shi et al., 2003).

Huang et al. (2003) extended their work on the cellular effects of PM. RAW 264.7 macrophages and human bronchial epithelial BEAS-2B cells were exposed to particles sampled from a variety of settings, fractionated trichotomously (as $PM_{0.1}$, $PM_{2.5-1}$ and $PM_{10-2.5}$), and extensively characterized chemically. Particle-stimulated cytokine (IL-8) production and lipid peroxidation by epithelial cells was size-dependent, being statistically significant only for the $PM_{0.1}$ fraction. TNFα production in RAW 264.7 cells was likewise only significantly greater than controls in cells exposed to the sub-micrometer range PM. Production of TNFα was substantially inhibited by pre-treatment with polymyxin B, indicating the importance of endotoxin in the cytokine response of macrophages to PM. Cytokine production was associated with the metal content of

$PM_{0.1}$. Lipid peroxidation by BEAS-2B cells correlated with elemental and organic carbon content.

The comparative *in vitro* pro-inflammatory effects of $PM_{10-2.5}$ and $PM_{2.5}$ were further replicated by Schins et al. (2004), who also assessed the comparative *in vivo* effects of particles instilled into the respiratory tract of rats. Dichotomously fractionated PM was sampled from both a rural and an urban location, providing four different PM samples. Coarse PM but not fine PM from both sources caused neutrophilic inflammation in rat lungs. Additionally, the rural sample of coarse PM increased TNFα and depleted glutathione in bronchoalveolar lavage fluid. The inflammatory effects of the PM samples were associated with their endotoxin content.

The importance of solvent-extractable organic compounds (SEOC) in the toxicity of PM was underscored in a study by Hsaio and colleagues (2000). They compared coarse and fine PM in terms of the cytotoxicity attributable to SEOC from the collected PM, using the MTT cell proliferation assay and the Comet assay for DNA damage on a well-established mammalian cell line, the Rat 6 rodent fibroblast. The cell damage from the $PM_{2.5}$ extracts greatly exceeded that from the $PM_{10-2.5}$, suggesting that toxic SEOC were essentially confined to the fine particles.

The epidemiological evidence that certain types of PM influence the development of allergy (D'Amato et al., 2001) is supported by *in vitro* studies showing that particles contain materials with adjuvant activity (Becker and Soukup, 2003). Human blood-derived monocytes upregulated the expression of several immune co-stimulatory receptors (HLA-DR, CD40, CD80 and CD86), in response to all of three tested particle fractions ($PM_{0.1}$, $PM_{2.5-0.1}$ and $PM_{10-2.5}$). Human alveolar macrophages produced the T-helper cell chemoattractant IL-16 only in response to $PM_{10-2.5}$.

b) Summary

Recently published toxicological data demonstrate effects of the coarse fraction of PM, as well as the fine and ultrafine fractions. This is consistent with current epidemiological evidence.

There are several pathways involved in the cytotoxic and cell-activating effects of PM. Organic compounds, transition metals and bacterial components such as lipopolysaccharide endotoxin appear to be influential, as does particle surface area *per se*. Some but not all of the cellular effects of $PM_{10-2.5}$ appear to be mediated by endotoxin content.

SECTION 7. FORMULATION OF DUST EXPOSURE GUIDELINES FOR PORT HEDLAND

Summary

- **The physicochemical profile of airborne PM in Port Hedland is unique. It has not been possible to base recommendations for acceptable levels of dust exposure on scientific data directly derived from this setting, as the relevant air quality data and epidemiological and toxicological studies are not available.**
- **The recommendations for acceptable levels of dust exposure in Port Hedland have therefore been based on scientific data from other, broadly comparable settings.**
- **Inhalation of iron and manganese ores is the principal health concern arising from exposure to airborne PM in Port Hedland. Epidemiological data from appropriate occupational settings and the London Underground are pertinent for comparison in this regard.**
- **The extensive literature on health effects of exposure to airborne PM in general has been reviewed at length, given the known toxicity of PM and possible implications for the population of Port Hedland.**
- **Recommendations are made for data collection and additional studies that would help to inform future decisions on an Air Quality Standard for Port Hedland.**
- **An interim guideline is suggested whereby 24 h average $PM_{10-2.5}$ mass concentrations would not be allowed to exceed 70 $\mu g/m^3$, with an annual average limit for manganese of 0.15 $\mu g/m^3$ (as PM_{10}).**

7.1 Introduction – the conceptual basis for recommendations

The key questions this literature review has set out to address are: what are acceptable levels of airborne particulate matter in the Port Hedland township, and should these be measured as average and/or peak levels of particulate matter? In addition, it is necessary to consider what methods should be used to measure particulate matter and

what type of monitoring is appropriate to provide information on potential health effects.

There are no data directly addressing the hazards to health associated with exposure to dusts containing the specific range of constituents present in Port Hedland dust, which is essentially unique in its composition. Definitive recommendations for acceptable levels of dust exposure in this setting are therefore not feasible. The scientific argument on which our recommendations are based is necessarily based on data from broadly comparable (although by no means identical) dust exposures in other settings. A principal concern is the presence in Port Hedland dust of metals, particularly iron oxides, providing a clear analogy with data from occupational settings such as haematite mining and welding, as well as the London Underground. As discussed in detail (Section 6), particle size, which is related to both the composition and respirability of particles, is a fundamental determinant of the health effects of dust.

The composition of the aerosol found in cities is very different from that in Port Hedland; in urban environments, very fine combustion particles from motor vehicles and power plants predominate whereas the township air comprises mainly mineral matter. The Ambient Air Quality National Environmental Protection Measure (AAQ-NEPM) specifies an air quality standard for PM_{10} of 50 $\mu g/m^3$, averaged over 24 hours, with the goal that there should be no more than five exceedences of this value at a specific location each year. The $PM_{2.5}$ reporting standard is 25 $\mu g/m^3$ as a 24-hour average and 8 $\mu g/m^3$ as an annual average. However, there is no reason to expect that the potential adverse health effects of the pollution from very different sources would be similar, and so there is no justification for the AAQ-NEPM, which has been developed for urban environments, to be uncritically applied to Port Hedland. We have therefore looked for a scientific argument that can be used to support the derivation of an interim standard for Port Hedland that could be applied until such time as appropriate monitoring and exposure studies have been performed.

7.2 Appropriate sampling and analysis methods

As noted in Section 5.3, no existing device is ideal for the measurement of particulate matter. The samplers normally used to measure air pollution in cities may not work reliably in the high winds and temperatures found in Port Hedland. Therefore previous

measurements of PM_{10} and $PM_{2.5}$ made at various locations around Port Hedland, including those made at Boodarie, may not reliably indicate the true airborne dust concentrations. The authors have previously used a high-volume elutriator sampler to measure respirable particles (approximately $PM_{3.5}$). However, the elutriator sampler is likely to be more influenced by wind direction than the widely used PM_{10} samplers with symmetrical openings, which are not unduly affected by wind speed and direction (M. Lippmann, personal communication). There are many sampling methods that could be used in the windy, dry, hot conditions usually prevalent in Port Hedland, as measurements of ambient PM have been successfully made in the Gobi Desert and in Death Valley, California. The publications of Chow (1995) and Watson & Chow (2001) should provide an indication of the sampling devices that are most likely to provide reliable ambient air quality data from the Port Hedland environment. Furthermore, coarse dusts predominate and are most likely to cause irritation of the tracheobronchial airways in the Port Hedland population, and it is therefore measurements of PM_{10} that will be most useful in assessing potential health risks. However, since elements such as Fe and Mn are also likely to be important, it would seem prudent to measure not only the mass of PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ (by difference) fractions, but also to try and obtain reliable data on the composition of these size fractions, including sulphate, nitrate, ammonia, sodium chloride, silica, organic and elemental carbon, as well as transition metals.

7.3 Comparison of the effect of Port Hedland dust with that of other mineral dusts

In the absence of scientific data that provides a basis for setting regulatory standards for dust exposure in Port Hedland, recommendations must either be based on an arbitrary level of improvement from existing conditions, or derived by extrapolation from guidelines developed in other contexts. Clearly, the validity of extrapolation from occupational settings is limited by the disparity in exposure conditions between the relatively time-limited high level dust exposures experienced in occupational settings and the chronic (potentially life-long) low level dust exposures experienced by the general population in a community such as Port Hedland.

Port Hedland dust is predominantly composed of iron oxides, clays, sea salt, and gypsum with smaller quantities of crystalline silica and combustion-generated

particulate matter. At the very high concentrations previously observed in industrial exposure to haematite, some workers will, after many years, suffer from pneumoconiosis, probably because of the quartz in the dust. These workers were probably exposed to concentrations in excess of 1,000 μg/m^3 of respirable dust, but the evidence is quite limited (Faulds, 1957)

There is a wider base of toxicological and epidemiological information available for other dusts. Overall the composition of airborne dust in Port Hedland might be fairly similar to that of dust from the London Underground. The $PM_{2.5}$ fraction of London Underground dust comprises 64-71% iron oxide by mass with 1-2 % quartz (Seaton et al., 2005). The toxicity of Port Hedland dust might also be reasonably considered to be similar to that of coalmine dust, iron-rich welding fumes or silica. Coalmine dust contains small quantities of crystalline silica, displays a relatively low level of toxicity, but is considerably more toxic than materials such as titanium dioxide. We have briefly reviewed the evidence from these potentially analogous exposures to determine whether these data can assist in setting an air quality standard for Port Hedland.

In addition, there has been considerable debate about the relative importance of the fine fraction of PM_{10} (i.e. $PM_{2.5}$) versus the coarse fraction, i.e. the difference between PM_{10} and $PM_{2.5}$ ($PM_{10-2.5}$), in giving rise to observed adverse effects. In most urban situations the $PM_{2.5}$ fraction is dominated by combustion-generated particles, whereas the $PM_{10-2.5}$ fraction is dominated by other dusts such as wind-blown mineral dust. The airborne particulate matter in Port Hedland might reasonably be expected to have a greater proportion of mineral dust particles than more typical urban PM_{10}. For this reason we have looked at the epidemiological evidence for effects from the coarse fraction of particulate matter, to assess its relevance to Port Hedland.

Limited toxicity studies have suggested that test aerosols containing iron oxide concentrations at least 100 times greater than those of PM_{10} in Port Hedland are not significantly toxic to the lungs. In the study on the toxicity of London Underground dust (Seaton et al., 2005), the concentrations necessary to detect effects *in vitro* were tens of thousands times greater than those likely to make contact with the relevant pulmonary cells *in vivo*. However, these data are not entirely reassuring, as exposure in Port Hedland would continue for 24 hours per day, seven days per week. In addition, the

exposed population would include individuals with pre-existing respiratory illness who would be potentially more susceptible to the effects of low levels of exposure.

Iron-rich welding fumes may provide a reasonable analogy for PM_{10} in Port Hedland air, and some studies have provided evidence to suggest that inhalation of an iron oxide-rich fume aerosol could give rise to an inflammatory response in the lung. However, such fumes are likely to reach the alveolar space and small airways, while only a very small proportion of the PM_{10} fraction would reach beyond the large airways. There is no WHO Air Quality guideline for iron oxide, but the Australian National Occupational Health and Safety Commission has set an occupational exposure standard for iron oxide fumes of 5,000 $\mu g/m^3$ (measures as iron) as the average airborne concentration calculated over a normal 8-h working day, for a 5-day working week. In Germany, the maximum allowable concentration for occupational exposure to iron oxide is 1,500 $\mu g/m^3$ in the respirable fraction. However, the extrapolation of occupational exposure to welding fumes to ambient dust exposure in Port Hedland is tenuous in the absence of information about the dose-response curve, the site of airway exposure and the effects of other hazardous substances in welding fume, including ozone, oxides of nitrogen, acids and metals such as manganese.

Coal mining may provide the best analogy of community risk from exposure to mineral dust emanating from a point source. In addition, the limited experimental information about the toxicity of particle mixtures dominated by iron oxide rich aerosols suggests that the toxicity of ambient particles in Port Hedland air may be similar to that of coal mine dust. It therefore seemed reasonable to assess the use of exposure-response information from the coal industry as a basis for setting an environmental standard for airborne particles.

Exposure-response information obtained by Soutar et al. (2004) for the British coal industry suggest that 35-40 years of workplace exposure to a respirable dust concentration of 1,500 $\mu g/m^3$ is associated with a 1.5% risk of developing pneumoconiosis (category 2 or greater) and a 0.8% risk of an attack of pulmonary massive fibrosis. Adjusting for the differences in duration of exposure (24 hours x 365 days x 70 years for lifetime environmental exposure and 40 hours x 40 weeks x 40 years for workplace exposure), the equivalent concentration for lifetime exposure in ambient air would be about 150 $\mu g/m^3$ as respirable dust. The equivalent concentration of PM_{10}

would be higher, perhaps 300 μg/m^3 (reflecting the use of a factor of 2 by WHO to interconvert between $PM_{2.5}$ and PM_{10} in their deliberations on particulate matter in urban air).

The epidemiological data suggest that the threshold exposure concentration of respirable dust in the workplace for effects on coal miners' health is less than 1,000 μg/m^3, if a threshold exists. The equivalent concentration for exposure to respirable dust in ambient air would be about 100 μg/m^3. The equivalent concentration of PM_{10} might be about 200 μg/m^3.

Although coal mine dust might reasonably be considered as an analogue for dust particles in Port Hedland air, this analogy does not stand up to close scrutiny, since the scientific evidence is that the pathological effects of air pollution particles differ fundamentally from those of occupational exposures to mineral dusts. Whereas long-term exposure to mineral dust is associated with development of pneumoconiosis and sometimes emphysema, epidemiological studies of the effects of urban and semi-rural air pollution particles consistently show short-term cardio-respiratory effects. In addition long-term exposure to air pollution is associated with risks of cardiac disease, which has never been recognised as a risk from occupational mineral dust exposure. Furthermore, it would be expected that there would be fewer ill individuals working in coal mines and susceptibility to adverse respiratory effects may therefore be less than that of the general population. Individuals in the general population with pre-existing respiratory illness may experience adverse effects at lower levels of exposure. Moreover young children whose lungs are not fully developed might be expected to be more sensitive to inhalation of mineral dusts.

The ambient dust impacting Port Hedland, which is a mixture of iron ore dust and naturally occurring dust, is known to contain silica. The current occupational exposure limit for respirable crystalline silica is 100 μg/m^3, although there have been recommendations that the limit of exposure, below which the risk of silicosis is considered negligible, should be lowered to 50 or 40 μg/m^3 (WHO, 1986; Rosenman et al., 1996; Finkelstein, 2000). More recently the Californian Office of Environmental Health Hazard Assessment has proposed a reference exposure limit (REL) of 3 μg/m^3 for chronic community exposure to respirable crystalline silica (California OEHHA, 2005). The derivation of this REL is supported by long-term studies of the incidence of

silicosis following occupational silica exposure, and the finding of a dose-response effect for silicosis in several of these studies. Furthermore, the no observed adverse effect levels (NOAEL) varied widely from 7 to 100 μg/m^3 (Rice & Stayner, 1995). However, there are many areas of uncertainty associated with the development of a REL for community exposure to silica. These include the limited follow-up in some studies, underestimation of the incidence of silicosis, variability in the toxicity of different forms of silica, limited information on silica particle size, variability in particle penetration and deposition in the respiratory tract, and the use of area samplers rather than personal samplers to estimate exposure. These uncertainties complicate the use of the proposed REL of 3 μg/m^3 for silica to inform decisions about the levels of community exposure to silica that would be acceptable in Port Hedland. Furthermore, there is currently insufficient data on the concentrations of silica present in the coarse particle fraction of ambient dust impacting Port Hedland to allow an appropriate recommendation to be made.

7.4 Epidemiological evidence of effects of coarse particles on health

Following a review of the then available data, the UK Expert Panel on Air Quality Standards (Expert Panel on Air Quality Standards, 2001a) concluded that for urban aerosols there was some evidence to suggest that the $PM_{2.5}$ fraction is more harmful than the coarse component of PM_{10}, although the data were not conclusive. In a more recent review, Brunekreef & Forsberg (2005) concluded that although there was some evidence linking the coarse fraction of PM_{10} to fluctuations in daily mortality, in most situations stronger effects were seen for fine particles. In contrast to the studies on the long-term effects of $PM_{2.5}$, the limited data on long term exposure to the coarse fraction of PM_{10} (i.e. $PM_{10-2.5}$) do not suggest any association with reduced life expectancy. The results of studies of the effects of short-term fluctuations in PM_{10} on chronic obstructive pulmonary disease, asthma and respiratory hospital admissions, however, suggest that the coarse fraction may have as strong an effect or a stronger effect than $PM_{2.5}$. The review also found limited evidence for an association between coarse particles and cardiovascular hospital admissions. Overall, therefore, the available information suggests that if the particle size characteristics of PM_{10} from Port Hedland are dominated by relatively coarse particles rather than combustion particles, there would be a markedly reduced effect relative to urban PM_{10}, but there may be some adverse

effect at higher exposures in individuals with airway diseases. Brunekreef & Forsberg (2005) noted that in some studies endotoxin levels were much higher in the coarse aerosol fraction compared with the fine fraction. This would be expected, as the coarse fraction contains most of the biological material, fungal spores and pollen grains. However, in Port Hedland this may not have as great an influence given the relatively arid nature of the environment and its effect on surrounding vegetation levels.

The US Environmental Protection Agency (EPA) has recently reviewed the National Ambient Air Quality Standards for Particulate Matter (US EPA, 2005) and has found that there is sufficient evidence to indicate that urban road, construction and industrial coarse dust ($PM_{10-2.5}$) has adverse health effects, but there is insufficient evidence of adverse effects from other sources of $PM_{10-2.5}$ such as agricultural and mining operations. Consistent with this evidence, the US EPA has proposed a new $PM_{10-2.5}$ National Ambient Air Quality Standard limiting 24 h average ambient $PM_{10-2.5}$ concentrations to less than 70 $\mu g/m^3$. However, this would only be applicable in urban areas with populations exceeding 100,000.

Relatively few epidemiological studies have specifically investigated the relationship between metals in ambient air and health effects. Roemer et al. (2000) investigated the relationship between PM_{10} composition and acute respiratory health effects in children with chronic respiratory symptoms. Both silicon and iron concentrations tended to be negatively associated with peak expiratory flow (PEF), and positively associated with the prevalence of phlegm. It was not possible, however, to determine the separate effects of silicon and iron. In a study of 32 adults living near a large steel industry in the Netherlands, Dusseldorp et al. (1995) reported a statistically significant decline in respiratory function (PEF) associated with increasing PM_{10} concentrations. Increased concentrations of iron showed a statistically non-significant association with a decline in PEF, with a lag of 2 to 3 days. In a study of markers of oxidative stress in white blood cells and urine collected from human volunteers with exposure to ambient urban particulate, Sorensen et al. (2005) found both vanadium and chromium in $PM_{2.5}$ to be associated with evidence of DNA damage in lymphocytes and 24-hour urine samples, as assessed from 7-hydro-8-oxo-2'-deoxyguanosine (8-oxodG) concentrations in lymphocytes. Similar effects were not found for iron (or for nickel, copper and platinum).

In conclusion, the results of most toxicological studies of the effects of PM_{10} in cellular test systems have suggested that iron radicals may play an important role in promoting an inflammatory response, probably through contributing to the generation of reactive oxygen species that are highly damaging to tissue. Importantly the results of one study, however, suggest that endotoxin may be much more important than iron in giving rise to the toxic effects of PM_{10}. The limited available epidemiological data do not suggest that iron plays an important role in giving rise to the health effects attributed to PM_{10}.

7.5 Conclusions and derivation of an Air Quality Standard for Port Hedland

The important question that needs to be addressed is what is the potential risk to the population of Port Hedland of the dust to which they are exposed at the concentrations which they have experienced. The dust is predominantly a mineral dust, whose major constituent is iron oxide. There is unfortunately little direct evidence either of the hazardous nature of the dust (i.e. knowledge of toxicity of dust of this composition) or the information needed for risk assessment, in particular reliable measurements of dust exposure at Port Hedland, on which to come to firm conclusions and base recommendations.

Coal dust might be considered an analogous material on which to base standards for the level of airborne dust in Port Hedland. This suggestion is based on the observations that coalmine dust and airborne dust in Port Hedland are mixed dusts that include silicates and small concentrations of crystalline silica, and that the toxicity of coal lies between that of titanium and silica. However, on closer scrutiny it is questionable whether coal dust is an appropriately analogous substance for ambient particulate matter in Port Hedland (A. Newman Taylor, personal communication). Like iron oxide, inhaled coal dust can cause the appearance of small nodules on the chest radiograph, whose profusion reflects the concentration of dust retained in the lungs. However unlike iron oxide dust, in the absence of associated silica, coal dust can also cause fibrosis of the lungs (progressive massive fibrosis), which causes loss of lung function, with associated disability, and reduced life-expectation. The analogy with coal dust may therefore exaggerate the adverse health effects of Port Hedland dust, with consequent inappropriate recommendations for dust control and air quality standards.

The results of studies of aerosols with a high iron oxide content, including iron-rich welding fume and London Underground dust suggest some similarities with PM_{10} from Port Hedland. However the significant differences in particle size and composition of welding fumes and London Underground dust mean that these are unlikely to be useful analogies from which to derive an air quality standard for Port Hedland. The available information indicates that regulation of exposure to Port Hedland dust should be analogous to that for a mixed mineral dust, adjusted to take account of duration of exposure over a lifetime and of the presence in the population of relatively vulnerable young and disabled people. Although silica is present as a component of the mineral dust impacting Port Hedland, it would seem inappropriate given the current uncertainty regarding the concentration and size of silica particles present, to attempt to derive an air quality standard for Port Hedland, based on the proposed REL of 3 $\mu g/m^3$ for community exposure to respirable crystalline silica.

The review by Brunekreef and Forsberg (2005) provides limited evidence that daily fluctuations in the concentration of the coarse fraction of air pollution aerosols may have a comparable effect on morbidity as the fine fraction. In addition the recent review by the US EPA (2005) found sufficient evidence to implicate urban $PM_{10-2.5}$ in adverse health effects. While specifically excluding contributions from mining operations, the EPA Clean Air Science Advisory Committee recommends that $PM_{10-2.5}$ be monitored in both urban and rural communities, and a short-term air quality standard of 70 $\mu g/m^3$ as a 24 h average has been proposed for $PM_{10-2.5}$.

In the absence of informative data, it is difficult to reach a conclusion regarding an Air Quality Standard that would be appropriate for the Port Hedland community. The most important recommendation that can be made at this time is that reliable, valid data should be collected in order to inform future decisions.

Specific recommendations are:

1) The establishment of a network of samplers that are capable of accurately and reliably measuring PM_{10}, $PM_{2.5}$, and by difference, $PM_{10-2.5}$ mass concentrations in ambient air at various locations around the Port Hedland townsite and BHPBIO processing and loading facility at Port Nelson. These monitors should be located both near the iron ore processing operations and in residential areas of Port

Hedland, where community exposure is likely to be greatest. Computer modelling should be used to assess the potential influences of wind direction and speed, daily activities at the iron ore loading facility and seasonal variations on the average daily $PM_{10-2.5}$ concentrations measured at specific monitors.

2) In addition to information on the mass concentrations of the various PM fractions, information should be obtained on the compositions of the PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ size fractions at various locations around Port Hedland. This should include analyses of elements that are of specific interest such as Fe, Cu, Mn, Zn, V, Mg, Al and Si, as well as other components likely to occur in Port Hedland dust, including sulphate, nitrate, ammonia, sodium chloride and organic and elemental carbon. Although combustion-derived carbonaceous PM is likely to be present at lower concentrations than in urban PM, it should not be completely discounted, as there are likely to be significant levels of diesel exhaust emissions from shipping and other vehicles and machinery used at the ore loading facility. It will be important to specify which chemical measurements will be undertaken, as all analyses cannot be performed on the same filter materials. Using a well-designed network (Chow et al., 2002), it should be possible to measure PM concentrations and compositions precisely and accurately, even under the difficult environmental conditions prevalent in Port Hedland (Chow, 1995). These measurements could be made at the fence line of the ore processing and loading facility, as well as in selected residential neighbourhoods, in order to gain information about the composition of the exposure and to determine the relative contributions from different sources, using source apportionment models (Watson et al., 2002; Watson & Chow, 2004).

3) A well-designed network of air sampling monitors, in conjunction with computer modelling techniques that take into account variations in weather patterns etc., should provide useful information on potential exposures to PM among Port Hedland residents. However, in order to obtain valid and representative measurements of the actual PM exposure experienced by the Port Hedland population, some form of personal sampling, in representative groups of the population, will be required. EcoTech of Melbourne manufactures a convenient portable minivol sampler that is well-suited for personal exposure studies (Chow & Watson, personal communication). Ideally, personal exposure studies should be

combined with a prospective study to determine whether there are any associated short-term adverse health outcomes.

4) A retrospective study should be undertaken to determine whether there has been any direct harm to the health of the community residing in Port Hedland (A. Newman Taylor, personal communication). This could take the form of a survey of the respiratory health of the population of Port Hedland, using a questionnaire, lung function tests and chest radiographs. Analysis of the results of such a survey in relation to cumulative dust exposure should provide additional evidence necessary to inform an appropriate Air Quality Standard for Port Hedland.

5) Samples of Port Hedland dust should be tested in *in vitro* cell toxicity studies on human respiratory cells. Such studies would provide information on the relative cytotoxicity of Port Hedland dust in comparison to titanium, coal dust and silica.

It is appreciated that these proposals will require time to implement and that there is a need to provide guidance on a shorter time scale. Nonetheless it would seem prudent to base a future standard for Port Hedland on direct evidence rather than on a questionable analogy such as occupational exposure to coal mine dust. In the meantime a practical approach would be to evaluate the relevance and appropriateness for Port Hedland of community health exposure standards, such as the US EPA National Ambient Air Quality Standards for coarse PM. In the absence of more informative data, and based on the available information that Port Hedland dust is mainly comprised of coarse particles ($PM_{10-2.5}$), we would recommend that the control of air pollution in Port Hedland be achieved by imposing an interim Air Quality Standard whereby the 24-h average $PM_{10-2.5}$ (the difference between co-located PM_{10} and $PM_{2.5}$ mass concentration measurements) would not be allowed to exceed 70 $\mu g/m^3$. In our view, however, a decision on the precise figures is a matter for discussion between the Western Australian authorities and BHPBIO. Because in some circumstances there may be manganese in the aerosol, we also recommend that the annual average manganese concentration should be maintained below the World Health Organisation Air Quality Guideline (WHO, 2000) of 0.15 $\mu g/m^3$ (as PM_{10}) to protect against potential neurotoxicity. No recommendation is made for other metals such as copper, as exposure to these is deemed to be negligible.

Any strategy to protect the health of the community and employees of the company from adverse effects of dust inhalation requires decisions on the numbers and siting of samplers. Such decisions should be made on the basis of a detailed understanding of the processes involved and local geography. We are happy to be involved in guiding this decision making if required.

SECTION 8. REFERENCES

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