



Report

REPORT

HEALTH RISK ASSESSMENT PORT HEDLAND

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GLOSSARY

AAQ NEPM	National Environment Protection (Ambient Air Quality) Measure
Atopy	Positive response to one or more allergens tested
ATSDR	Agency for Toxic Substances and Disease Registry
BAM	Beta Attenuation Monitor
BMC	Benchmark Concentration
CAPS	Childhood Asthma Prevention Study
CO	Carbon monoxide
CO ₂	Carbon dioxide
COPD	Chronic Obstructive Pulmonary Disease
DoH	Western Australian Department of Health
DSD	Department of State Development WA
EPA	Environment Protection Authority
EPHC	Environment Protection and Heritage Council
FCV (litres)	Forced Vital Capacity
FEF _{25-75%}	Average of expired flow over the middle half of FVC
FEV ₁ (litres)	Forced Expiratory Volume in one second
FEV ₁ /VC	FEV ₁ as a percentage of vital capacity or forced vital capacity
FIFO	Fly-in-fly-out worker
HRA	Health Risk Assessment
HRAM	Health Risk Assessment Methodology
ISSAC	International Study of Asthma and Allergies in Children
LALN	Lung associated lymph node
Long-term exposures	Exposures for 1 year to several years
Lifetime exposure	Exposure over a lifetime assumed to be 70 years
LOAEL	Lowest observed adverse effect level
µg/m ³	Micrograms per cubic meter
Mn	Manganese
MMAD	Mass median aerodynamic diameter
MRL	Minimal Risk Level
NEPC	National Environment Protection Council
NO	Nitric oxide
NO ₂	Nitric dioxide
NO _x	Oxides of nitrogen (NO + NO ₂)
NOAEL	No observed adverse effects level
ng/m ³	Nanograms per cubic metre
O ₃	Ozone
OEHHA	Office Environmental Health Hazard Assessment, Californian EPA
OEL	Occupational exposure limit
PAH	Polycyclic aromatic hydrocarbons
PEF	Peak Expiratory Flow
PHIC	Port Hedland Industries Council

PM	Particulate matter
PM ₁₀	Particulate matter less than 10 µm in diameter
PM _{2.5}	Particulate matter less than 2.5 µm in diameter
PM _{2.5-10}	Particulate matter between 2.5 µm and 10 µm in diameter
POD	Point of departure
ppb	Parts per billion
ppm	Parts per million
RCS	Respirable Crystalline Silica
RDDR	Regional Deposition Dose Ratio
REVIHAAP	Review of Evidence of Health Aspect of Air Pollution
RIVM	National Institute Public Health and Environment, The Netherlands
SCCHS	Southern Californian Children's Health Study
SCEW	Standing Council on Environment and Water
SEIFA	Socioeconomic Indices for Areas – index of relative socioeconomic advantage and disadvantage
SES	Socioeconomic Status
Short-term exposure	Exposures of 1-hour to days
SO ₂	Sulfur dioxide
SPFR	standardized peak flow rates
TEOM	Tapered Element Oscillating Microbalance
USEPA	United States Environmental Protection Agency
WHO	World Health Organization

1 EXECUTIVE SUMMARY

A health risk assessment (HRA) has been conducted for Port Hedland to guide future planning and development decisions for the town. The HRA has calculated the risks posed to the residents of Port Hedland and South Hedland from exposure to ambient air pollution including PM₁₀, PM_{2.5}, NO₂, SO₂, respirable crystalline silica, asbestos fibres, manganese, copper and iron oxide. Monitoring data was obtained from monitoring stations operated for Port Hedland Industry Council (PHIC) between 2012 and 2014.

The HRA has combined local health statistics and population data and combined this with ambient monitoring data collected at locations considered to be representative of the exposure of the population including Richardson St (representing residential areas in the west end of Port Hedland), Taplin St (representing residential areas at the interface of the east and west of Port Hedland), Neptune Place (representing residential exposures in the east end of Port Hedland) and Acacia Way (representing residential exposure in South Hedland). Data was also collected at Yule River which is indicative of regional background dust levels experienced across the Pilbara region arising from natural sources.

The monitoring data collected in 2012-2014 at the Port Hedland and South Hedland sites show that with the exception of PM₁₀ and PM_{2.5} all other pollutants meet the air quality standards and guidelines adopted for the HRA. Nitrogen dioxide and sulphur dioxide levels were well below the air quality standards in the National Environment Protection (Ambient Air Quality) Measure (AAQ NEPM). Monitoring for manganese, iron oxide and copper were below the Toxicity Reference Values (TRVs) adopted for the HRA. Monitoring for asbestos found no levels above the limit of detection used in the analysis. The data for respirable crystalline silica (RCS) showed levels below the adopted TRV.

PM_{2.5} levels exceeded the 24-hour NEPM advisory reporting standard on two occasions in the monitoring period at the Richardson St site but were met at the Taplin St and South Hedland locations. For PM₁₀ there are numerous exceedances of the 24-hr PM₁₀ NEPM air quality standard and the interim guideline adopted by the Port Hedland Taskforce of 70 µg/m³. In 2013 four exceedances of the taskforce guideline of 70 µg/m³ at both the Richardson and Taplin St sites compared with 22 and 14 observed at these sites respectively in 2012. In 2013 47 exceedances of the NEPM standard was observed at Richardson St and 33 at Taplin St. The number of exceedances of the standards was lower in South Hedland with two exceedances of the taskforce guideline and nine exceedances of the NEPM standard observed in 2013. Only six months of data was available for 2014. During that period twenty one exceedances of the NEPM standard were observed at both Richardson and Taplin St, with seventeen observed at Neptune Place, eight at South Hedland and three at Yule River. For the same period the taskforce standard was exceeded five times at Richardson St, twice at Taplin St, seven times at Neptune Place, twice at South Hedland and once at Yule River.

All data provided for the HRA was used in the analysis. No data was removed and data for each site was analysed independently. The data was not averaged across the sites. In 2013 peak levels of PM₁₀ reached as high as 400 µg/m³ at the Taplin St site and analysis of the data indicates that these exceedances were not due primarily to regional dust events but to local sources of dust in the Port Hedland area. Analysis of PM₁₀ levels from all monitoring sites showed that on 16 days when exceedances were observed in Port Hedland they were not exceeded at South Hedland or across all sites. If dust from regional sources was the key source of PM₁₀ on these days then it would be expected that elevated levels would be observed at all sites which was not the case. Further investigation is required to identify the key sources and develop dust management plans to reduce PM₁₀ levels in Port Hedland.

Analysis of the PM₁₀ and metals data shows an influence of activities at the Port and associated industrial areas on ambient levels in the west End of Port Hedland with elevated levels of both mean and maximum values at the Richardson St monitoring location which is closest to the Port. Monitoring from the other locations shows a reduction in ambient levels at sites further from the Port. For manganese, the monitored levels at the Richardson St site are close to the TRV adopted for the HRA. This data suggests that management actions should be implemented to ensure that the manganese levels do not reach or exceed the TRV in the future and with increased exports through the Port.

The risk characterisation has shown that the pollutant that is having the greatest impact on public health in both Port Hedland and South Hedland is PM₁₀ with increases in mortality and hospital admissions associated with exposure to PM₁₀ at current levels. The most substantive impact is for hospital admissions for respiratory disease and pneumonia and bronchitis in people over 65 years of age. If the PM₁₀ levels in Port Hedland could be reduced to meet the taskforce guideline of 70 µg/m³ then the risk per 100,000 population in Port Hedland is significantly reduced with the number of hospital admissions for these outcomes reduced to about one third of what is currently being experienced. Similar reductions are observed for increases in mortality from both long-term and short-term exposures if PM₁₀ levels can be reduced. The risks per 100,000 population are higher in Port Hedland than observed in cities such as Sydney, Melbourne and Perth. The PM₁₀ levels in Port Hedland are higher than those observed in these cities.

Further reductions in PM₁₀ levels to meet the NEPM standard of 50 µg/m³ leads to further reductions in risk but this is much less than that observed in reducing the current levels to meet the taskforce guideline of 70 µg/m³. Public health benefits would be achieved if this target could be met. As the population of Port Hedland is expected to grow to approximately 17,000 people the population exposed to PM₁₀ and associated population risk will increase. The number of attributable cases will increase with population unless reductions in PM₁₀ can be achieved.

The current PM₁₀ in Port Hedland should be reduced to better protect public health. PM₁₀ levels should not exceed 70 µg/m³ for the current level of population. As population increases across Hedland further reduction in exposure will be warranted. Exposure reduction through regulation and a range of other strategies should be considered.

2 BACKGROUND

The high PM₁₀ levels that are experienced in Port Hedland have raised concerns within the Western Australian Government about the potential impact on the health of the community. Fugitive dust from ship loading and associated industry activities were identified as being a potential health concern to people living in Port Hedland. In early 2009, the Western Australian Environmental Protection Authority expressed concern at dust levels in Port Hedland. It stated: *a coordinated government and industry approach to the development and execution of an integrated government and industry strategy with explicit emission reduction strategies and explicit exposure reduction strategies is required with strong and inclusive governance arrangements*. A Port Hedland Taskforce of senior government officers from relevant agencies together with representatives from industry and the Town of Port Hedland (TOPH) was convened to advise Ministers and coordinate and implement decisions made by government. The taskforce reviewed the available evidence and recognised there were five broad categories that required clear direction for action. These were:

- a) health risk assessment and analysis;
- b) environmental management controls;
- c) land use planning;
- d) industry initiatives; and
- e) governance.

In 2009, the Port Hedland Taskforce made a number of considered recommendations in their report *Port Hedland Air Quality and Noise Management Plan 2009* (the Report) (DSD, 2009) to reduce exposure to dust. Recommendations were largely based on an extensive review on potential health impacts of exposure to crustal material undertaken by the Lung Institute of Western Australia in 2007 (LIWA, 2007) and an exploratory study of hospital admissions undertaken by the Department of Health (DoH) in 2006 (DoH, 2006). Of the recommendations in the Report, two key recommendations are directly relevant to this assessment:

- *To maintain the co-existence of industry and community and manage potential risk to human health, it is important that an interim air quality target be developed for Port Hedland. **The Taskforce recommends adoption of an interim air management criteria of 70 µg/m³ (24 hour average) with 10 exceedences per calendar year.** It is expected that this criteria will be met east of Taplin Street and that significant reductions will be achieved between Taplin and McKay Streets.*
- *The Taskforce recommends a long term health [risk assessment] study for the region is undertaken to provide critical information for the ongoing management of Port Hedland air quality.*

2.1 Summary of the DoH 2006 Exploratory Study

The DoH (2006) study examined hospital admissions from settlements within the Hedland boundary. Residential areas both within close proximity and away from the large-scale multi-industry and port facilities were included. Hospital admissions for the years 1993 to 2004 were extracted from the Western Australian Hospital Morbidity Database System for residents of Port Hedland as defined by the postcodes 6721 and 6722 (WA DoH, 2006).

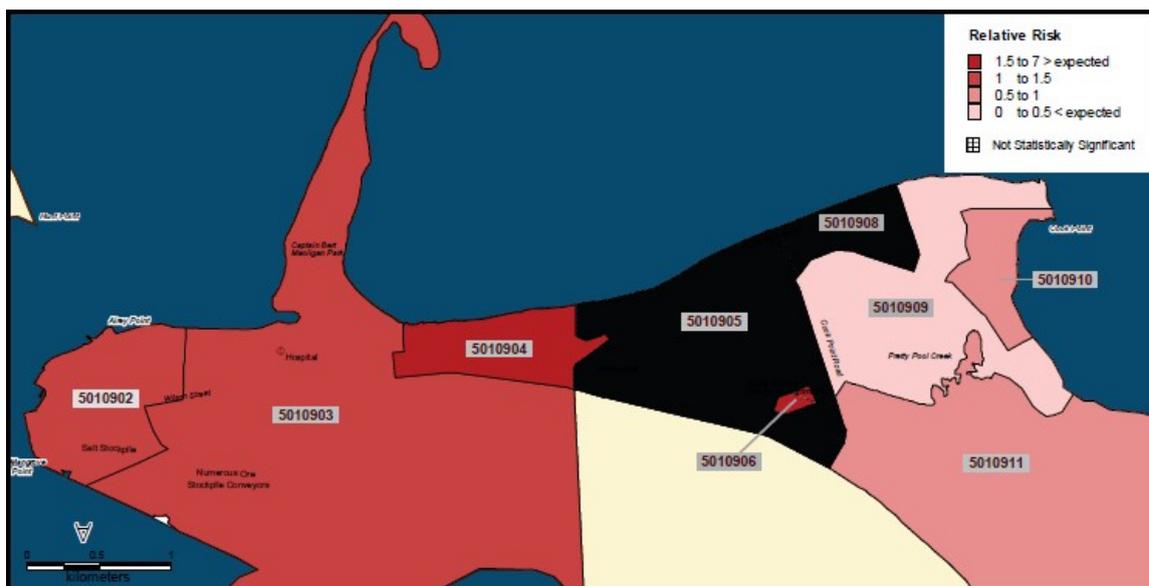
Previous research had shown that the hospital admission rate for the population in remote Western Australia was higher than that of the population of the state (Sommerford, 1995). The results also showed that the rate of respiratory admissions for the Shire of Port Hedland as a whole is significantly higher than that of the state population (1.29 times higher). The 2006 DoH study presented the results of an exploratory investigation into the variation of respiratory, cardiovascular and digestive hospital

admissions within the Port Hedland Township. Examining the town in this way provided a greater understanding of the population characteristics and admissions rates.

The study found that there was a higher risk of admissions for respiratory diseases in the western section of Port Hedland Township compared with the east. All diseases studied (respiratory, cardiovascular and digestive) showed a similar pattern of admissions that reflected the residential population. The geographical trend of disease risk was such that there are higher risks of admissions for Census Collection Districts on the western side of Port Hedland when compared to the state for all disease types investigated. However, this high relative risk was not reflected on the eastern side of the town and was, in fact, statistically significantly lower than the state average. This pattern suggested that additional factors, beyond the demographic and economic factors that were contributing to this variation (DoH, 2006).

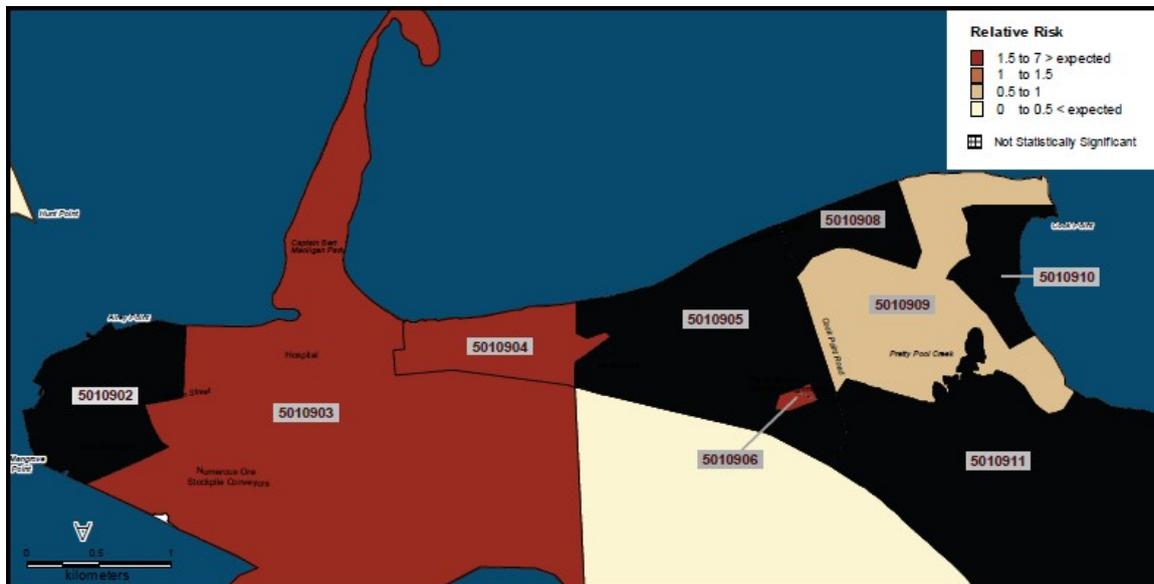
Figure 1 and **Figure 2** (reproduced from the WA DoH 2006 report) show the variation in relative risks for hospital admissions for respiratory diseases (**Figure 1**) and cardiovascular diseases (**Figure 2**).

Figure 1: Map of Risk Estimates for Respiratory Hospital Admissions Port Hedland 1993-2004.



As can be seen from **Figure 1** the relative risks in the west end of Port Hedland all exceed 1 showing that there was a higher risk for admissions to hospital in Port Hedland than the rest of Western Australia. The relative risks for the east end were substantially lower and less than 1. The highest relative risk observed was 6.6 for the Aboriginal community at Census Collection District 5010906. For the west end of the Port Hedland township the highest relative risk was 2.1 which is double the State average (WA DoH, 2006).

Figure 2: Map of Risk Estimates for Cardiovascular Hospital Admissions Port Hedland 1993-2004.



As with the respiratory admissions, the relative risks for cardiovascular admissions were also higher in the west end of Port Hedland compared to the east end and the State average. The relative risks for cardiovascular admissions were lower than those observed for respiratory disease. The highest relative risk was again for the Aboriginal community at Census Collection District 5010906 which was 5.9. For the west end of the Port Hedland township the highest relative risk was 2 which is again double the State average (WA DoH, 2006).

2.2 Summary of the Lung Institute of Western Australia (LIWA) 2007 literature review

The LIWA reviewed the available scientific literature about the hazards associated with ambient air components of the dust in Port Hedland. This information has been updated in Section 6 of this assessment. The intent of the LIWA review was to determine if the potency of PM from sparsely populated, arid, rural Port Hedland was different to PM in heavily populated, urban areas. A definitive conclusion could not be made due to the absence of informative data from similar environmental conditions as those experienced in Port Hedland. On the weight of evidence of available studies LIWA suggested that the Taskforce adopt the US EPA Clean Air Science Advisory Committee standard of 70 $\mu\text{g}/\text{m}^3$ as a 24-hour average for coarse particles ($\text{PM}_{10-2.5}$) while Hedland specific data could be collected.

2.3 Objectives of the HRA

Subsequent to the LIWA report air shed modelling identified that the proposed 70 $\mu\text{g}/\text{m}^3$ guideline could be met east of Taplin St but not west of Taplin St. The proposed guideline with 10 exceedances was introduced east of Taplin St as an interim measure in 2010. To reduce exposure west of Taplin St tighter industry regulation, land-use restrictions and building restrictions were introduced. A monitoring program was established to collect data for this assessment.

The aim of this HRA is to enable a good understanding of the health risks associated with the quality of air in Port Hedland. The scope of the HRA and the pollutants of concern were determined in the HRA methodology (ToxConsult, 2012). The questions addressed in this assessment include:

1. What are the health effects associated with the current air quality at Port Hedland?
2. What are the incremental health impacts over background^a due to the Port's activities?
3. What are the potential health impacts associated with the anticipated increase in activity at the Port?

In examining these questions the HRA has addressed the questions:

- Is the interim guideline of 70 µg/m³ for non-specific PM₁₀ currently used for judging air pollution monitoring at Port Hedland appropriately health protective and defensible?
- What are the potential health impacts^b at different airborne concentrations of dust?

3 HEALTH RISK ASSESSMENT METHODOLOGY

3.1 Approach to Human Health Risk Assessment

Health is defined by the World Health Organization (WHO) as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity (WHO, 1948). Well-being is broadly described as an individual's self-assessment of their state of happiness, healthiness and prosperity. It relates to the quality of life and one's ability to enjoy it. There are many social and economic factors that impinge upon well-being.

The following are examples of determinants of health well-being (enHealth 2012, NHC 2004):

- Social and cultural factors (e.g. social support, participation, access to cultural resources).
- Economic factors (e.g. income levels, access to employment).
- Environmental factors (e.g. land use, air quality).
- Population-based services (e.g. health and disability services, leisure services).
- Individual/behavioural factors (e.g. physical activity, smoking).
- Biological factors (e.g. biological age).

A health risk assessment (HRA) is an analysis that uses information about pollutants to estimate a theoretical level of risk for people who might be exposed to defined levels of these substances. The information on the pollutants comes from scientific studies and measurement data of emissions or ambient data.

Risk assessments are often conducted by considering possible or theoretical community exposures predicted from air dispersion modelling or using environmental concentrations that have been

^aIn this HRA 'background' air pollution is taken to mean the contribution arising from all sources minus that of Port activities. Port activities include all processes associated with exporting ore concentrates including the infrastructure required for Port activity, rail, truck and ship movement. 'Background' is therefore consequently defined as air pollution arising from other commercial activity, domestic doings (both likely to be insignificant), sea spray and crustal sources excluding stockpiles (near and far).

^bThis dot point relates to being able to judge the health impacts of any given PM air concentration (i.e. exposure) for a range of health endpoint. The potency of PM to cause a specific health effect is different for the different endpoints.

measured in the potentially affected population. Conservative safety margins are built into a risk assessment analysis to ensure protection of the public. During the risk assessment analysis the most vulnerable people (e.g. children, the sick and elderly) are carefully considered to make sure that all members of the public will be protected.

The risk assessment helps answer common questions for people who might be exposed to hazardous pollutants in the environment, in this case dust levels in Port Hedland. The HRA is a useful tool for estimating the likelihood and severity of risks to human health, safety and the environment and for informing decisions about how to manage those risks. It is a document that assembles and synthesizes scientific information to determine whether a potential hazard exists and/or the extent of possible risk to human health.

Although this report describes certain technical aspects of the risk assessment, it does not address the important processes of risk management and risk communication.

The risk assessment process comprises five components: issues identification, hazard (or toxicity) assessment, exposure assessment, risk characterisation and uncertainty assessment. These are detailed in the document "*Environmental Health Risk Assessment: Guidelines for assessing human health risks from environmental hazards*" (enHealth, 2012).

Some of the key factors and questions that are taken into consideration at each of these stages include the following.

1. **Issue Identification** – Identifies issues that can be assessed through a risk assessment and assists in establishing a context for the risk assessment.
2. **Hazard Assessment** – Identifies hazards and health endpoints associated with exposure to hazardous agents and provides a review of the current understanding of the toxicity and risk relationship of the exposure of humans to the hazards.
3. **Exposure Assessment** – This task identifies the groups of people who may be exposed to hazardous agents and quantifies the exposure concentrations.
4. **Risk Characterisation** – This task provides the qualitative evaluation of potential risks to human health. The characterisation of risk is based on the review of concentration response relationship and the assessment of the magnitude of exposure.
5. **Uncertainty Assessment** – identifies potential sources of uncertainty and qualitative discussion of the magnitude of uncertainty and expected effects on risk estimates.

3.2 Health Risk Assessment Methodology for Port Hedland

The HRA methodology was established in the Health Risk Assessment Methodology (HRAM) document (ToxConsult, 2012). The HRAM identified that the pollutants of concern in Port Hedland that should be assessed in the HRA were PM₁₀, PM_{2.5}, NO₂, SO₂, respirable crystalline silica, asbestos fibres, and metals including manganese, copper, iron oxide, chromium VI and chromium III. With the exception of chromium VI and chromium III all these pollutants have been included in the HRA. Chromium VI and chromium III could not be assessed due to issues with the filter media used in the sampling for these metals which contaminated the samples and invalidated the dataset. Therefore no data was available to assess the risk posed by these metals in Port Hedland.

The HRAM identified that PM₁₀ levels in Port Hedland arise from a range of sources including industrial emissions, regional dust, combustion sources and salt (both sea-salt and salt from industry). It was recommended that the HRA assess the risks associated with each of these sources and assumed that the results of air dispersion modelling and source apportionment studies would be available to inform this assessment. Although air dispersion modelling and source apportionment studies were undertaken

in parallel to the HRA, the results of these studies were not available for use in the HRA. Therefore the HRA has been undertaken for total PM₁₀ only as no other data was available. It is recommended that once these studies are complete that this information be made available for a future extension to this HRA.

The PHIC monitoring network was established primarily for the HRA but the data is used for a range of reasons including providing data for the HRA, compliance and for dust management purposes. The data from the Richardson St, Kingsmill St, Taplin St, Neptune Place and Acacia Way sites have been used in the HRA to assess the risk from PM₁₀ on the health of the exposed populations in Port Hedland and South Hedland. Although the Kingsmill data was used in risk calculations there was no difference in the results obtained using the Richardson St data and the Kingsmill data. As the Richardson St monitoring data was the more comprehensive and complete dataset the risk calculations based on this data have been reported in the HRA.

The HRAM recommended that the Wedgefield area be included in the HRA. Wedgefield is an industrial area that has no residential development. Some industrial sites have limited site worker accommodation information on the number of people who reside in Wedgefield and the length of time of residence, the age of the population and baseline health status was not available. Therefore it was not possible to calculate the risk to people living in Wedgefield. Given the nature of the activities in Wedgefield and the fact that there are no published plans to develop Wedgefield for residential purposes, the exclusion of Wedgefield from the HRA does not impact on the findings of the HRA. The monitoring data at Wedgefield indicates that PM₁₀ levels at this location are generally higher than those in other parts of Port Hedland and South Hedland. The impact of dust from Wedgefield is reflected in the monitoring data collected at sites within Port Hedland and South Hedland and is therefore accounted for through the use of this data.

The HRAM also recommended that the HRA assess the risk to the Tjalkaboorda Aboriginal Community located in the east end of Port Hedland. In the previous DoH (2006) report this community was reported as having a higher rate of hospital admissions for respiratory and cardiovascular disease than the rest of Port Hedland or the Pilbara region as a whole. Although requests were made to DoH and indigenous health authorities in Port Hedland and the Pilbara region, no information was available on the population that live within this community. Health statistics specific to this community were not available nor were population demographics. Therefore a separate assessment was not possible for this community. However, the monitoring data collected at Taplin St and Neptune Point can be considered as being representative of the exposure of this community. Therefore, risks calculated for the east end of Port Hedland can be considered to be representative of the risk to the Tjalkaboorda Aboriginal Community.

The HRAM proposed equations that could be used to calculate the attributable risk associated with exposure to PM₁₀ in Port Hedland. The risk characterisation has been conducted using these equations. The HRAM recommended that the exposure response functions used be modified to account for the toxicity of sea salt. Given that only preliminary data on the contribution from sea salt to total PM₁₀ was available for the HRA it was not possible to adjust the PM₁₀ levels to account for sea salt. In addition, the recent scientific literature, as cited in WHO (2013), is inconclusive about the toxicity of sea salt. At this stage it is not possible to conclude that sea salt particles behave any differently to any other particle in the same size range. The epidemiological studies indicate that particle size is still the major determinant in the observed health effects. Therefore no adjustment has been made for the contribution of sea salt. As more information becomes available through air dispersion modelling and source apportionment studies, the contribution from sea salt may be able to be determined and accounted for in the HRA.

A number of toxicity reference values were identified in the HRAM for use in the HRA. These are summarised in Table 1:

Table 1: Toxicity Reference Values for use in Health Risk Assessment

Substance	Averaging period	Standard or guideline $\mu\text{g}/\text{m}^3$	Source
Nitrogen oxide	1 hr	246	NEPM(1998, as varied in 2003)
	1 year	62	NEPM(1998, as varied in 2003)
Sulfur dioxide	1 hr	572	NEPM(1998, as varied in 2003)
	24 hr	229	NEPM(1998, as varied in 2003)
	1 year	57	NEPM(1998, as varied in 2003)
PM ₁₀	24 hr	50	NEPM(1998, as varied in 2003)
Asbestos		0.01fibres/ml	DOH (2009)
Silica	Annual	3	OEHHA 2005, EPA Victoria 2007, US EPA 1996
Chromium III total	1 hr	10	TCEQ 2009
	24 hr	0.5	Toxikos 2010
Chromium VI	Annual	0.0002 $2.3 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$ unit risk factor	DEFRA 2009, TCEQ 2013
	24 hr	0.3	ATSDR 2012, Toxikos 2010
Copper	24 hr	1	RIVM 2001, OEHHA 1999
Manganese	Annual	0.15	WHO 2000, Roels et al 1992
Iron oxide	24 hr	120	ACGIH 2006, Safe Work 2005

4 TOXICITY REFERENCE VALUES

As part of the overall scope of work related to the Health Risk Assessment for Port Hedland, a review of the toxicity reference values (TRVs) as provided in the Health Risk Assessment Methodology (HRAM) was undertaken to ensure the most scientifically rigorous and valid values were used.

Most of the TRVs outlined in the HRAM have been adopted without change. As shown in **Table 2**, the values where changes are proposed through subsequent review are those for chromium (III) total (24-hour), chromium VI (24-hour) and copper (presented in bold in **Table 2**). The TRVs proposed for use in the HRA are shown in **Table 2**.

Table 2: Ambient Air Quality Standards and Guidelines for the Contaminants of Concern.

Substance	Averaging period	Standard or guideline as proposed in the ^a HRAM	Source	Proposed Changes	Source
		µg/m ³			
Nitrogen oxide	1 hr	246	NEPM	No Change	
	1 year	62	NEPM	No Change	
Sulfur dioxide	1 hr	572	NEPM	No Change	
	24 hr	229	NEPM	No Change	
	1 year	57	NEPM	No Change	
PM ₁₀	24 hr	50	NEPM	No Change	
PM _{2.5}	24 hr	25	NEPM	No Change	
	Annual	8	NEPM	No Change	
Respirable Crystalline Silica	Annual	3	OEHHA 2005, EPA Victoria 2007, US EPA 1996	No Change	
Copper	24 hr	1		100 µg/m³	OEHHA, 2013
Manganese	Annual	0.15	WHO 2000, Roels et al 1992	No Change	
Iron oxide	24-hr	120	ACGIH 2006; Safe Work 2005; Toxikos, 2010	No Change	

^aHRAM – Health Risk Assessment Methodology

4.1 Iron Oxide – 24-hour

The iron oxide TRV was derived for the HRAM (ToxConsult, 2012) and is presented below.

Prolonged inhalation of high concentrations of fine particles of metallic iron, or iron compounds in an occupational situation causes pulmonary siderosis. This is a relatively benign pneumoconiosis, characterised by large accumulation of inorganic containing macrophages in the lungs with minimal reactive fibrosis. In its pure form (i.e. due to iron oxide exposure only) the condition probably does not progress to true nodulation, as seen with silicosis and is usually asymptomatic, it does however show up as abnormal changes on X-rays (McLaughlin et al. 1945, Teculesu and Albu 1973, Morgan 1978, Brooks 1986, Sentz and Rakow 1969).

When iron is inhaled with other fibrogenic mineral dusts pulmonary fibrosis can be induced (ACGIH 2006). This is called mixed dust pneumoconioses or silicosiderosis. Haematite pneumoconiosis occurs in iron miners who are exposed to iron oxide in combination with free silica and silicates. It is characterised by a brick red coloured lung surface and has been likened to a simple form of coal workers' pneumoconiosis (Brooks 1986).

Of the studies where exposure was to iron oxide dust alone, two (Teculescu and Albu 1973, Sentz and Rakow 1969) contain information on exposure concentrations. In the first (Teculescu and Albu 1973), subjects were male workers in a plant manufacturing pure red iron oxide ('rouge'). Dust concentrations (30% was <1 µm, 45% 1-3 µm, 23% 3-5 µm, and 2% 5-10 µm in diameter) varied according to the place and phase of the production process. They were 10 to 15 mg/m³ in the chemical reaction and filter room, 45 to 700 mg/m³ in the drying and mill room, 306 to 770 mg/m³ in the calcination room, and 330 to 500 mg/m³ in the packing room. The silica content was negligible (<1%). Clinical and X-ray investigations were made in 1965, and repeated in 1967 and again in 1969. A high prevalence of respiratory symptoms was found related to the smoking habits of subjects, but X-ray changes were also found. In the last survey (1969), 38 of the 113 workers had opacities on their standard chest film^a. Comparison with earlier films revealed progression in 41%, regression in 20% and no change in the rest over a 3-year interval. Fourteen subjects of those with nodular shadows, who had not been exposed to other dusts or noxious gases, were selected to undergo pulmonary function tests. It is not stated in the paper which exposure group these subjects belonged to (i.e. chemical reaction, drying, calcination, or packing room concentrations). They had been exposed to iron oxide dust for 4-13 (mean 10) years. The group included four smokers, three ex-smokers and seven non-smokers. The authors found no restrictive ventilatory impairment in pulmonary function tests and the static lung compliance was normal. The only effects observed were slight hypoxemia at rest in one subject and a fall in the transfer coefficient in another; these were attributed to chronic bronchitis and recent respiratory disease, respectively. This study indicates long-term exposure to respirable particles (≤10 µm) of pure iron oxide dust between 10 and 770 mg/m³ is associated with minimal changes on X-ray diagnosis that are potentially reversible, but not with decrements in pulmonary function. This is consistent with another study (Sentz and Rakow 1969), in which electric arc and powder-burning workers exposed to iron oxide fume well over 10 mg/m³ had no discernible changes in their chest X-rays. It is unknown if this study investigated pulmonary function.

^a According to the ILO classification these were 'pinhead' (p) type in 22, micronodular (m) type in 9, and of nodular (n) type in the remaining 7. No conglomeration was found.

From these brief considerations, 10 mg/m³ could be interpreted as either a no effect (based on pulmonary function) or a low effect (based on iron accumulation) concentration. In this document it has been assumed to represent a conservative low effect concentration for respirable iron oxide dust.

Two agencies have derived air guideline values for iron or iron oxide:

- Ontario (Ontario MoE 2012) for metallic iron: 4 µg/m³ (24 hrs)
- Arizona (ADHS 1999): 150 µg/m³ (1 hr) and 40 µg/m³ (24 hrs)

4.1.1 Ontario

Ontario MoE (2012) does not provide any background documentation explaining the derivation of their guideline value; therefore we are unable to comment on its appropriateness.

4.1.2 Arizona

Arizona (ADHS 1999) calculates 1-hour and 24-hour air guideline values (AGVs) using occupational exposure limits (OELs) established or recommended by the United States Occupational Safety and Health Administration (OSHA), the National Institute of Occupational Safety and Health (NIOSH), the National Institute for Environmental Health Sciences (NIEHS) and presumably the American Conference of Industrial Hygienists (ACGIH).

- Twenty-four hour AGVs are established by dividing the most recent and lowest OEL, or OEL recommendation, by 126. This factor incorporates adjustment for continuous exposure (24/8 hours x 7/5 days), and a safety factor of 30 to protect sensitive members of the population. Most other jurisdictions use a factor of 10 for the latter.
- Although not directly stated in the Arizona documentation, presumably the 24-hour guideline for iron oxide (40 µg/m³) was based on the ACGIH threshold limit value of 5 mg/m³ for iron oxide (respirable fraction, i.e. PM₁₀) (ACGIH 2006) to protect against the development of X-ray changes in the lung following long term exposure not associated with any clinical changes ($5 \text{ mg/m}^3 \div 126 = 0.04 \text{ mg/m}^3$, i.e. 40 µg/m³).
- The 1-hour AGV for iron oxide (150 µg/m³) was calculated by multiplying the 24-hour AGV by 3.8 (the proportional difference in the lowest adverse effect level observed for 24-hour and 1-hour exposure to the common irritant, SO₂) ($40 \text{ µg/m}^3 \times 3.8 = 152 \text{ µg/m}^3$, rounded to 150 µg/m³).

4.1.3 Discussion

From the above it was considered more appropriate to derive an air quality guideline value for iron oxide dust *de novo* using the Australian workplace exposure standard from Safe Work (2005) and the threshold limit value from ACGIH (2006) of 5 mg/m³ (8 hour time-weighted average). From the ACGIH (2006) documentation, the threshold limit value suggested appears to be rationally supported and protective of health effects. Application of a correction factor to adjust for assumed continuous exposure in the general population (24/8 hour x 7/5 days = 4.2) and an uncertainty factor of 10 to

account for potentially more susceptible individuals in the general population compared to a healthy workforce gives an indicative air guideline value of 0.119 mg/m³, i.e. 120 µg/m³. The averaging time for this guideline is suggested to be 24 hours. This is to minimise the potential for development of minimal X-ray changes in the lung on long term exposure, without any physical impairment of lung function (ACGIH 2006).

It is recommended that the indicative air guideline value of 120 µg/m³ be used to assess the health risks of exposure to iron oxide.

However, since any target PM₁₀ guideline for locations at Port Hedland is lower than a specific guideline for iron oxide, and the iron oxide content of total ambient PM (as measured) is less than 100%, the suggested target PM₁₀ guideline will supersede a guideline for iron oxide and be protective against health effects potentially associated with the anticipated exposures to iron oxide. For the purposes of this assessment the iron oxide TRV was used to assess risk.

4.2 Copper

Copper is an essential element and there is no convincing evidence for genotoxicity. The rationale in the HRAM for the proposed TRV was a key animal study that noted respiratory and immunological effects (RIVM2001). In addition to the value from the Netherlands National Institute for Public Health and the Environment (Dutch: Rijksinstituut voor Volksgezondheid en Milieu (RIVM), OEHHA derived a value based on occupational exposures.

4.2.1 RIVM

The RIVM (2001) derived their TRV of 1 µg/m³ based on a No Observed Adverse Effect Concentration of 0.6 mg/m³ for histological and immunological effects in rabbit lungs after inhalation exposure to copper chloride at 0.6 mg Cu/m³ for 6 weeks (6hr/d, 5 d/wk) (Johansson et al. 1984). A correction factor for continuous exposure (6hr/24hr x 5d/7d) and an extrapolation factor of 100 (10x interspecies and 10x for intraspecies) was applied to give their TRV of 1 µg/m³.

4.2.2 OEHHA

Based on the combined studies from Gleason, 1968 and Whitman, 1957; 1962, OEHHA derived their acute reference level (REL) for copper. The following section summarises the guideline derivation procedure.

4.2.2.1 Acute REL for Copper

The current REL is based on the ACGIH TLV of 1 mg/m³ of copper dust. The TLV of 1 mg/m³ is reported as a NOAEL based on the report from Whitman (1957) indicating that exposure to copper dust was detectable by taste but that no other symptoms occurred following exposure to 1 - 3 mg/m³ for an unknown duration.

The NOAEL was then divided by an uncertainty factor of 10 to account for variability in individual response. No time extrapolation was applied because the duration of the exposure was not clearly defined by either of the available reports.

4.2.3 DISCUSSION

It is noted that there is a 100-fold difference between the values from the RIVM and OEHHA. In examining the rationales for both of the values, it is evident that the animals in the RIVM study are more susceptible to adverse effects due to copper exposure than are humans. Results from occupational exposure studies have noted much higher LOAEL/NOAEL values than that of the study of Johansson

et al. (1984), which was the basis of the TRV from RIVM. Given the availability of occupational exposure studies and the apparent species differences between animals and humans with respect to the toxicity of copper via inhalation the TRV value from OEHHA of 100 µg/m³ has been recommended for use in the HRA.

5 POPULATION PROFILE PORT HEDLAND

Population statistics were obtained from the Town of Port Hedland (via DoH) and the Australian Bureau of Statistics (ABS). These statistics apply to the permanent population of Port Hedland. Data on the population of fly-in-fly-out (FIFO) workers were taken from Port Hedland Council data and the draft FIFO strategy (Town of Port Hedland, 2010).

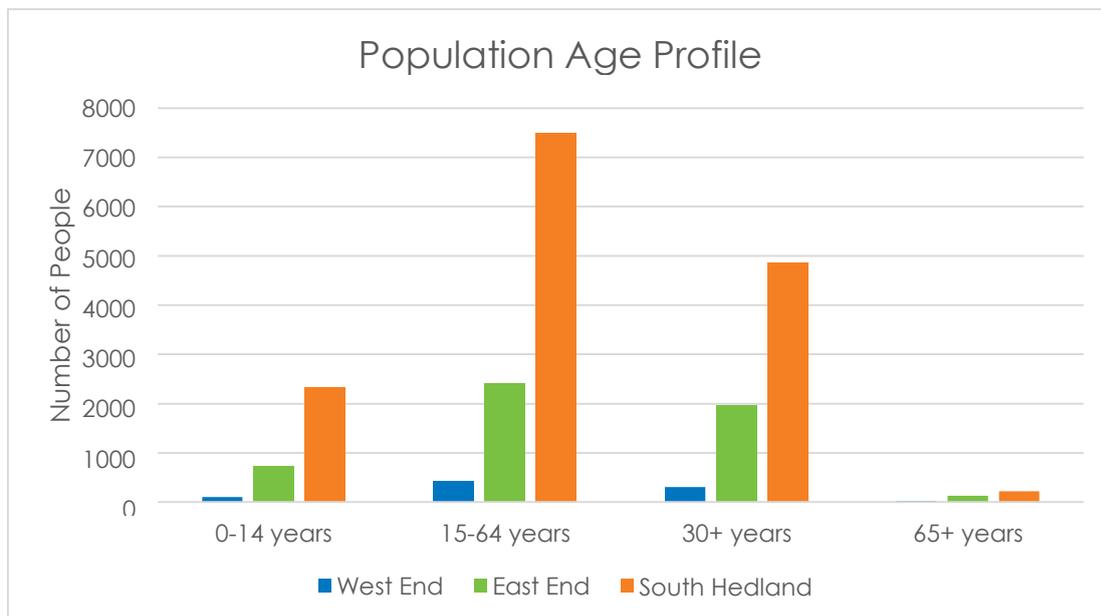
For the permanent population data was available for Port Hedland as a whole, the West End (west of Taplin St), the East End (east of Taplin St) and South Hedland. **Table 3** summarises the population data as a whole compared with the Western Australia average.

Table 3: Population Profile Port Hedland (Source Australian Bureau of Statistics)

Population profile	Hedland	Port Hedland	West End	East End	South Hedland	Western Australia
Total Population	13772	4590	529	4061	9782	2,239,170
% 65 years and older	2.6	2.6	2.6	2.6	2.6	12.3
% less than 14 years of age	22	22	22	22	22	19.7
Median age	30	30	30	30	30	36
% indigenous persons	14	14	14	14	14	3

Figure 3 shows the population age profile for all study areas. It is clear that the age distribution is consistent across all areas being between 15-64 years of age. Children and people older than 65 years of age form a very small percentage of the population across all study areas. These two groups are known to be populations sensitive to the effects of air pollution.

Figure 3: Population Age Profile Port Hedland Study Area



Information obtained on the FIFO workforce shows that the majority of workers are in the 15-64 years age group. The draft FIFO strategy (Town of Port Hedland, 2010) indicates that the FIFO workforce at that time was 3000 people. It was predicted to increase to 8000 by 2014 peaking at 15,000 by 2016. The majority of these workers are housed in mining camps close to South Hedland and the airport.

Recent studies (WHO, 2013) have shown that people who have a low socioeconomic status (SES) form a group within the population that is particularly vulnerable to the effects of air pollution. This is largely due to the fact that people within these groups usually have poorer health status than people within higher SES groups. They may also have poorer access to medical care. In addition, they usually live in areas that are more polluted (e.g., near major roads or near industry) as property is generally cheaper in these areas. There are several indices of social deprivation used to assess SES status in Australia. One commonly used is the SEIFA index. The SEIFA index is a measure of relative social disadvantage and takes into account 20 variables to assess relative social disadvantage. The lower the SEIFA index the greater the level of disadvantage.

The SEIFA index for Port Hedland (including South Hedland) is higher than that of Western Australia as a whole which indicates that the population is less disadvantaged than the rest of the State. This is largely due to the median wage of people in the mining industry in Port Hedland which is approximately double the median weekly income of the rest of the State. Within Port Hedland there is variation in the SEIFA index. In general the SEIFA index in the town of Port Hedland (east and west end combined) is above the State average (1036 -1131 compared to State 1000). Areas of South Hedland experience a greater level of disadvantage compared to the town of Port Hedland and the rest of WA. The SEIFA indices for South Hedland range from 974 – 1055. A SEIFA index less than 1000 indicates a higher level of disadvantage compared with the rest of the State.

Sensitive locations such as schools and hospitals have been identified. The Port Hedland hospital has been moved from the west end of Port Hedland to South Hedland. Two schools have been identified, Cassia Primary School and Hedland Senior High School both of which are located in South Hedland. There are two primary schools are located in Port Hedland which are included within the study area. These may be affected by Port operations. All other schools and the hospital are located in South Hedland away from the Port operations.

6 HEALTH RISK ASSESSMENT PM₁₀ AND PM_{2.5}

6.1 Hazard Assessment

The health effects of particles linked to ambient exposures have been well studied and reviewed by international agencies (NEPC, 2010; USEPA, 2004, 2009, 2012; WHO, 2013, 2006; OEHHA, 2000). In recent years a large amount of research has focussed on the health effects of particles and an increasing body of literature reports associations between particles and adverse health effects. Effects have been found for both PM₁₀ and PM_{2.5} and to a lesser extent, ultrafine particles (UFPs). Most information comes from population-based epidemiological studies that find increases in daily mortality, as well as morbidity outcomes such as increases in hospital admissions and emergency room attendances, and exacerbation of asthma associated with daily changes in ambient particle levels. There has been an increasing focus on the link between exposure to particles and cardiovascular outcomes. In addition to studies on the various size metrics for particles, research has also investigated the role of particle composition in the observed health effects (USEPA, 2009, 2012; WHO, 2013).

The evidence on the health effects of particles comes from several major lines of scientific investigation: characterisation of inhaled particles; consideration of the deposition and clearance of particles in the respiratory tract and the doses delivered to the upper and lower airway and the alveoli; animal and cellular studies of toxicity; studies involving inhalation of particles by human volunteers; and population-based epidemiological studies. The findings of these different lines of investigation are complementary and each has well-identified strengths and limitations. While the findings of epidemiological studies have been given the greatest weight in setting standards for airborne particles, studies on human volunteers (clinical studies) can provide information on exposure–response relationships for acute, transient effects in healthy and potentially susceptible individuals. Studies of this design, involving both healthy persons and adults with chronic diseases, have been carried out using exposure to concentrated ambient particles (USEPA, 2009).

There is substantial new evidence from time series studies of daily mortality, particularly from multi-city studies that span Europe and North America (USEPA, 2012, 2009; WHO, 2013) and also Australia (NEPC, 2010). Several studies conducted in Australia also show adverse effects of both PM₁₀ and PM_{2.5} on mortality and morbidity outcomes (Simpson et al., 2005a, b; Barnett et al., 2005; 2006) similar to those observed in overseas studies. The effects observed in the Australian studies appear to be higher than those observed in the US and Europe but comparable to the results of Canadian studies. The epidemiological evidence is supported by an increasingly strong foundation of toxicological research. Various mechanisms have been proposed by which particles may cause and/or exacerbate acute and chronic diseases. Inflammation due to the production of reactive oxygen species is emerging as a central mechanism.

6.1.1 PM₁₀

6.1.1.1 Short-term effects

Most of the evidence of an association between short-term exposure to particles and adverse health outcomes comes from time-series epidemiological studies looking at daily increases in mortality and hospital admissions and emergency room attendances linked to ambient particle concentrations. In addition, the results of panel studies and controlled exposure studies add further evidence for the association between short-term exposure to particles and adverse health effects. The results of recent reviews and studies are summarised below.

6.1.1.1.1 Mortality

The association between exposure to both PM₁₀ and PM_{2.5} and increases in daily mortality have been the subject of extensive research (NEPC, 2010; USEPA, 2004, 2009, 2012; WHO, 2013, 2006). The results of these studies show that PM₁₀ and PM_{2.5} are linked to increases in all-cause mortality and well as cause specific mortality such as cardiovascular and respiratory outcomes. There is also some evidence that exposure to thoracic particles, PM_{10-2.5}, is linked with increases in daily mortality and morbidity (WHO, 2013; USEPA, 2009).

The epidemiological literature indicates consistent positive associations between short-term exposure to PM₁₀ and all-cause mortality. The results of multicity studies report an approximate 0.12–0.81% increase in all-cause mortality per 10µg/m³ increase in PM₁₀ with 24-hour average PM₁₀ concentrations ranging from 13 to 53.2µg/m³. Consistent positive associations have also been found between PM₁₀ and respiratory and cardiovascular-related mortality. Studies conducted in Australia have found similar results with a 0.2% (-0.8–1.2%) increase in all-cause mortality per 10µg/m³ increase in 24-hour average PM₁₀ (Simpson *et al.*, 2005a).

Heterogeneity in PM₁₀ mortality risk estimates is observed between cities and studies, including Australian studies. In the US studies regional heterogeneity and seasonal patterns in PM₁₀ risk estimates were also observed, with the greatest effects occurring in the Eastern U.S. and during the summer, spring and autumn, respectively. Similar heterogeneity and seasonality has been observed in Australian studies (Simpson *et al.*, 2005a; Barnett *et al.*, 2005). An examination of potential confounders (i.e., temperature and co-pollutants) using different study designs (i.e., time series and case crossover) observed that neither is likely to account for differences in PM₁₀-mortality risk estimates between studies. The USEPA (2012, 2009) found that the consistent evidence observed across epidemiological studies is sufficient to conclude that a causal relationship is likely to exist between short-term exposure to ambient concentrations of PM₁₀ and mortality. NEPC (2010), WHO (2013, 2006) and OEHHA (2001) came to similar conclusions.

In recent years there has been a substantial increase in studies showing associations between particles and cardiovascular effects. Epidemiological studies that examined the association between PM₁₀, PM_{2.5} and mortality have provided strong evidence for particle-related cardiovascular effects (USEPA, 2012, 2009; WHO, 2013, 2006).

The association between PM₁₀ and mortality in Europe has been extensively studied. Katsouyanni *et al.* (2003) presented the results from the Air Pollution and Health: a European Approach (APHEA2) study, a multicity study that examined PM₁₀ effects on total mortality in 29 European cities. In a later APHEA study Analitis *et al.* (2006) published a report on effect estimates for cardiovascular and respiratory deaths also based on the 29 European cities, within the APHEA2 study. The results of this study found for the average of 0- and 1-day lags, PM₁₀ risk estimates per 10µg/m³ of 0.76% (95% CI: 0.47–1.05) for cardiovascular deaths and 0.71% (95% CI: 0.22–1.20) for respiratory deaths.

The APHENA study (Samoli *et al.*, 2008) was a collaborative effort by the APHEA, NMMAPS, and the Canadian multicity study investigators to evaluate the coherence of PM₁₀ mortality risk estimates across locations and possible effect modifiers of the particle-mortality relationship using a common protocol. The results of the APHENA study showed that generally, the risk estimates from Europe and the U.S. were similar, but those from Canada were substantially higher. For example, the percent excess risks per 10µg/m³ increase in PM₁₀ for all ages 0.84% (0.30, 1.40), 0.33% (0.22, 0.44), and 0.29% (0.18, 0.40) for the Canadian, European, and U.S. data, respectively. Analysis by age shows that the risk estimates for the older age group (age ≥ 75) were consistently larger than those for the younger age group (age <75) e.g., 0.47% vs. 0.12% (for the U.S. data) for all the three data sets. It is important

to note that the results observed in the Canadian studies are comparable to those observed in the Australian studies (Simpson *et al.*, 2005a).

A study conducted in four Australian cities (Brisbane, Melbourne, Perth and Sydney), found statistically significant associations between particles and all-cause mortality. Meta-analyses carried out for three cities yielded estimates for the increase in the daily total number of deaths of 0.2% (-0.8% to 1.2%) for a 10 $\mu\text{g}/\text{m}^3$ increase in PM₁₀ concentration (Simpson *et al.*, 2005a).

A study conducted in Melbourne found statistically significant positive associations between PM₁₀ and all cause and respiratory mortality in the warm season (November-March). For PM₁₀, a 10 $\mu\text{g}/\text{m}^3$ was associated with an increased risk of 0.18% (95% CI, 0.03–0.33%) for all-cause mortality and 0.59% (95% CI, 0.06–1.13%) for respiratory mortality. Statistically significant associations were also found in the 65+ age group in the warm season (Simpson *et al.* 2000; EPA Victoria, 2000).

6.1.1.1.2 Morbidity

The majority of recent evidence for an association between short-term exposure to PM₁₀ and cardiovascular health effects is derived from epidemiological studies of hospital admissions and emergency department visits. Although some regional heterogeneity is evident in the single-city effect estimates, consistent increases in hospital admissions and emergency department visits for cardiovascular diseases, have been observed across studies, with the majority of estimates ranging from 0.5–1.0% per 10 $\mu\text{g}/\text{m}^3$ increase in PM₁₀ (WHO, 2013). A detailed examination of specific cardiovascular health outcomes has suggested that ischemic heart disease and chronic heart failure are responsible for the majority of particle-related cardiovascular disease hospital admissions however, one large multicity study provides evidence of an association between PM₁₀ and ischemic stroke (USEPA, 2009). Overall, the literature provides consistent evidence for associations between short-term exposure to PM₁₀ and increased risk of cardiovascular hospital admissions and emergency department visits in cities with mean 24-hour average concentrations ranging from 16.8 to 48 $\mu\text{g}/\text{m}^3$.

Large studies conducted in the U.S., Europe, and Australia and New Zealand have confirmed these findings for PM₁₀. The association between particles and hospital admissions for cardiovascular disease and ischemic heart disease appear to be greater in Europe and Australia/New Zealand than in the U.S (NEPC, 2010; USEPA, 2012, 2009; WHO, 2013, 2006). The multicity Spanish EMECAS study (Ballester *et al.*, 2006) found that the statistically significant positive associations observed between PM₁₀ and cardiac hospital admissions were robust to control for other pollutants.

Animal toxicology studies have shown impacts on the cardiovascular system. An inhalation study in animals found lowered cardiac contractility upon exposure to PM₁₀, while several intra-tracheal instillation studies found altered vasoreactivity and elevated levels of systemic inflammatory and blood coagulation markers (USEPA, 2004). In addition, several epidemiological studies have observed physiologic alterations in cardiovascular function including: heart rate variability (HRV), systemic markers of inflammation, coagulation, and oxidative stress in cities with mean 24-hour average concentrations ranging from 10.5 to 46.1 $\mu\text{g}/\text{m}^3$. These findings, along with those reported in the toxicological literature contribute to the biological plausibility of PM₁₀-related cardiovascular effects. Overall, consistent and coherent evidence exists across toxicological and epidemiological studies, which supports the conclusion that short-term exposure to PM₁₀ is associated with an increased risk of cardiovascular morbidity. Furthermore, findings of altered autonomic function, cardiac contractility, systemic inflammation, coagulation, and vasoreactivity provide biological plausibility that exposure to PM₁₀ could lead to more severe effects, including hospital admissions or emergency department visits for ischemic heart disease, congestive heart failure, or ischemic stroke. The USEPA (2009) concluded that collectively, these studies provide sufficient evidence to conclude that a causal relationship is

likely to exist between short-term exposure to ambient concentrations of PM₁₀ and cardiovascular morbidity.

Epidemiological studies that examined the association between short-term exposure to PM₁₀ and respiratory morbidity found consistent positive effects in asthmatic children and adults, but no evidence of an association in healthy individuals in both Australian and overseas studies. The majority of the studies that examined the association between PM₁₀ and respiratory symptoms and medication use found an increased risk ranging from ~1.0 to 1.75 for cough, phlegm, difficulty breathing, and bronchodilator use in asthmatic children in cities with mean 24-hour average concentrations ranging from 16.8 to 64.5µg/m³. Positive, but less consistent effects for respiratory symptoms and medication use were observed in asthmatic adults. An evaluation of respiratory emergency department visits and hospital admission studies found consistent positive associations at ambient PM₁₀ concentrations ranging from 13.3 to 60.8µg/m³, among asthmatic children (~ 2% increase) and older adults with chronic obstructive pulmonary disease (COPD)(~ 0 to 3% increase). Although no toxicological or human clinical studies have examined the effect of short-term exposure to PM₁₀ on respiratory morbidity, the consistent epidemiological evidence alone was sufficient for the USEPA (2009) to conclude that a causal relationship is likely to exist between short-term exposure to ambient concentrations of PM₁₀ and respiratory morbidity.

6.1.1.1.3 Other morbidity outcomes

Worldwide, asthma is one of the most common chronic diseases of childhood. The underlying increased airways responsiveness that is inherent in asthma may increase susceptibility to inhaled pollutants generally and particles specifically (WHO, 2006; OEHHA, 2001). The association between exposure to air pollution and asthma has been studied by tracking hospital admission and GP visit rates and by panel studies of children that evaluate symptom status, medication use, or physiological indicators in relation to PM exposure. Delfino *et al.* (1998; 2002) reported findings of a representative study of 19 California children who were tracked for two-week intervals with measurement of FEV₁; personal exposures to particles were monitored as well. In Europe, the multicentre PEACE study addressed childhood asthma and air pollution, including particles (2006). While not all studies have linked particles to increased risk of exacerbation, the weight of evidence indicates that ambient particles do adversely affect children with asthma (WHO, 2006).

Epidemiological studies of asthmatic children have found increases in respiratory symptoms and asthma medication use associated with higher PM_{2.5} or PM₁₀ concentrations. Associations with respiratory symptoms and medication use are less consistent among asthmatic adults (USEPA, 2009).

6.1.1.2 Long term effects

Most studies investigating the effects of long-term exposure to air pollution have focussed on PM_{2.5}. However there are some that have investigated the effects of PM₁₀ and these are summarised in the following sections.

6.1.1.2.1 Mortality

In an analysis for the Seventh-Day Adventist cohort in California (AHSMOG), a positive association with coronary heart disease mortality was reported among females (92 deaths; RR = 1.42 [95% CI: 1.06–1.90] per 10 µg/m³ PM_{2.5}), but not among males (53 deaths; RR = 0.90 [95% CI: 0.76-1.05] per 10µg/m³ PM_{2.5}) (Chen *et al.*, 2005). The results of this study are suggestive that females may be more sensitive to air pollution-related effects, based on differences between males and females in dosimetry and exposure (USEPA, 2009). As was found with fine particles, a positive association with coronary heart disease mortality was reported for PM₁₀₋₂₅ and PM₁₀ among females (RR = 1.38 [95% CI: 0.97-1.95] per 10µg/m³ PM_{10-2.5}; RR = 1.22 [95% CI: 1.01–1.47] per 10µg/m³ PM₁₀), but not for males (RR = 0.92 [95% CI: 0.66–1.29] per 10µg/m³ PM_{10-2.5}; RR = 0.94 [95% CI: 0.82–1.08] per 10µg/m³ PM₁₀); (Chen *et al.*, 2005).

Two additional studies explored the effects of PM₁₀ on cardiovascular mortality. The Nurses' Health Study (Puetz *et al.*, 2008) is an ongoing prospective cohort study examining the relation of chronic PM₁₀ exposures with all-cause mortality and incident and fatal coronary heart disease consisting of 66,250 female nurses in the north eastern region of the U.S. The association with fatal coronary heart disease occurred with the greatest magnitude when compared with other specified causes of death (hazard ratio 1.42 [95% CI: 1.11–1.81]). The North Rhine-Westphalia State Environment Agency (LUA NRW) initiated a cohort of approximately 4,800 women, and assessed whether long-term exposure to air pollution originating from traffic and industrial sources was associated with total and cause-specific mortality (Gehring *et al.*, 2006). They found that cardiopulmonary mortality was associated with PM₁₀ (RR = 1.52 [95% CI: 1.09-2.15] per 10µg/m³ PM₁₀).

6.1.1.2.2 Morbidity

Children may be at greater risk from long-term exposures to particles or other air pollutants because the growth and development of the respiratory system may be permanently affected by early environmental insults. The Southern Californian Children's Health Study was designed as a 10-year investigation of the impacts of southern California air pollution on lung growth and development and other indices of respiratory health among 3,676 fourth-, seventh-, and tenth-graders in 12 communities, which were chosen to emphasize different long-term air pollution conditions. For data collected in 1986-90, the 24-hr average PM₁₀ concentration ranged from 28.0µg/m³ in Atascadero and Santa Maria to 84.9µg/m³ in Mira Loma and Riverside. In 1994, the mean 24-hr average PM₁₀ concentration across the 12 communities was 34.8µg/m³ (range = 13.0µg/m³ in Lompoc to 70.7µg/m³ in Mira Loma) (McConnell *et al.*, 1999; Peters *et al.*, 1999a).

At enrolment, neither PM₁₀ nor PM_{2.5} were associated with respiratory illness among the total cohort (ever or current asthma, bronchitis, cough, or wheeze) assessed by questionnaire (Peters *et al.*, 1999a). In contrast, among children with asthma, respiratory symptoms increased with increasing particle levels (McConnell *et al.*, 1999). Specifically, there was about a 40% increase in the odds of bronchitis among asthmatics per 19µg/m³ change in PM₁₀ measured over 2-week intervals (OR=1.4, 95% C.I. = 1.1-1.8). Exposure to a 15µg/m³ increment in fine particles resulted in about the same magnitude of increase in risk, which was not statistically significant. Both measures of particles were also associated with at least a doubling of risk of phlegm in asthmatic children. Acid vapors and NO₂ were also associated with respiratory symptoms in asthmatic children. However, because PM₁₀, PM_{2.5}, NO₂, and acid vapor were highly correlated, it is not possible to definitively attribute these effects to any single pollutant (McConnell *et al.*, 1999).

In another cross-sectional analysis of the Children's Health Study PM₁₀ and PM_{2.5}, as well as NO₂, were significantly associated with decreased lung function (forced vital capacity [FVC], forced expiratory volume in one second [FEV₁], and maximal mid-expiratory flow [MMEF]), especially in girls who spent more time outdoors (Peters *et al.*, 1999b). These results were supported in an analysis of lung function growth over a four-year period (Gauderman *et al.*, 2000). Examining the data from a sample of children who were fourth graders at enrollment, the investigators found statistically significant effects on lung function growth associated with PM₁₀, PM_{2.5}, PM_{10-2.5}, NO₂, and inorganic acid vapors. The effects were more pronounced for tests measuring airflow at low lung volumes, especially for children spending more time outdoors. There were no differences observed by gender. Although the effects on the children who were seventh- and tenth-graders at enrollment were generally also negative, these were not statistically significant, in part because the sample sizes in the higher grades were markedly smaller. As with the cross-sectional symptom data, the independent effects of the different pollutants cannot be assessed because of high inter-pollutant correlations.

The Australian Child Health and Air Pollution Study (ACHAPS, (SCEW, 2011)) used a similar study design as that used in the Southern Californian Children's Health Study. The results of a cross-sectional study

of approximately 4,000 Australian school children aged 7-11 years showed varied results for the particulate matter exposures used in ACHAPS. PM₁₀ was associated with decline in FEV₁ post-bronchodilator and increase in exhaled NO, but no overall increase in current symptoms. PM_{2.5} was associated with an adverse effect on FVC post-bronchodilator and on exhaled NO, with no overall effects on current symptoms, but showed increased risk of lifetime wheezing, asthma, and asthma medication, and current asthma, use of beta-agonists and itchy rash in non-atopic children. Females had an increase in FEV₁/FVC ratio pre-bronchodilator for recent PM_{2.5}, and recent PM₁₀ exposures, with non-significant effects in males. Despite the absence of effect on current symptoms, a reduction in lung volume at this age may have longer-term adverse consequences if it persists into later life (SCEW, 2011).

The Swiss Study on Air Pollution and Lung Disease in Adults (SAPALDIA) examined the long-term effects of air pollution exposure in a cross-sectional study of 9,651 adults residing in eight areas in Switzerland in 1991. Eligibility for the study was conditional on having lived in the same area for at least three years. Particle measurements used in the analysis were taken over a 1-year period (1991 for TSP, and 1993 for PM₁₀), on the assumption that air pollution concentrations had not changed substantially over the proceeding several years. Statistically significant associations were observed between chronic symptoms (chronic phlegm, chronic cough, breathlessness at rest during the day or at night, and dyspnea on exertion) and the pollutant metrics TSP, PM₁₀ and NO₂ (Zemp *et al.*, 1999). These associations were strongest for PM₁₀. The investigators estimated that an increase of 10µg/m³ PM₁₀ (within the observed range across cities of 10.1 – 33.4µg/m³), would correspond to increases in risk among never smokers of 30% for chronic phlegm (OR=1.30, 95% C.I. = 1.04–1.63), 41% for breathlessness during the day (OR=1.41, 95% C.I. = 1.13–1.76), and 23% for dyspnea on exertion (OR = 1.23, 95% C.I. = 1.09–1.39). Nevertheless, the roles of PM₁₀ versus NO₂ in the observed associations could not be ascertained, as NO₂ concentrations were strongly correlated with PM₁₀ levels.

The SAPALDIA investigators also examined lung function (FEV₁ and FVC) in study participants in relation to several air pollutants, controlling for age, sex, height, weight, atopy, educational level, nationality, smoking status (never, ever, and current), workplace exposures, residential gas stove, serious respiratory infection before age 5, and other potentially covariates (Ackermann-Lieblich *et al.*, 1997). Statistically significant decrements in both indices of lung function were found in relation to annual mean levels of PM₁₀, sulfur dioxide, and nitrogen dioxide, with the strongest effects being related to PM₁₀ (-3.4% for FVC and -1.6% for FEV₁ in healthy never-smokers, per 10µg/m³ annual average PM₁₀). The mean PM₁₀ concentration in this study was 21.2µg/m³, with a range of 10.1 – 33.4 µg/m³.

In summary, the evidence of particle effects in these studies of morbidity in relation to chronic exposures is not as consistent as for mortality. Overall, there is evidence of a particle-related effect on chronic morbidity, as measured by chronic respiratory symptoms and lung function. However, it is not possible, based on current evidence, to identify which size fractions or specific constituents are likely to be most influential (USEPA, 2009; OEHHA, 2001).

6.1.2 PM_{2.5}

The health effects of PM_{2.5} have been extensively studied and reviewed in recent years (WHO, 2013; USEPA, 2012, 2009; NEPC 2010). There is a large database that supports a causal association between exposure to PM_{2.5} and a range of both short-term and long-term mortality and morbidity outcomes. In 2013 a large European cohort study found that long-term exposure to PM_{2.5} is linked to increases in cancer deaths (Cesaroni *et al.*, 2013).

6.1.2.1 Short-term effects

In recent years there has been a substantive increase in studies showing associations between particles and cardiovascular effects. Epidemiological studies that examined the association between

PM₁₀, PM_{2.5} and mortality have provided strong evidence for particle-related cardiovascular effects. Multicity studies have found consistent, positive associations between short-term exposure to PM_{2.5} and cardiovascular mortality ranging from 0.47 to 0.85% at mean 24-hour average PM_{2.5} concentrations above 13µg/m³. These associations were reported at short lags (0-1 days). Although examinations of potential confounders of the PM_{2.5}-cardiovascular mortality relationship are limited, the observed associations are supported by PM₁₀-mortality studies, which found that particle risk estimates remained robust to the inclusion of co-pollutants in models. Although the overall effect estimates reported in the multicity studies are consistently positive, it should be noted that a large degree of variability exists between cities when examining city-specific effect estimates potentially due to differences between cities and regional differences in PM_{2.5} composition.

A study conducted in four Australian cities (Brisbane, Melbourne, Perth and Sydney), found statistically significant associations between particles and all-cause mortality. Meta-analyses carried out for three cities yielded estimates for the increase in the daily total number of deaths of 0.2% (-0.8% to 1.2%) for a 10µg/m³ increase in PM₁₀ concentration, and 0.9% (-0.7% to 2.5%) for a 10µg/m³ increase in PM_{2.5} concentration.

A study conducted in Melbourne found statistically significant positive associations between the particle measures considered and all cause and respiratory mortality in the warm season (November-March). A 10µg/m³ increase in 24-hour PM_{2.5} in the warm season was associated with a 0.38% (95% CI, 0.06–0.70%) increase in risk of death for all-cause mortality and a 1.18% (95% CI, 0.05–2.32%) increase in risk for respiratory mortality. For PM₁₀, a 10µg/m³ was associated with an increased risk of 0.18% (95% CI, 0.03–0.33%) for all-cause mortality and 0.59% (95% CI, 0.06–1.13%) for respiratory mortality. Statistically significant associations were also found in the 65+ age group in the warm season (Simpson et al. 2000). A study of ambient levels of air pollution in Melbourne and daily mortality due to all causes, respiratory and cardiovascular disease found that after controlling for the effects of weather and other confounding factors, air pollution in Melbourne is associated with increases in daily mortality. Associations were found between mortality and O₃, NO₂, CO and PM_{2.5} with the strongest and most robust relationships being observed for ozone and nitrogen dioxide with smaller increases in mortality being noted with PM_{2.5} (EPA Victoria, 2000).

An evaluation of the epidemiological literature indicates consistent positive associations between short-term exposure to PM_{2.5} and all-cause, cardiovascular- and respiratory-related mortality. The evaluation of multicity studies found that risk estimates for all-cause (non-accidental) mortality ranged from 0.29% to 1.21% per 10µg/m³ increase in 24-hour average PM_{2.5} at lags of 1 and 0–1 days. These consistent effects were observed in study locations with mean 24-hour average PM_{2.5} concentrations as low as 13µg/m³. Cardiovascular-related mortality risk estimates were found to be similar to those for all-cause mortality whereas, the risk estimates for respiratory-related mortality were consistently larger: 1.01–2.2% using the same lag periods and averaging indices. Results of studies in the US showed regional and seasonal patterns in PM_{2.5} risk estimates with the greatest effect estimates occurring in the eastern U.S. and during the spring. Of the studies evaluated by the USEPA in their most recent review (USEPA, 2009) no U.S.-based multicity studies conducted a detailed analysis of the potential confounding of PM_{2.5} risk estimates by gaseous pollutants. However Burnett et al. (2004) found mixed results, similar to those observed for PM₁₀, with possible confounding by NO₂ when analysing gaseous pollutants in a multicity Canadian-based study. An examination of effect modifiers (e.g., demographic and socioeconomic factors), specifically air conditioning use as an indicator for decreased pollutant penetration indoors, has suggested that PM_{2.5} risk estimates increase as the percent of the population with access to air conditioning decreases (USEPA, 2009). Collectively, the USEPA (2009) concluded that the epidemiological literature provides evidence that a causal relationship is likely to exist between short-term exposures to PM_{2.5} and mortality. This finding is supported by WHO (2006) and Cal EPA (2001).

Franklin et al. (2007) analysed 27 cities across the U.S. that had PM_{2.5} monitoring and daily mortality data for at least 2 year of a 6 year period 1997 to 2002. The mortality data up to year 2000 were obtained from the National Centre for Health Statistics, while the 2001–2002 data were obtained from six states (California, Michigan, Minnesota, Pennsylvania, Texas, and Washington), resulting in 12 out of the 27 cities having data up to 2002. The start year for each city included in the study was set at 1999, except for Milwaukee, Wisconsin (1997) and Boston, Massachusetts (1998), as PM_{2.5} data was available in these two cities. In the case crossover analysis in each city, control days for each death were chosen to be every third day within the same month and year that death occurred in order to reduce auto-correlation. The first stage regression examined the interaction of effects with age and gender, while the second stage random effects model combined city-specific PM_{2.5} risk estimates and examined possible effect modifiers using city-specific characteristics (e.g., prevalence of central air conditioning and geographic region). For all of the mortality categories, the estimates for lag 1-day showed the largest estimates. The combined estimates at lag 1 day were: 1.2% (CI: 0.29–2.1), 0.94% (CI: -0.14 to 2.0), 1.8% (CI: 0.20–3.4), and 1.0% (CI: 0.02–2.0) for all-cause, cardiovascular, respiratory, and stroke deaths, respectively, per 10µg/m³ increase in 24-hour average PM_{2.5}.

Zanobetti and Schwartz (2009) analysed PM_{2.5} associations with all-cause, cardiovascular disease, myocardial infarction, stroke, and respiratory mortality for the years 1999–2005 in the US. The overall combined excess risk estimates were: 0.98% (0.75, 1.22) for all-cause; 0.85% (0.46, 1.24) for cardiovascular disease, 1.18% (0.48, 1.89) for myocardial infarction; 1.78% (0.96, 2.62) for stroke, and 1.68% (1.04, 2.33) for respiratory mortality for a 10µg/m³ increase in PM_{2.5} at lag 0–1 days. When the risk estimates were combined by season, the spring estimates were the largest for all-cause and for all of the cause-specific mortality outcomes examined. The risk estimate for all-cause mortality for the spring was 2.57% (1.96–3.19) with the estimates for the other seasons ranging from 0.25% to 0.95%. When examining cities that had both PM_{2.5} and PM_{10-2.5} data (i.e., 47 cities), the addition of PM_{2.5-10} in the model did not alter the PM_{2.5} estimates substantially, only decreasing slightly from 0.94% in a single pollutant model to 0.77% in a co-pollutant model with PM_{2.5-10}. When the risk estimates were combined by climatic regions, the estimated PM_{2.5} risk for all-cause mortality were similar (all above 1% per 10µg/m³ increase) for all the regions except for the “Mediterranean” region (0.5%) which include cities in California, Oregon and Washington, though the estimates in that region were significantly heterogeneous.

Multicity studies that examined the association between PM_{2.5} and respiratory mortality, Franklin et al. (2007) and Zanobetti and Schwartz (2009), found consistent, positive associations between short-term exposure to PM_{2.5} and respiratory mortality ranging from 1.67 to 2.20% at lag 0–1 days for mean 24-hour PM_{2.5} average concentrations above 13µg/m³.

Although examinations of potential confounders of the PM_{2.5}-respiratory mortality relationship are limited, the observed associations are supported by PM₁₀-mortality studies, which found that particle risk estimates remained robust to the inclusion of co-pollutants in models. The associations are consistent with those presented by Ostro et al. (2006) in a study that examined the PM_{2.5}-mortality relationship in 9 California counties (2.2% [95% CI: 0.6–3.9] per 10µg/m³). An evaluation of studies that examined additional lag structures of associations found smaller respiratory mortality effect estimates when using the average of lag days 1 and 2 (1.01% [95% CI: -0.03 to 2.05] per 10µg/m³) (Franklin et al., 2008), and associations consistent with those observed at lag 0–1 days when examining single day lags, specifically lag 1 (previous day) (1.78% [95% CI: 0.2–3.36]). Although the overall effect estimates reported in the multicity studies evaluated are consistently positive, it should be noted that a large degree of variability exists between cities when examining city-specific effect estimates both in the US and in Australia, potentially due to differences between cities and regional differences in PM_{2.5} composition.

A large body of evidence from studies of the effect of PM_{2.5} on hospital admissions and emergency department visits for cardiovascular diseases has shown that associations with PM_{2.5} are consistently positive with the majority of studies reporting increases in hospital admissions or emergency department visits ranging from a 0.5 to 3.4% per 10µg/m³ increase in PM_{2.5}. A large U.S.-based multicity study reported excess risks in the range of approximately 0.7% with the largest excess risks in the North East (1.08%) and in the winter (1.49%), providing evidence of regional and seasonal heterogeneity (Bell et al., 2008; Dominici et al., 2006). Weak or null findings for PM_{2.5} have been observed in two single-city studies both conducted in Washington State (Slaughter et al., 2003; Sullivan et al., 2007) and may be explained by this heterogeneity. Weak associations were also reported in Atlanta for PM_{2.5} and cardiovascular disease emergency department visits, with PM_{2.5} traffic components being more strongly associated with cardiovascular disease emergency department visits (Tolbert et al., 2007). The results of multicity studies conducted outside the U.S. and Canada have shown positive associations with PM_{2.5}. Studies of specific cardiovascular disease outcomes indicate that ischemic heart disease and congestive heart failure may be driving the observed associations. Although estimates from studies of cerebrovascular diseases are less precise and consistent, ischemic diseases appear to be more strongly associated with PM_{2.5} compared to haemorrhagic strokes. The available evidence suggests that these effects occur at very short lags (0-1 days), although effects at longer lags have rarely been evaluated. Overall, the results of these studies provide support for associations between short-term PM_{2.5} exposure and increased risk of cardiovascular hospital admissions in areas with mean concentrations ranging from 7 to 18µg/m³.

A number of studies have found consistent associations between PM_{2.5} and hospital admissions and emergency department visits for respiratory disease with effect estimates in the range of ~1–4% per 10µg/m³ increase in PM_{2.5}. These associations have been observed in areas with mean 24-hour PM_{2.5} concentrations between 6.1 and 22µg/m³. Further, studies have focused on increasingly specific disease endpoints such as asthma, COPD and respiratory infection. The strongest evidence of an association comes from large multicity studies of COPD, respiratory tract infection and all respiratory diseases among Medicare recipients (65+ years old) (Dominici et al., 2006; Bell et al., 2008). Studies of children have also found evidence of an effect of PM_{2.5} on hospital admissions for all respiratory diseases, including asthma and respiratory infection. One of the strongest associations observed in the Atlanta based SOPHIA study was between PM₁₀ and paediatric asthma visits; PM_{2.5} makes up a large proportion of PM₁₀ in Atlanta (Peel et al., 2005); Positive associations between PM_{2.5} (or PM₁₀) and hospital admissions for respiratory infection are supported by animal toxicological studies which add to previous findings of increased susceptibility to infection following exposure to PM_{2.5}. These include studies demonstrating reduced clearance of bacteria (*Pseudomonas*, *Listeria*) or enhanced pathogenesis of viruses (influenza, RSV) after exposure to diesel exhaust or residual oil fly ash.

The majority of the studies that examined the association between PM_{2.5} and respiratory symptoms and medication use found a consistent increase in asthmatic children (effect estimates ranging from ~1.0–1.3) with less consistent evidence for an association in asthmatic adults in cities with mean 24-hour average PM_{2.5} concentrations ranging from 6.1 to 19.2µg/m³. An evaluation of epidemiological studies that examined specific physiologic alterations in the respiratory health of asthmatic children (i.e., pulmonary function and pulmonary inflammation) found a decrease in forced expiratory volume (FEV₁) ranging from 1-3.4% per 10µg/m³ increase in PM_{2.5}; and an increase in eNO ranging from 0.46 to 6.99ppb, respectively. In addition, epidemiological studies that examined the effect of short-term exposure to PM_{2.5} on respiratory hospital admissions and emergency department visits found consistent associations (ranging from ~0 to 5%) for respiratory diseases (e.g. COPD and respiratory infections) among older adults, but less consistent effects were reported for asthma hospital admissions and emergency department visits. These respiratory hospital admissions and emergency department visit studies were conducted in cities with mean 24-hour average PM_{2.5} concentrations ranging from 13.8 to 18.9µg/m³.

The evidence for PM_{2.5} induced respiratory effects is strengthened by similar associations found for hospital admissions and emergency department visit for PM₁₀, along with the consistent positive associations observed between PM_{2.5} and respiratory mortality in multicity studies. Panel studies also indicate associations with PM_{2.5} and respiratory symptoms, pulmonary function, and pulmonary inflammation among asthmatic children.

Controlled human exposure studies in adults demonstrating increased markers of pulmonary inflammation following diesel exhaust and other traffic-related exposures, oxidative responses to diesel exhaust and wood smoke and exacerbations of allergic responses and allergic sensitization following exposure to diesel exhaust particles add further support for these effects (USEPA, 2009). Some controlled human exposure studies have reported small decrements in various measures of pulmonary function following controlled exposures to PM_{2.5}. Numerous toxicological studies demonstrating a wide range of responses provide biological plausibility for the associations between PM_{2.5} and respiratory morbidity observed in epidemiological studies. Altered pulmonary function, mild pulmonary inflammation and injury, oxidative responses, Airway hyperresponsiveness in allergic and non-allergic animals, exacerbations of allergic responses and increased susceptibility to infections were observed in a large number of studies involving exposure to concentrated ambient particles, diesel exhaust, other traffic-related particles and wood smoke. The numerous and wide range of respiratory responses observed in both the human clinical and toxicological studies provide biological plausibility for an association between short-term exposure to PM_{2.5} and respiratory morbidity. The USEPA, (2009) concluded that the consistent and coherent results found in the epidemiological, human clinical, and toxicological literature provide sufficient evidence that a causal relationship is likely to exist between short-term exposures to ambient concentrations of PM_{2.5} and respiratory morbidity.

Epidemiological studies of asthmatic children have found increases in respiratory symptoms and asthma medication use associated with higher PM_{2.5} or PM₁₀ concentrations. Associations with respiratory symptoms and medication use are less consistent among asthmatic adults, and there is no evidence to suggest an association between respiratory symptoms with PM_{2.5} among healthy individuals (USEPA, 2009). In addition, respiratory symptoms have not been reported following controlled exposures to PM_{2.5} among healthy or health-compromised adults.

Although epidemiological studies of pulmonary function and PM_{2.5} have yielded somewhat inconsistent results, the majority of studies have found an association between PM_{2.5} concentration and FEV₁, PEF, and/or MMEF. In asthmatic children, a 10 µg/m³ increase in PM_{2.5} is associated with a decrease in FEV₁ ranging from 1-3.4%. A limited number of controlled human exposure studies have reported small decreases in arterial oxygen saturation and MMEF following exposure to PM_{2.5} concentrated ambient particles with more pronounced effects observed in healthy adults than in asthmatics or older adults with COPD (USEPA, 2009). In toxicological studies, changes in pulmonary function have been observed in healthy and compromised rodents after inhalation exposures to concentrated ambient particles from a variety of locations or diesel exhaust.

A large body of evidence, primarily from toxicological studies, indicates that various forms of particles induce oxidative stress, pulmonary injury, and inflammation. Notably, concentrated ambient particles from a variety of locations induce inflammatory responses in rodent models, although this generally requires multiday exposures. The toxicology findings are consistent with several epidemiologic studies of PM_{2.5} and the inflammatory marker eNO, which reported statistically significant, positive effect estimates with some inconsistency in the lag times and use of medication. In asthmatic children, a 10 µg/m³ increase in PM_{2.5} is associated with an increase in eNO ranging from 0.46 to 6.99ppb.

Several new controlled human exposure studies report traffic or diesel-induced increases in markers of inflammation (e.g., neutrophils and IL-8) in airway lavage fluid from healthy adults. Some studies have

provided additional evidence in support of a pulmonary oxidative response to diesel exhaust in humans, including induction of redox-sensitive transcription factors and increased urate and GSH concentrations in nasal lavage. In addition, exposure to wood smoke has been demonstrated to increase the levels of eNO and malondialdehyde in breath condensate of healthy adults (Barregard et al., 2008). Preliminary findings indicate little to no pulmonary injury in humans following controlled exposures to fine urban traffic particles or diesel exhaust, in contrast to a number of toxicological studies demonstrating injury with concentrated ambient particles or diesel exhaust.

6.1.2.2 Long-term effects

The earlier studies on the long-term effects of PM_{2.5} on mortality – the Six Cities Study (Dockery et al., 1993) and the American Cancer Society (ACS) study (Pope et al., 2002) – have been pivotal in the development of air quality standards and guidelines worldwide. These studies have been updated several times with systemic increases in the number of years of analysis and deaths that were followed in these cohorts and in the statistical approaches used in the analysis (Laden et al., 2006; Lepuele et al., 2012; Krewski et al., 2009). These reanalyses continue to find a consistent, statistically significant association between long-term exposure to PM_{2.5} and the risk of mortality. The magnitude of the effects estimate (the mortality effect per unit of exposure) remains consistent with that of the original study (WHO, 2013). Using the 51 cities from the ACS study Pope et al., (2009) reported that reductions in PM_{2.5} across the metropolitan regions between 1980 and 2000 were strongly associated with increases in life expectancy after controlling for other risk factors.

A large number of new prospective cohort studies from Asia, Canada, Europe and the US have been reported since 2005 (summarised in WHO, 2013). These studies provide additional evidence of the effects of long-term exposure to PM_{2.5} on mortality. These effects have been observed at lower concentrations than previously studied and there is still no evidence of a threshold below which adverse effects do not occur. These studies have been undertaken in areas that cover a variety of environmental settings, PM mixtures, baseline health conditions, socioeconomic settings and personal characteristics. Given the consistency in the findings of these studies WHO (2013) and USEPA (2012; 2009) have determined that it is appropriate to extrapolate the findings of these studies to other regions. The risk of ischemic heart disease has particularly strong associations with PM_{2.5}.

Hoek et al (2013) conducted a systematic review of the literature on the long-term effects of air pollution on all cause, cardiovascular and respiratory mortality. Where more than 5 studies were identified a meta-analysis was conducted to obtain an overall effects estimate for each outcome. The authors identified a number of cohort studies conducted in various parts of the world that found associations between PM_{2.5} and PM₁₀ and all cause, cardiovascular and respiratory mortality. The effects estimates identified per 10 µg/m³ increase in annual average PM_{2.5} were 6% all cause, 11% cardiovascular and 3% respiratory mortality. For PM₁₀ a 3.5% increase in all-cause mortality per 10 µg/m³ increase in annual average PM₁₀ was found. There was significant heterogeneity in the effects estimates from individual studies which was thought to be due to differences in particle composition, indoor exposures as well as population and baseline health status of the exposed populations.

The evidence for a biological mechanism, derived from both epidemiological and toxicological studies, has increased substantively in recent years and indicates that exposure to PM_{2.5} is associated with systemic inflammation, oxidative stress and alteration of the electrical processes in the heart (Brooks et al., 2010). Epidemiological studies show variations in cardiovascular biomarkers such as C-reactive protein and fibrinogen. These biomarkers have been consistently linked to subsequent cardiovascular disease and death (WHO, 2013).

Recent studies have also shown the effects of long-term exposure to PM_{2.5} on diseases other than cardiovascular and respiratory diseases (WHO, 2013). Evidence suggests effects on diabetes,

neurological development in children and neurological disorders in adults (Ruckerl et al., 2011). Epidemiological studies in Germany (Kramer et al., 2010) and Denmark (Anderson et al., 2012; Raaschou-Nielsen et al., 2013) have all found strong associations between exposure to PM_{2.5} and diabetes. These findings have been supported by mechanistic studies (WHO, 2013).

Studies on the effects of PM_{2.5} on birth outcomes have been studied in a number of cohort studies (Brauer et al., 2007; Gehring et al., 2010; MacIntyre et al., 2011; Morgenstern et al., 2007). Evidence is accumulating for PM_{2.5} effects on low birth weight and infant mortality, especially due to respiratory causes during the post-neonatal period. The mean PM_{2.5} concentrations during the study periods ranged from 5.3–27.4 µg/m³ with effects becoming more precise and consistently positive in locations with mean PM_{2.5} concentrations of 15 µg/m³ and above (USEPA, 2009). Exposure to PM_{2.5} was usually associated with greater reductions in birth weight than exposure to PM₁₀. The evidence from a few studies that investigated PM₁₀ effects on foetal growth, which reported similar decrements in birthweight, provide consistency for the PM_{2.5} associations observed and strengthen the interpretation that particle exposure may be causally related to reductions in birth weight.

The epidemiological literature does not consistently report associations between long-term exposure to particles and preterm birth, growth restriction, birth defects or decreased sperm quality (USEPA, 2009). Toxicological evidence supports an association between PM_{2.5} and PM₁₀ exposure and adverse reproductive and developmental outcomes, but provided little mechanistic information or biological plausibility for an association between long-term particle exposure and adverse birth outcomes (e.g., low birth weight or infant mortality). Overall, the USEPA concluded that the epidemiological and toxicological evidence is suggestive of a causal relationship between long-term exposures to PM_{2.5} and reproductive and developmental outcomes.

6.1.3 Coarse fraction – PM_{2.5-10}

In recent years there has been a significant amount of research into the health effects of coarse particles. These studies have been conducted in urban areas and well as areas affected by desert dust. The WHO REVIHAAP review concluded that there is new evidence that suggests that short-term exposure to coarse particles (including crustal material) are associated with adverse respiratory and cardiovascular effects on health including premature mortality (WHO, 2013). They also concluded that toxicological studies have shown that coarse particles can be as toxic as PM_{2.5} on a mass basis. The difference in risk between coarse and fine PM can, at least partially, be explained by differences in uptake and biological mechanisms (WHO, 2013).

In 2009 the USEPA completed their Integrated Science Assessment (ISA) for the review of the standards for particles (USEPA, 2009). They concluded at that time that there was 'suggestive evidence of a causal relationship between short-term exposure to coarse particles and cardiovascular and respiratory health effects and mortality'. There was not enough evidence at that time for the EPA to draw any conclusions about the long-term health effects associated with exposure to coarse particles. Since that time the evidence of short-term effects of coarse particles on cardiorespiratory health and mortality has increased substantively.

In 2012, the USEPA completed their provisional assessment of the recent literature on the health effects of PM (USEPA, 2012). This review focussed on studies conducted in the US and Canada. In this review a number of new studies investigating the association between exposure to coarse particles and adverse health effects were identified. With respect to long-term studies, two studies by Puett et al., (2011; 2009) were identified. The USEPA concluded that the results of these studies did not change their conclusion from the 2009 ISA that there was insufficient evidence to draw any conclusions about the causality associated with the health effects associated with long-term exposure to coarse particles. The USEPA concluded that evidence from new studies on the short-term effects of coarse

particles is suggestive of a causal relationship between short-term exposures to PM_{2.5-10} and both respiratory and cardiovascular effects.

6.1.3.1 Short-term effects

A number of studies have examined the short term effects of coarse particles on both mortality and morbidity. These are summarised in the following sections.

6.1.3.1.1 Mortality

Several studies have examined the association between exposure to coarse particles and increases in mortality from cardiovascular (Atkinson et al., 2010; Chen et al., 2011; Malig and Ostro, 2009; Mallone et al., 2011), respiratory (Chen et al., 2011) and all-cause mortality (Meister, Johansson and Forsberg, 2012; Tobias et al., 2011).

The study by Malig and Ostro (2009) examined the association between daily coarse particles and all cause and cardiovascular mortality across 15 Californian counties. To address exposure misclassification, case deaths were limited to those residing within 20km of an air monitoring station. County estimates were pooled in a random-effects meta-analysis to create overall study estimates. The effect of race and educational status were also analysed. The study found an increase in both all-cause and cardiovascular mortality associated with exposure to coarse particles. The strongest effects were 0.7% increase in all-cause mortality and 1.3% increase in cardiovascular mortality associated with a 10µg/m³ increase in daily PM_{2.5-10} at a 2-day lag. Greater effects were found for Hispanics and people without high school education. The results were not affected by adjustment for fine particles or restricting the analysis to a 10km radius from an air monitoring station.

A study by Perez et al., (2009) found an association between PM_{2.5-10} and cardiovascular and cerebrovascular mortality in Barcelona, Spain. A 10µg/m³ increase in daily PM_{2.5-10} was associated with increases in both cardiovascular and cerebrovascular mortality with Odds Ratios of 1.059 (95%CI: 1.026-1.094) and 1.098 (95%CI: 1.030-1.171), respectively.

A national study conducted in the US (Zanobetti and Schwartz, 2009) examined the association between both PM_{2.5} and PM_{2.5-10} and all-cause mortality as well as mortality from cardiovascular disease, myocardial infarction, stroke and respiratory disease. This study found statistically significant associations between coarse particles across the 47 cities studies with the strongest effects found for respiratory mortality: a 1.2% increase per 10µg/m³ increase in daily PM_{2.5-10}. Controlling for other pollutants did not change the observed effects.

Other studies including a large study conducted in Mediterranean cities in Europe (Samoli et al., 2013) found positive but not statistically significant associations between exposure to coarse particles and all-cause, cardiovascular and respiratory mortality.

Studies looking at PM_{2.5-10} and cardiovascular mortality have also found associations with this size fraction (USEPA, 2009). Zanobetti and Schwartz (2009) examined PM_{2.5-10} mortality associations in 47 U.S. cities and found evidence for cardiovascular mortality effects (0.32% [95% CI: 0.00–0.64] per 10 µg/m³ at lag 0–1 days) similar to those reported for all-cause (non-accidental) mortality (0.46% [95% CI: 0.21–0.67] per 10µg/m³). Seasonal (i.e., larger in spring and summer) and regional differences in PM_{2.5-10} cardiovascular mortality risk estimates were observed in this study. The study found a statistically significant association between the computed PM_{2.5-10} and all-cause, cardiovascular disease, stroke, and respiratory mortality.

The combined estimate for the 47 cities using the average of 0- and 1-day lag PM_{2.5-10} for all-cause mortality was 0.46% (95% CI: 0.21–0.71) per 10µg/m³ increase with the estimate obtained using the

distributed lag model being smaller (0.31% [95% CI: 0.00–0.63]). The seasonal analysis showed larger risk estimates in the spring for all-cause (1.01%) and respiratory mortality (2.56%), however, for cardiovascular mortality, the estimates for spring (0.95%) and summer (1.00%) were comparable. Zanobetti and Schwartz (2009) also found an association between PM_{2.5-10} and respiratory mortality (1.16% [95% CI: 0.43, 1.89] per 10µg/m³ at lag 0–1 days), with effect estimates somewhat larger than those reported for all-cause (non-accidental) mortality (0.46% [95% CI: 0.21, 0.671] per 10µg/m³). In addition, Zanobetti and Schwartz (2009) reported seasonal (i.e., larger in spring) and regional differences in PM_{2.5-10} respiratory mortality risk estimates.

A few single-city studies evaluated also reported associations, albeit somewhat larger than the multicity study, between PM_{2.5-10} and cardiovascular mortality in Phoenix, Arizona (Wilson et al., 2007) (3.4–6.6% at lag 1) and Vancouver, Canada (Villeneuve et al., 2003) (5.4% at lag 0). The difference in the PM_{10-2.5} risk estimates observed between the multi- and single city studies could be due to a variety of factors including differences between cities and compositional differences in PM_{2.5-10} across regions (USEPA, 2009).

Single-city studies conducted in Atlanta, Georgia (Klemm et al., 2004) and Vancouver, Canada (Villeneuve et al., 2003) reported no associations between short-term exposure to PM_{2.5-10} and respiratory mortality. The difference in the results observed between the multi- and single- city studies could be due to a variety of factors including differences between cities and compositional differences in PM_{2.5-10} across regions. Only a small number of studies have examined potential confounding by gaseous co-pollutants or the influence of model specification on PM_{2.5-10} mortality risk estimates.

A study by Perez et al. (2008) investigated the association between Saharan dust events and the effects of PM_{2.5} and PM_{2.5-10} on daily mortality. Changes of effects between Saharan and non-Saharan dust days were assessed using a time-stratified case-crossover design involving 24,850 deaths between March 2003 and December 2004 in Barcelona, Spain. Saharan dust days were identified from back-trajectory and satellite images. Chemical speciation, but not an analysis for microbes or fungi, was conducted approximately once a week during the study period. On Saharan dust days, mean concentrations were 1.2 times higher for PM_{2.5} (29.9µg/m³) and 1.1 times higher for PM_{2.5-10} (16.4µg/m³) than on non-Saharan dust days. During Saharan dust days (90 days out of 602), the PM_{2.5-10} risk estimate was 8.4% (95% [CI: 1.5–15.8]) per 10µg/m³ increase at lag 1 day, compared with 1.4% (95% CI: -0.8% to 3.4%) during non-Saharan dust days. In contrast, there was not an additional increased risk of daily mortality for PM_{2.5} during Saharan dust days (5.0% [95% CI: 0.5–9.7]) compared with non-Saharan dust days (3.5% [95% CI: 1.6–5.5]). However, differences in chemical composition (i.e., PM_{2.5} was primarily composed of non-mineral carbon and secondary aerosols; whereas PM_{2.5-10} was dominated by crustal elements) did not explain these observations. Canadian studies have also shown an association between PM_{2.5-10} and mortality (Burnett et al., 2004; Villeneuve et al. 2003). The Burnett study found a 0.65% (CI: -0.10 to 1.4) increase in all-cause mortality per 10µg/m³ increase at lag 1 day. When both NO₂ and PM_{2.5-10} were included in the regression model, the PM_{2.5-10} effect estimate was reduced to 0.31% (95% CI: -0.49 to 1.1) per 10µg/m³ increase in 1-day lag PM_{2.5-10}. These risk estimates are similar to those reported for PM_{2.5}, which were also reduced upon the inclusion of NO₂ in the two-pollutant model, but to a greater extent, from 0.60% (95% [CI: -0.03 to 1.2]) to -0.1% (95% [CI: -0.86 to 0.67]). The study by Villeneuve et al. (2003) analysed the association between PM_{2.5}, PM_{2.5-10}, TSP, PM₁₀, SO₄²⁻, and gaseous co-pollutants in Vancouver, Canada, using a cohort of approximately 550,000 between 1986 and 1999. In this study PM_{2.5} and PM_{2.5-10} were directly measured using dichotomous samplers. The authors examined the association of each air pollutant with all-cause, cardiovascular, and respiratory mortality, but only observed statistically significant results for cardiovascular mortality at lag 0 for both PM_{2.5-10} and PM_{2.5}. They found that PM_{2.5-10} (5.4% [95% CI: 1.1–9.8] per 10µg/m³), was more strongly associated with cardiovascular mortality than PM_{2.5} (4.8% [95% CI: -1.9 to 12.0] per unit increase).

6.1.3.1.2 Morbidity

In contrast to the evidence for mortality, there are a number of studies that have found strong associations between hospital admissions and other morbidity outcomes with exposure to coarse particles. A study by Stafoggia et al., (2013) found that coarse particles were associated with hospital admissions for cardiovascular disease in 8 Mediterranean cities in Europe. A 0.73% increase in admissions for people ≥ 15 years was associated with a $10\mu\text{g}/\text{m}^3$ increase in daily $\text{PM}_{2.5-10}$. Associations were also found for PM_{10} and $\text{PM}_{2.5}$. The results indicate that the effects estimates for coarse particles for an equal increment in mass is around 40% higher than that observed for $\text{PM}_{2.5}$. Associations were also found for respiratory admissions at lag 1 with a 0.6% increase in admissions per $10\mu\text{g}/\text{m}^3$ increase in daily $\text{PM}_{2.5-10}$.

Several epidemiological studies report associations between $\text{PM}_{2.5-10}$ and hospital admissions for respiratory disease with the most consistent evidence among children (WHO, 2013; USEPA, 2009). Although a number of studies provide evidence of respiratory effects in older adults, an analysis of MCAPS data reports that weak associations of $\text{PM}_{2.5-10}$ with respiratory hospitalisations are further diminished after adjustment for $\text{PM}_{2.5}$. An examination of $\text{PM}_{2.5-10}$ mortality associations on a national scale found a strong association between $\text{PM}_{2.5-10}$ and respiratory mortality, but this association varied when examining city-specific risk estimates (Zanobetti and Schwartz, 2009). Additionally, co-pollutant analyses were not conducted in this study, and the associations observed are inconsistent with those reported in single-city studies. There is greater spatial heterogeneity in $\text{PM}_{2.5-10}$ compared to $\text{PM}_{2.5}$ and consequently greater potential for exposure measurement error in epidemiological studies relying on central site monitors. This exposure measurement error may bias effect estimates toward the null.

Mar et al. (2004) provide evidence for an association with increased respiratory symptoms in asthmatic children but not asthmatic adults. Consistent with this, controlled human exposures to $\text{PM}_{2.5-10}$ have not been observed to affect lung function or respiratory symptoms in healthy or asthmatic adults. However, increases in markers of pulmonary inflammation have been demonstrated in healthy volunteers. In these studies, an increase in neutrophils in BAL fluid or induced sputum was observed, with additional evidence of alveolar macrophage activation associated with biological components of $\text{PM}_{2.5-10}$ (i.e., endotoxin). Toxicological studies using inhalation exposures are still lacking, but pulmonary injury and inflammation have been observed in animals after IT exposure and both rural and urban $\text{PM}_{2.5-10}$ have induced these responses. In some cases, $\text{PM}_{2.5-10}$ from urban air was more potent than $\text{PM}_{2.5}$. $\text{PM}_{2.5-10}$ respiratory effects may be due to components other than endotoxin (Wegesser and Last, 2008).

Overall, the most compelling evidence comes from a number of epidemiological studies conducted in Canada and France showing statistically significant associations between respiratory emergency department visits or hospital admissions and short-term exposure to $\text{PM}_{2.5-10}$ (WHO, 2013). Effects have been observed in areas where the mean 24-hour average $\text{PM}_{2.5-10}$ concentrations ranged from 7.4 to $13.0\mu\text{g}/\text{m}^3$. The strongest relationships were observed among children, whereas studies of adults and older adults show less consistent evidence of an association.

While controlled human exposure studies have not observed an effect on lung function or respiratory symptoms in healthy or asthmatic adults in response to exposure to $\text{PM}_{2.5-10}$, healthy volunteers have exhibited increases in markers of pulmonary inflammation. Toxicological studies using inhalation exposures are still lacking, but pulmonary injury has been observed in animals after intra-tracheal exposure to both rural and urban $\text{PM}_{2.5-10}$, which may not be entirely attributed to endotoxin. Overall, the USEPA (2009) concluded that epidemiological studies, along with the limited number of controlled human exposure and toxicological studies that examined $\text{PM}_{2.5-10}$ and respiratory outcomes, provide evidence that is suggestive of a causal relationship between short-term $\text{PM}_{2.5-10}$ exposures and respiratory effects.

A study by Yeatts et al., (2007) found that exposure to PM_{2.5-10} is associated with changes in heart rate variability, blood lipids and circulating eosinophils in adults with asthma. A further study by Brook et al., 2014, found that exposure to PM_{2.5-10} was associated with rapid elevation in blood pressure and heart rate during the exposure period. The authors concluded that this may be due to the triggering of an autonomic imbalance and that the findings of this study contributed to the biological plausibility that coarse particles could contribute to triggering acute cardiovascular events.

Coarse particles were also associated with increased hospital admissions for cardiovascular and respiratory conditions in a large study conducted in Southern Europe (Staffagio et al, 2013b). A 6.3µg/m³ increase in 24-hour average PM_{2.5-10} was associated with a 0.46% increase in cardiovascular hospital admissions. Stronger effects were found for respiratory admissions. A study by Peng et al., (2009) found that after controlling for PM_{2.5} no statistically significant associations were found for hospital admissions for cardiovascular and respiratory causes with PM_{2.5-10} among Medicare Patients in the US.

A multicentre European panel study found that exposure to coarse particles was associated with an increase in symptoms in people with asthma and COPD (Karaktasani et al., 2012). A 10µg/m³ increase in PM_{2.5-10} with a 0.6 to 0.7% increase in symptoms and limitation in walking. The panel was conducted in 4 European cities with daily symptoms being recorded for a period of 6 months.

A study in Hong Kong found that coarse particles were associated with emergency hospital admissions for respiratory disease (Qui et al., 2012). A 10.9 µg/m³ increase in PM_{2.5-10} (4-day moving average) was associated with a 1.94% increase in emergency hospital admissions for respiratory disease. The association was not affected by controlling for PM_{2.5}.

6.1.3.2 Toxicology of Coarse Particles.

6.1.3.2.1 Cardiovascular and Systemic Effects

Toxicological studies on the cardiovascular effects of inhaled particles have focused on the following areas: (a) heart rate (HR) and heart rate variability (HRV); (b) cardiac arrhythmias; (c) cardiac ischemic disease; (d) cardiac contractility; (e) vasomotor effects; (f) blood pressure; (g) systemic inflammation; (h) systemic and cardiovascular oxidative stress; and (i) effects on the clotting system. These areas of investigation have been largely stimulated by the known epidemiological associations between exposure to PM₁₀ and PM_{2.5} and increased mortality and morbidity from cardiovascular and respiratory disease.

Relatively little mechanistic research regarding the epidemiological association between coarse particle exposure and effects on HR and HRV have been performed.

A two-hour inhalation exposure of healthy and mildly asthmatic adults (with intermittent exercise) to concentrated ambient urban coarse particles (mean 2 hour time weighted average of 157 µg/m³) resulted in small, but statistically significant, increases in HR and decreases in HRV in both groups (Gong et al., 2004). Cardiac ectopy was not increased and nor were there any clinical effects on the respiratory system.

A decrease in overall HRV was demonstrated at 20 hours post-exposure to concentrated ambient urban coarse particles in healthy young adult humans (2-hour exposure at 89 µg/m³ mass median aerodynamic diameter (MMAD) 3.9 µm, with intermittent exercise) (Graff et al., 2009). There was evidence of mild post-exposure pulmonary inflammation in this study.

Overall there is limited evidence that inhalation of concentrated coarse particles can have mild effects on HR and HRV in humans. There is no experimental information regarding the mode of action of these effects.

Sub-acute daily intra-pharyngeal instillation of urban coarse particles (Ottawa ambient particles, EHC-93; total of 8 mg/kg over 5 days and 16 mg/kg over 4 weeks) resulted in the inhibition of acetylcholine-induced, endothelium-dependent, nitric oxide-mediated carotid artery relaxation, up-regulation of markers of systemic inflammation (serum IL-6) and pulmonary inflammation in New Zealand White rabbits (Tanagawa et al., 2008). This provides some evidence to support the hypothesis that coarse particles may impair the arterial vasodilation response.

Controlled acute (five 130-min exposures at rest) inhalation exposure of healthy humans to ambient coarse particles ($200 \mu\text{g}/\text{m}^3$) resulted in increases in total peripheral blood leukocytes, and neutrophils at 24 hours post-exposure (Behbod et al., 2013). The endotoxin content of the particles is believed to have contributed to these effects.

Similar controlled healthy human acute inhalation exposure to rural ambient coarse particles ($76.2 \pm 51.5 \mu\text{g}/\text{m}^3$, single 2 hour exposure at rest) were associated with increased endothelial progenitor cell levels at 2 and 20 hours post-exposure (Brook et al., 2013). It is hypothesized that this effect is due to an acute systemic endothelial injury effect.

Marginal decreases (by 32.9% from baseline per $10 \mu\text{g}/\text{m}^3$ increase in ambient coarse particles concentration) in blood tissue plasminogen activator (involved in fibrinolysis and the breakdown of blood clots) were present 20 hours after a 2-hour inhalation exposure to concentrated ambient coarse particles ($89.0 \mu\text{g}/\text{m}^3$) (Graff et al., 2009). Consumption of mediators involved in the breakdown of clots/fibrinolysis provides some indirect evidence that exposure to concentrated ambient coarse particles may result in increased blood clotting tendency.

Intra-tracheal instillation of urban (Dusseldorf, Germany) coarse particles (known to contain substantial amounts of iron, nickel, and vanadium; $10 \mu\text{g}/\text{mouse}$ or approximately $400\text{-}500 \mu\text{g}/\text{kg}$) into mice (C57BL/6) resulted in decreases in bleeding time (decreased 32%), prothrombin time (decreased 13%), active partial thromboplastin time (decreased 16%) and ferric chloride-induced left coronary artery occlusion time at 24 hours post exposure time. These effects were associated with elevated platelet counts, and levels of clotting Factors II, VIII and X at 24-hour hours post exposure (Muttu et al., 2007). These pro-thrombotic effects are dependent upon IL-6 production. The authors concluded that exposure to particulate matter triggers IL-6 production by alveolar macrophages, resulting in reduced clotting times, intravascular thrombin formation, and accelerated arterial thrombosis (Muttu et al., 2007).

Intra-tracheal instillation of urban (Barcelona, Spain and Prague, Czech Republic) coarse particles ($7 \text{ mg}/\text{kg}$) was associated with pro-thrombotic changes at 24-hour post-exposure in spontaneously hypertensive rats that were characterized by elevated plasma fibrinogen levels (Gerlof-Nihland et al., 2009). It is notable that in this study, the potency of coarse particles was higher than that of fine particles as were particles that contained high levels of polycyclic aromatic hydrocarbons.

Brief inhalation of coarse particles from a rural location elicited an increase in EPCs that persisted for at least 20 hours. The underlying mechanism responsible may reflect a systemic reaction to an acute endothelial injury and/or a circulating EPC response to sympathetic nervous system activation.

6.1.3.2.2 Respiratory Effects

Toxicological studies on the respiratory effects of inhaled coarse particles have focused on the following areas: (a) induction of respiratory clinical signs; (b) effects on pulmonary function; (c) induction of inflammation within the respiratory tract.

Inhalation by healthy and mildly asthmatic human volunteers of concentrated urban coarse particles (mean 157 $\mu\text{g}/\text{m}^3$) for two hours did not induce changes in pulmonary function as measured using spirometry and pulse oximetry (Gong et al., 2004) or any evidence of inflammation of the respiratory tract.

Healthy young adult humans exposed to concentrated urban coarse particles (mean 89 $\mu\text{g}/\text{m}^3$) for 2 hours with intermittent exercise did not have any substantive changes to pulmonary functions (Graff et al., 2009). However mild increases in markers of pulmonary inflammation (elevated neutrophils in bronchial lavage fluid) were observed at 20 hours post-exposure (Graff et al., 2009).

Healthy young adult humans exposed to concentrated urban coarse particles (mean 89 $\mu\text{g}/\text{m}^3$) for 2 hours with intermittent exercise displayed mild increases. Healthy human volunteers who inhaled nebulized urban ambient coarse particles, or heated urban ambient coarse particles (in an attempt to denature any biological material present on/in the coarse particles) had evidence of inflammation of the respiratory tract (elevated polymorphonuclear cells, macrophage mRNA and tumor necrosis factor-alpha, eotaxin, immunologically-activated macrophages and phagocytic activity in sputum) at 2-3 hours post-exposure (Alexis et al., 2006). Heat denaturation of the biological components of the coarse particles did not affect sputum polymorphonuclear cell counts, but was associated with reduction of inflammatory markers (macrophage mRNA, eotaxin, phagocytosis and immunologically-activated macrophages). These results support the hypothesis that coarse particles exposure may "skew the airways toward an allergic phenotype by enhancing eotaxin levels that may enhance responses to allergens or bacteria in individuals with allergy" (Alexis et al., 2006). The findings of this study are that exposure to coarse particles might enhance the response of individuals with allergy to airborne bacteria.

Intra-tracheal instillation of urban coarse particles (1 and 10 mg/kg) into C57BL/6 mice in a dose response study was associated with evidence of increased levels of pro-inflammatory cytokines (IL-6, tumor necrosis factor alpha, keratinocyte-derived chemokine) in bronchoalveolar lavage fluid at 4 hours, but not 12 or 24 hours post exposure (Happo et al., 2007). By 24 hours post-exposure, animals exposed to coarse particles had increased cellularity, neutrophil counts, and total protein in the bronchoalveolar lavage fluid. The results of this study showed the urban coarse particles caused higher levels of pulmonary inflammation compared with $\text{PM}_{2.5}$ derived from the same urban areas (Happo et al., 2007). Urban coarse particles from different European locations used in this study had different inflammatory marker profiles and significantly different composition (Pennanen et al., 2007) suggesting that the composition and source of particles influences the type of inflammatory processes occurring. Using a repeat-dose sub-acute exposure system (3 exposures over a 6 day period), the same investigators replicated the finding that coarse particles from these European sources are more inflammogenic to the respiratory system than fine particles (Happo et al., 2010).

Oropharyngeal instillation of coarse particles into mice was associated with evidence of pulmonary inflammation was assessed by bronchoalveolar lavage (Tong et al., 2010); however coarse particle exposure was not associated with cardiac injury in this model.

Coarse particles from wildfire smoke is more toxic to lung macrophages on an equal dose (by mass) basis than coarse particles isolated from ambient air, as evidenced by decreased numbers of macrophages in lung lavage fluid 6 and 24 hours after coarse particle instillation into mouse lungs *in vivo* and by cytotoxicity to a macrophage cell line observed directly *in vitro* (William et al., 2013). Coarse particles from wildfire are cytotoxic to mouse bronchoalveolar lavage macrophages *in vivo* and are potent inducers of free isoprostanes (a marker of oxidative stress) in bronchoalveolar lavage fluid (Williams et al., 2013). Wildfire smoke coarse particles induce alveolar Clara cell responses associated with decreased levels of Clara cell secretory protein CCSP. These results appear to support the hypothesis that oxidative stress-mediated macrophage toxicity plays a key role in the initial response of the mouse lung to wildfire PM exposure (Williams et al., 2013).

In vitro studies of mouse macrophage responses to the same urban coarse particles used in Happo et al., 2007, demonstrated that the inflammatory and cytotoxic responses were mostly associated with the insoluble fraction of the particles; however both water and soluble and insoluble fractions triggered pro-inflammatory cytokine production, cytotoxicity and apoptosis (Jalava et al., 2008). The link between pulmonary inflammogenicity and the insoluble fraction of coarse particles has been noted by other investigators in the mouse intra-tracheal exposure bronchoalveolar lavage model (Wegesser et al., 2008). Sea salt and soluble soil components were positively correlated with the induced toxic responses (Jalavar et al., 2008).

Intra-tracheal instillation of industrial and urban coarse particles from rats was associated with increases in neutrophils and pro-inflammatory cytokines (tumor necrosis factor alpha) and glutathione depletion in bronchoalveolar lavage fluid at 18 hours post-exposure (Schins et al., 2004). There were differences in the responses between the industrial and rural coarse particles with greater induction of pro-inflammatory cytokines and glutathione depletion in the rural coarse particles (Schins et al., 2004). Notably, irrespective of the source, the level of pulmonary inflammation was consistently higher with coarse particle exposure than with fine particle exposure (Schins et al., 2007). The differences in the inflammatory responses of the rural versus industrial coarse particles was associated with differences in their endotoxin content (higher in the rural coarse particles) and their ability to trigger the release of pro-inflammatory cytokines (Schins et al., 2004). However, opposite results in a similar mouse model have been reported for urban versus rural coarse particles in the USA where urban coarse particles were more inflammogenic (Gilmour et al., 2004). Differences in the concentration of soluble metals (iron, copper, vanadium, nickel, chromium or aluminum) and redox potential were not consistent with the different capacity of rural versus industrial coarse particles to trigger pulmonary inflammation (Schins et al., 2004). Coarse particles, irrespective of their source, were more inflammogenic than fine particles, however coarse particles did not result in substantial pulmonary pathology in this study.

Intra-tracheal instillation of coarse particles (3 or 10 mg/kg) from different European locations with varying traffic densities into spontaneously hypertensive rats was also associated with evidence of pulmonary cytotoxicity and inflammation in dose-dependent manner at 24 hours post-exposure (Gerlofs-Nihland et al., 2007). Increases in blood viscosity were also noted following coarse particle exposure. Coarse particles derived from urban areas with high motor-vehicle traffic tended to be more toxic than those from urban areas with lower motor-vehicle traffic.

Studies of different urban coarse particles with differing levels of metals and polycyclic aromatic hydrocarbons (PAH) using the spontaneously hypertensive mouse intra-tracheal instillation bronchoalveolar lavage model demonstrated that overall coarse particles are more potent at producing pulmonary inflammatory responses than fine particles and that metal rich coarse particles are more inflammogenic than coarse particles with lower metal content (Gerlofs-Nihland et al., 2009). Furthermore, PAH-rich coarse particles are more inflammogenic than coarse particles with lower PAH content.

Evaluation of ambient particles from different European cities demonstrated relationships between particle composition and effects in various animal (markers of respiratory inflammation, adjuvant potency studies) and *in vitro* (cytokine release) allergy models (de Haar et al., 2006). Particles derived from areas with traffic, industrial combustion and/or incinerators, and combustion of black and brown coal/wood smoke were associated primarily with adjuvant activity for respiratory allergy, whereas particles of crustal material and sea spray are predominantly associated with measures for inflammation and acute toxicity (de Haar et al., 2006). Particles that were derived from secondary inorganic aerosol and long-range transport aerosol were exclusively associated with systemic allergy.

6.1.3.2.3 Effects Specific to Sea Salt

Particles produced by the evaporation of seawater and sea spray are a substantial contributor to the total mass of coarse particles in many geographic locations. However there is very little experimental

toxicology data generated on these particles. Sea salt from coarse particles is a stimulant of pro-inflammatory cytokine (tumour necrosis factor-alpha and IL-6) production and cytotoxicity in cultured mouse macrophage cells (Jalava et al., 2008). This is consistent with the epidemiological evidence on the respiratory inflammogenic properties of these particles.

6.1.3.2.4 Conclusions

There is limited evidence that inhalation of concentrated coarse particles can have mild effects on HR and HRV, vascular autonomic responses and systemic inflammation. However, there is reasonable and consistent evidence that entry of coarse particles into the lung results in the induction of a pro-thrombotic state in animal models. It should be noted that the experimental routes of exposure used to generate this data are non-physiological and the doses used to induce these effects are extremely high. It is difficult to extrapolate between these experimental exposure scenarios and real-world exposures. Furthermore, only acute (single exposure) single dose exposures have been studied. This makes it very difficult to define dose response curves and to derive adequate toxicological thresholds. While single acute high-dose exposure episodes may be suitable for examining the effects of periodic short-term increases in particulate air pollution (i.e. pollution events), there has been no examination of the effects of longer durations of exposure. Most of the data pertaining to the pro-thrombogenic effect of coarse particles has been derived from studies using urban particles. Detailed investigations into urban versus non-urban, and crustal versus non-crustal coarse particles is generally lacking.

While there is little evidence that single acute exposures to coarse particles significantly affect short-term pulmonary function (i.e. up to 24 hours post-exposure), there is substantial experimental evidence that acute coarse particle exposures induce inflammation of the respiratory tract. The results of toxicological studies indicate that irrespective of their origin/source, coarse particles are generally as, or more inflammogenic than fine and ultrafine particles. Particle composition appears to affect both the degree of inflammogenicity as well as the type of inflammatory processes occurring (i.e. the pro-inflammatory cytokine and cellular milieu). Metal, polycyclic aromatic hydrocarbon, sea salt, water-soluble and water insoluble fractions all appear to modulate the inflammatory processes.

While large and rapidly growing volume of research on fine and ultrafine particles is available, much less work has been performed on coarse particles. The limited experimental toxicology data set allows the following conclusions:

1. High acute doses of humans to coarse particles have minimal to small effects on heart rate and heart rate variability. The overall clinical importance of these findings is not clearly established.
2. Although the currently available results may be conflicting because of differences between animal models, there is a small amount of experimental evidence that acute exposures to coarse particles in rodents may have effects on vasomotor function. The overall clinical importance of these findings to humans is not clearly established.
3. There is limited experimental data suggesting that exposure to coarse particles results in systemic inflammatory reactions and possible endothelial injury.
4. There is a large body of experimental studies that provide support to the respiratory inflammogenic properties of coarse particles. While the source and composition of coarse particles affects the underlying mechanisms of inflammation it is not possible to make clear associations between specific particle sources and composition and specific effects.
5. Although only geographically limited samples have been studied, coarse particles derived from areas with traffic, industrial combustion and wood smoke are associated with immune adjuvant activity in various experimental models of respiratory allergy.
6. There is limited experimental evidence that acute high-level exposure to coarse particles induces a pro-thrombotic state in laboratory rodents. These effects may predispose humans to cardiac, cerebrovascular and other vasculo-occlusive diseases.

7. There is little toxicological experimental evidence regarding the toxicity of inhaled sea salt particles.

6.1.3.2.5 Crustal Particles

The relative toxicity of crustal particles compared to non-crustal particles has been an issue subject to substantial debate in recent years. The WHO REVIHAAP report (WHO, 2013) noted that desert dust episodes have been linked with cardiovascular hospital admissions and mortality in a number of epidemiological studies. Saharan dust is known to impact on Southern European cities and can lead to high levels of PM₁₀ during these events. Desert dust is also known to impact on Canadian and US cities and has been the subject of significant research.

Studies of Saharan dust events on mortality in Southern European cities have shown varied results. A study conducted in Madrid (Diaz et al., 2012) found that on days affected by Saharan desert dust the risk of cause-specific mortality per 10µg/m³ PM₁₀ was greater than on non-Saharan dust days. The greatest effects on Saharan dust days were seen for respiratory mortality during the cold season (3.34% compared with 2.87%) while for circulatory effects the effects were greater during the warm season (4.19% compared with 2.65%). Mean PM₁₀ levels on Saharan dust days (about 20% days) was 47.7µg/m³ compared with 31.4µg/m³ on non-Saharan dust days. The results of the study by Diaz et al., 2012 are consistent with a study conducted in Rome (Malone et al., 2011). A study conducted in the Emilia-Romana region of Italy concluded that Saharan dust days are an independent risk factor that increases the respiratory mortality (Sajani et al., 2011).

Perez et al., (2008) investigated the association between coarse particles on Saharan dust days and daily mortality compared with non-Saharan dust days. The study included 24,850 deaths. During Saharan dust days a daily increase of 10µg/m³ of PM_{2.5-10} increased daily mortality by 8.4% compared to 1.4% on non-Saharan dust days. By contrast there was no increase in risk of mortality for PM_{2.5} during the Saharan dust days. The difference in chemical composition did not explain the findings of the study (Perez et al., 2008).

A study conducted in Sydney examined the association between PM₁₀ levels and increases in emergency department attendances and hospital admissions during the September 2009 dust storm that impacted the city (Merrifield et al., 2013). PM₁₀ during that period was extremely high with daily average levels between 783 and 11,705 µg/m³ and mainly of crustal origin. Compared to non-dust event periods there was a 20% increase in respiratory emergency department attendances and 23% increase in asthma emergency department attendances. There was also a 10% increase in respiratory hospital admissions during the dust storm period. No associations were found for cardiovascular outcomes.

6.1.4 Summary

The review of the international literature in regard to particles shows that adverse health effects are observed with PM₁₀, PM_{2.5} and coarse particles, PM_{2.5-10}. These health effects include increases in mortality and morbidity outcomes including hospital admissions and emergency department attendances for respiratory and cardiovascular diseases. Studies investigating the effects of dust storms, including a study of the 2009 Sydney dust storm (Jalaludin et al., 2009), has shown that particles of crustal origin also lead to adverse health effects. The findings of epidemiological studies are supported by the results of toxicological studies.

6.1.5 Health Endpoints to be Considered

The results of epidemiological studies have shown that a wide range of health effects are associated with exposure to PM₁₀. Australian studies (NEPC, 2012; EPHC 2006) have found associations between PM₁₀ levels currently experienced in Australian cities and the following health outcomes:

- Increases in daily mortality
- Hospital Admissions
 - Respiratory disease
 - Cardiovascular disease
 - Cardiac disease
 - Pneumonia and bronchitis
- Emergency room attendances asthma

These health outcomes have been assessed in this health risk assessment for the relevant age groups.

Although no studies investigating the long term effects of exposure to PM₁₀ on health have been conducted in Australia, there have been several international studies that have shown strong associations between long-term exposure to PM₁₀ and increases in mortality. On the basis of the findings of these studies long-term mortality has also been assessed.

6.1.6 Sensitive Populations to be included in HRA

There are several groups within the general population that have been identified as being more vulnerable to the effects of air pollution. These include:

- Elderly
- People with existing cardiovascular and respiratory disease
- People with asthma
- Low socioeconomic groups
- Children

Compared to healthy adults, children are generally more sensitive to air pollutants as their exposure is generally higher. The reasons for this are that children inhale more air per minute and have a larger contact lung surface area relative to their size compared to adults. Other factors that increase the potential for exposure in children are that children generally spend more time outdoors and more time exercising.

Epidemiological studies (USEPA, 2012; 2009) have shown that people who have a low socioeconomic status (SES) also form a group within the population that is particularly vulnerable to the effects of air pollution. This is largely due to the fact that people within these groups usually have poorer health status than people within higher SES groups. They may also have poorer access to medical care. In addition, they usually live in areas that are more polluted (e.g., near major roads or near industry) as property is generally cheaper in these areas.

6.1.7 Exposure-response functions

To calculate the number of people that might be impacted by air pollution exposure-response functions for each outcome being assessed are required. These functions are a measure of the change in the health outcome within the population for a given change in PM₁₀ or PM_{2.5} concentration.

The exposure-response functions in **Table 4** and **Table 5** have been taken from Australian studies and in particular two multicity meta-analyses (Simpson et al., 2005 a and b; EPHC, 2011). The use of Australian meta-analyses is consistent with the NHMRC (2006) and NEPC (2011) recommendations for selecting exposure response functions for risk assessments for air pollution.

The exposure-response functions for long-term exposure to PM₁₀ and PM_{2.5} have been taken from the American Cancer Society study (HEI, 2009). This study is considered by the WHO as the most reliable study to use to assess long-term effects of air pollution. The use of this value is also consistent with the recommendations made by NHMRC (2006) and NEPC (2011).

Table 4: Exposure Response Functions for PM₁₀ Selected Health Outcomes (Taken from EPHC, 2011; HEI, 2009)

Outcome	Averaging Period	Exposure Response Function per 1 µg/m³ increase in PM₁₀
Annual all-cause mortality (non-accidental) 30+ years	Annual Average	0.004
Daily all-cause mortality(non-accidental) all ages	24 hours	0.002
Daily mortality cardiovascular disease - all ages	24 hours	0.002
Hospital Admissions respiratory disease 65+ years	24 hours	0.003
Hospital Admissions cardiac disease 65+ years	24 hours	0.002
Hospital Admissions pneumonia and bronchitis 65+ years	24 hours	0.0013
Hospital Admissions respiratory disease 15-64 years	24 hours	0.003
ED Visits Asthma 1-14 years	24 hours	0.015

Table 5 shows the exposure response functions used for PM_{2.5}.

Table 5: Exposure Response Functions for PM_{2.5} Selected Health Outcomes Taken from EPHC, 2011; HEI, 2009)

Outcome	Averaging Period	Exposure Response Function per 1 µg/m ³ increase in PM _{2.5}
Annual all-cause mortality (non-accidental) 30+ years	Annual Average	0.006
Annual cardiopulmonary mortality 30+	Annual average	0.014
Annual mortality ischemic heart disease 30+ years	Annual average	0.024
Annual mortality lung cancer 30+ years	Annual average	0.014
Daily all-cause mortality(non-accidental) all ages	24 hours	0.0023
Daily mortality cardiovascular disease - all ages	24 hours	0.0013
Hospital Admissions respiratory disease 65+ years	24 hours	0.004
Hospital Admissions cardiac disease 65+ years	24 hours	0.005
Hospital Admissions cardiovascular disease 65+ years	24 hours	0.003
Hospital Admissions ischemic heart disease 65+ years	24 hours	0.004
Hospital Admissions COPD 65+ years	24 hours	0.004
Hospital Admissions pneumonia and bronchitis 65+ years	24 hours	0.005
Hospital Admissions respiratory disease 15-64 years	24 hours	0.003
ED Visits Asthma 1-14 years	24 hours	0.0015

6.2 Exposure Assessment

The Port Hedland Industries Council (PHIC) was established by industry in 2009 to develop an integrated approach to air quality (and noise) monitoring in Port Hedland, Western Australia. This has included the establishment of a network of 8 ambient air quality monitoring stations across the area.

PHIC has established an ambient monitoring network in place to ensure that dust generated by port and industry operations does not adversely impact the Port Hedland community. This includes both ambient air monitoring located within residential areas that was established for to provide data for the HRA (DoH, personal communication), Additional site boundary monitoring captures data that is used to ensure compliance with regulatory commitments and requirements for dust mitigation and management. The real-time data is also made accessible to the community via a monitoring website.

Not all air monitoring stations have been established to enable assessment of population exposure and therefore not all data collected by the network is relevant for the HRA.

The eight monitoring site locations are shown in Figure 4. The Richardson Street, Kingsmill Street, Taplin Street, and Neptune Place monitoring locations are within urban or residential land use areas of Port Hedland. The Richardson St site is at the interface of the commercial and residential areas and can be considered as the site most affected by activities at the Port.

The Wedgefield monitoring location is within a light industrial area that also provides site-worker accommodation. The population of Wedgefield is unknown and no data was available for use in this HRA. Given the industrial nature of Wedgefield as opposed to urban or residential use, the data generated at this site is not appropriate for use in the HRA.

The Acacia Way monitoring station is positioned within South Hedland in an urban residential location and serves as a representative site for the population based in South Hedland. The data from this site has been used to assess the risk to the population of South Hedland in the HRA. The data from Acacia Way was used as the background urban site for the HRA as it is not influenced by the Port activities.

The Bureau of Meteorology (BoM) and Yule River locations are relatively distant to industrial and related activities, and populations, and both serve as regional background monitoring locations. As no populations live in proximity to these sites the data has not been used in the HRA to calculate population risks. The data from the Yule River site has been used for non-urban regional background comparison purposes for this study as works at the Port Hedland Airport during 2013 impacted on the data collected at the BoM site.

The Taplin Street monitoring station is located in a position that is likely to be impacted by emissions from various industry operations in the Port Hedland area. This monitoring station is also positioned with adequate line of site to nearby industry operations. It is this location at which the Taskforce has set the Port Hedland Air Management Assessment Criteria (DSD, 2010). The Port Hedland Dust Management Taskforce (PHDMT) has specified an interim guideline of $70 \mu\text{g}/\text{m}^3$ for PM_{10} (24 hour average) with 10 exceedences per year, as determined at the Taplin Street monitoring station. This interim guideline has been specified in order to maintain the co-existence of industry and community as well as to manage potential risk to human health. This criterion is part of a continuous improvement framework within which industries in Port Hedland can work to reduce emissions over time (DSD, 2010).

The Taplin St site is at the East –West interface in Port Hedland. The west end of Port Hedland is represented by the monitoring stations to the west of Taplin St – Richardson and Kingsmill St. Neptune Place is located to represent the population in the east end of Port Hedland and is to the east of Taplin St.

The parameters monitored at each of PHIC monitoring sites are listed in Table 6.

Table 6: Sites and Parameters Monitored

Monitoring Station	Parameter
Taplin Street	PM ₁₀ (including metals), NO _x , SO ₂
Richardson Street	PM ₁₀ (including metals)
Neptune Place	PM ₁₀
Kingsmill Street	PM ₁₀
Acacia Way	PM ₁₀ (including metals), NO _x , SO ₂
Wedgefield	PM ₁₀ (including metals),
Yule River	PM ₁₀ (including metals)
Bureau of Meteorology (BoM)	PM ₁₀ (including metals), NO _x , SO ₂

The monitoring methods for each parameter in the PHIC monitoring network are listed in Table 7. This includes the type of equipment in use at each site, as well as the measurement standard or method applicable to the monitoring equipment in use.

Table 7: Sites and Monitoring Methods

Parameter	Equipment	Measurement Standard	Site
PM ₁₀	ThermoBAM	AS/NZS 3580.9.11:2008 & AS/NZS 3580.9.3:2003 – BAM 1020/THERMO/HVAS	BoM
PM ₁₀	High Volume Air Sampler 3000	AS/NZS 3580.9.11:2008 & AS/NZS 3580.9.3:2003 – BAM 1020/THERMO/HVAS	BoM, Acacia Way, Richardson Street, Wedgefield, Yule River
PM ₁₀	BAM	AS/NZS 3580.9.11:2008 & AS/NZS 3580.9.3:2003 – BAM 1020/THERMO/HVAS	Acacia Way, Kingsmill Street, Neptune Place, Richardson Street, Taplin Street, Wedgefield, Yule River
NO _x	Ecotech ML9841	AS/NZS 3580.4.1:2008 & AS/NZS 3580.5.1:2011 – NO _x & SO ₂	BoM, Taplin Street, Acacia Way
SO ₂	Ecotech EC9850	AS/NZS 3580.4.1:2008 & AS/NZS 3580.5.1:2011 – NO _x & SO ₂	BoM, Taplin Street, Acacia Way
PM ₁₀ (including metals)	Hi-Vol	AS/NZS 3580.9.11:2008 & AS/NZS 3580.9.3:2003 – BAM 1020/THERMO/HVAS	BoM, Acacia Way Richardson Street, Taplin Street, Wedgefield, Yule River
Respirable Crystalline Silica (RCS)	Hi-Vol	Airborne samples analysed according to AS 2985 for Respirable Dust or AS 3640 for Inhalable Dust. Quartz analysed in accordance with NIOSH 7603	Richardson St, Taplin St, Neptune Place
Asbestiform Fibres	Hi-Vol	The initial analysis was a fibre count as per <i>NOHSC:3003 (2005) Guidance Note On The Membrane Filter Method For Estimating Airborne Asbestos Fibres</i> and was conducted at MPL Laboratory. Any samples returning fibre counts of 2 or more fibres were sent for scanning electron microscopy, SEM, analysis at MicroAnalysis laboratory.	Richardson St, Taplin St, Neptune Place

The location of the PHIC monitoring stations is shown in Figure 4:

Figure 4: Location of PHIC Monitoring Sites in Port Hedland



Figure 5 shows the PM₁₀ data collected at all sites between January 2012 and December 2013. It is clear from the data shown in **Figure 5** that there are numerous exceedances of the NEPM PM₁₀ standard of 50 µg/m³ (between 9 and 74 days in 2013) and the interim standard adopted by the Port Hedland Taskforce of 70 µg/m³ (between 2 and 16 days in 2013). Table 8 shows the summary statistics for the PM₁₀ data including the number of exceedances at each monitoring location. The highest levels of PM₁₀ are observed at the regional background site at Yule River. On the days when the highest levels were observed at Yule River, there does not appear to be an influence of regional dust on the

monitoring sites in Port Hedland. In 2013 there was only one day, 14 December, when dust levels were elevated at all sites including Yule River. There were four other days when the interim standard was exceeded at Yule River. On those days no exceedances were observed at the Hedland sites. Conversely when elevated levels of PM₁₀ were observed for the Port Hedland sites, the levels at Yule River are not substantially elevated. This suggests that over the monitoring period PM₁₀ levels in Port Hedland when elevated levels of PM₁₀ were observed they were not significantly impacted by regional dust. If regional dust from the Pilbara was impacting on Port Hedland elevated levels would be expected across all monitoring locations. There was only one day in 2013 when this occurred.

Figure 5: PM₁₀ Data from PHIC Monitoring Sites

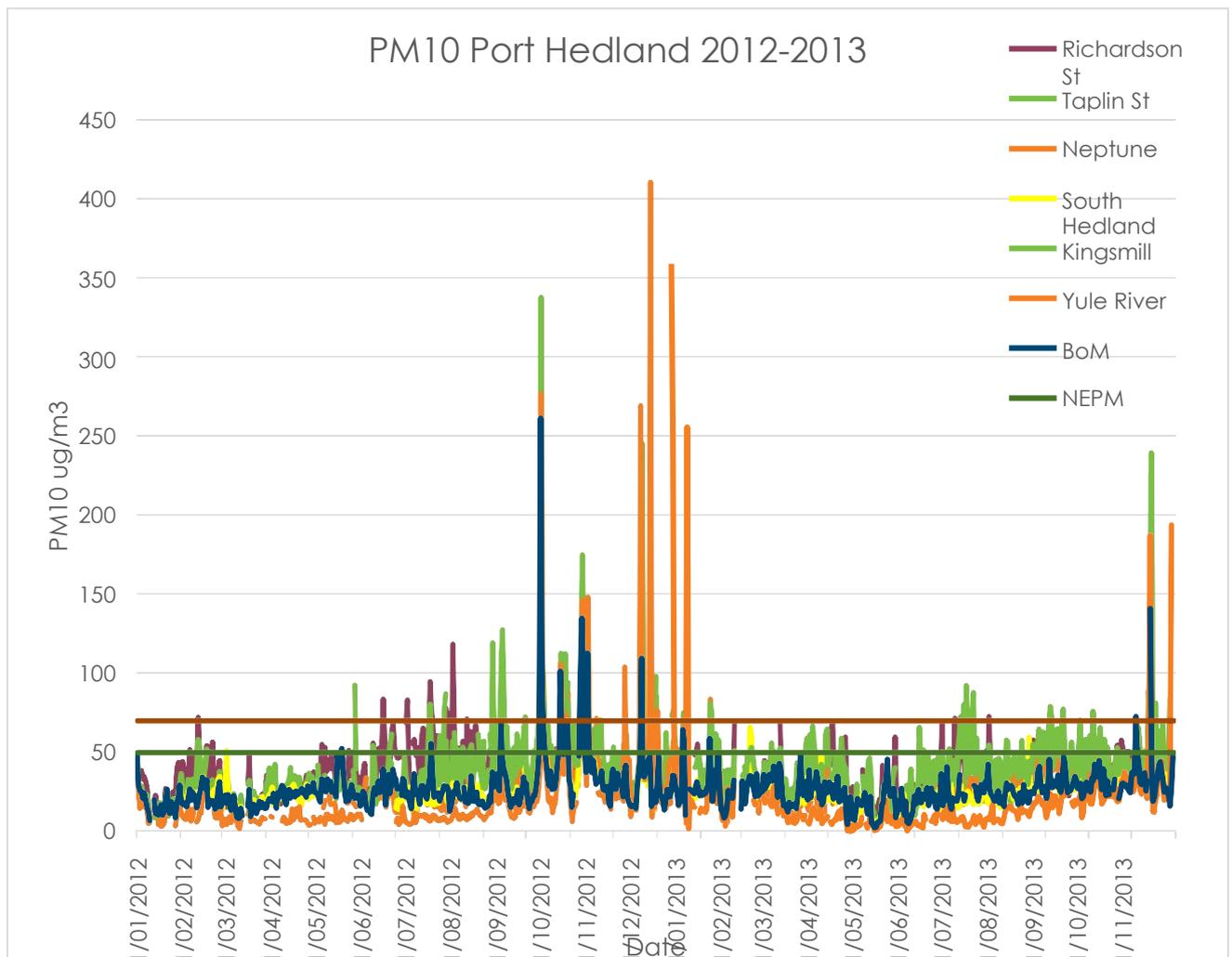
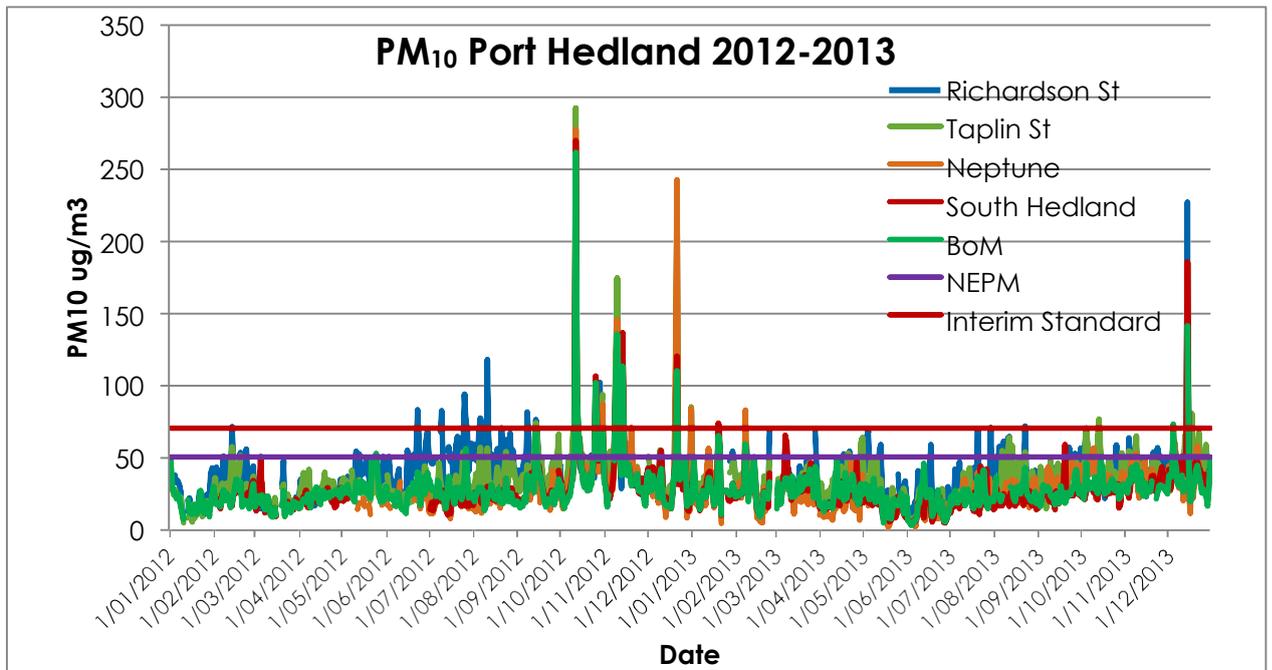


Figure 6 shows the PM₁₀ data for the Port Hedland sites only (minus Yule River). Examination of the PM₁₀ data for the Port Hedland sites only (minus Yule River) shows that there are numerous exceedances of both the NEPM and interim taskforce guideline at all sites during the monitoring period (see Table 8 for summary statistics for each monitoring location). Examination of the data shows that when exceedances occur they do not occur at all sites. Wind direction data was obtained from the PHIC online database via DoH. The predominant wind direction on most days when there were exceedances of the guidelines at Richardson St and Taplin St indicates that they are from the S-SE (south-southeast). On 16 of these days PM₁₀ levels in South Hedland were not elevated. This can be

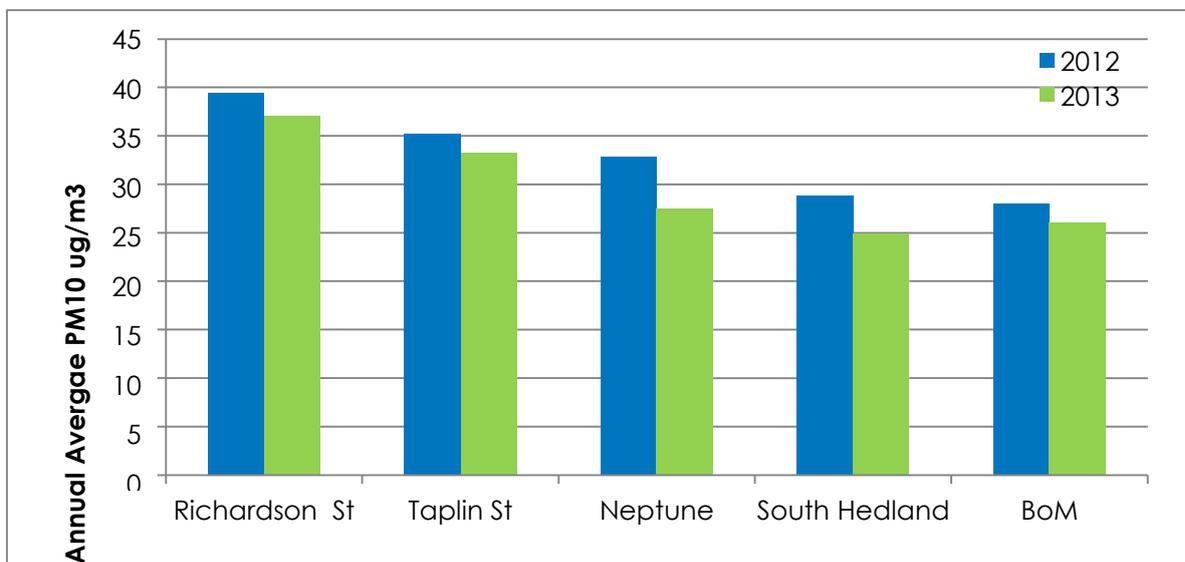
seen in **Figure 6**. What this indicates is that there is a source between South Hedland and the Richardson and Taplin St sites that is being transported by winds from the S-SE. If this was regional dust it would be seen across all sites.

Figure 6: PM₁₀ data PHIC monitoring sites minus Yule River



The trend in the annual average PM₁₀ levels for 2012-13 is shown in **Figure 7**. This data shows that there appears to be an influence of industry / Port activities on the PM₁₀ levels in Port Hedland with the highest levels observed for Richardson St with decreasing levels observed with increasing distance from the Port operations. The trend in the peak values is more variable.

Figure 7: Annual Average PM₁₀ Levels from PHIC Monitoring Sites



The summary statistics for PM₁₀ data collected at the PHIC monitoring sites are shown in Table 8.

Table 8: Summary PM₁₀ Statistics

		Richardson St	Taplin St	Neptune	South Hedland	Kingsmill	Yule River	BoM
2012	Max	240.34	292.25	276.98	269.22	337.15	410.08	261.00
	Average	39.60	35.20	33.30	28.99	53.98	18.33	28.09
	99 th percentile	133.7	153.4	248	124.7	274	268	110.9
	95 th percentile	77	66.8	81.5	60.5	109	52	51.7
	90 th percentile	64.9	52.7	55.6	44.6	86	34	41
	Number of days exceeding 70 µg/m ³	22	14	9	6	25	8	8
	Number of days exceeding 50 µg/m ³	73	43	20	19	74	14	19
2013	Max	226.63	80.27	184.38	185.08	238.70	358.96	140.71
	Average	36.94	33.31	27.35	24.96	41.82	19.18	26.08
	99 th percentile	70.4	71.5	63	66.5	88	237.2	61.9
	95 th percentile	59.2	58.5	48.5	45	70.9	44.3	43
	90 th percentile	54	50	43	38.7	61	32	37
	Number of days exceeding 70 µg/m ³	4	4	2	2	16	7	2
	Number of days exceeding 50 µg/m ³	47	33	12	9	74	13	5

Further PM₁₀ data collected at Spoilbank was collected during 2014 for LandCorp (EcoTech 2014). This data was only available in late December 2014 and does not form part of the PHIC network. Data was collected at two locations as shown in

Figure 8. This area has been identified for development of a new marina and residential development. Presently there is no residential population on Spoilbank and the data is not reflective of exposure of the population. This data has not been used in the HRA.

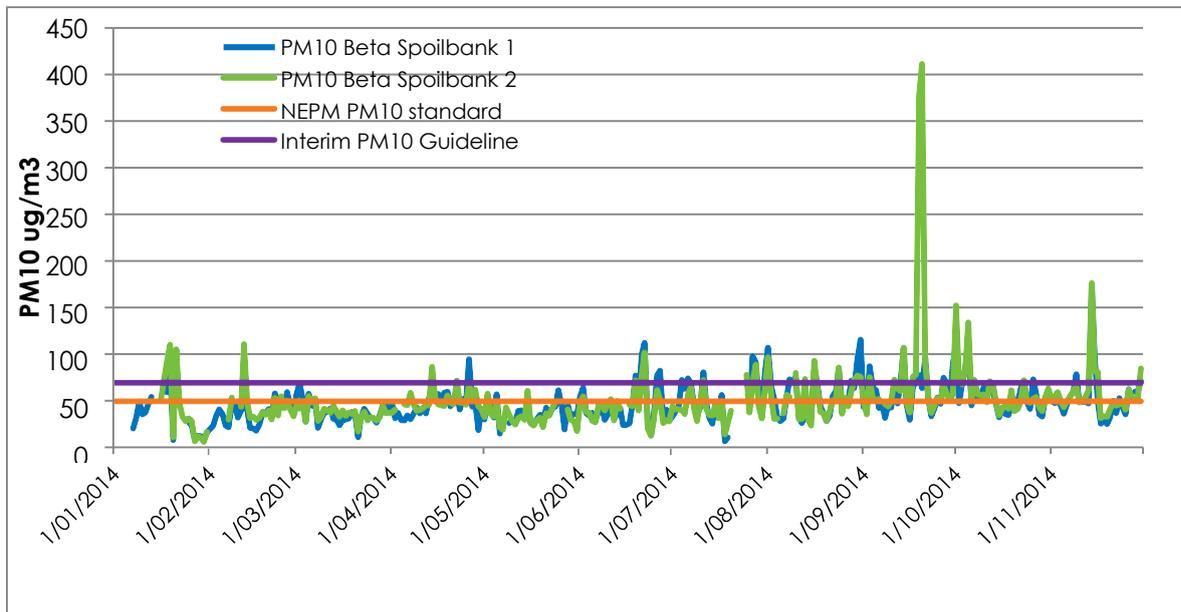
Figure 8: Location of Landcorp PM₁₀ Spoilbank monitoring locations.



The data collected at these locations is shown in

Figure 9.

Figure 9: PM₁₀ data from Landcorp Spoilbank Monitoring locations



As can be seen from

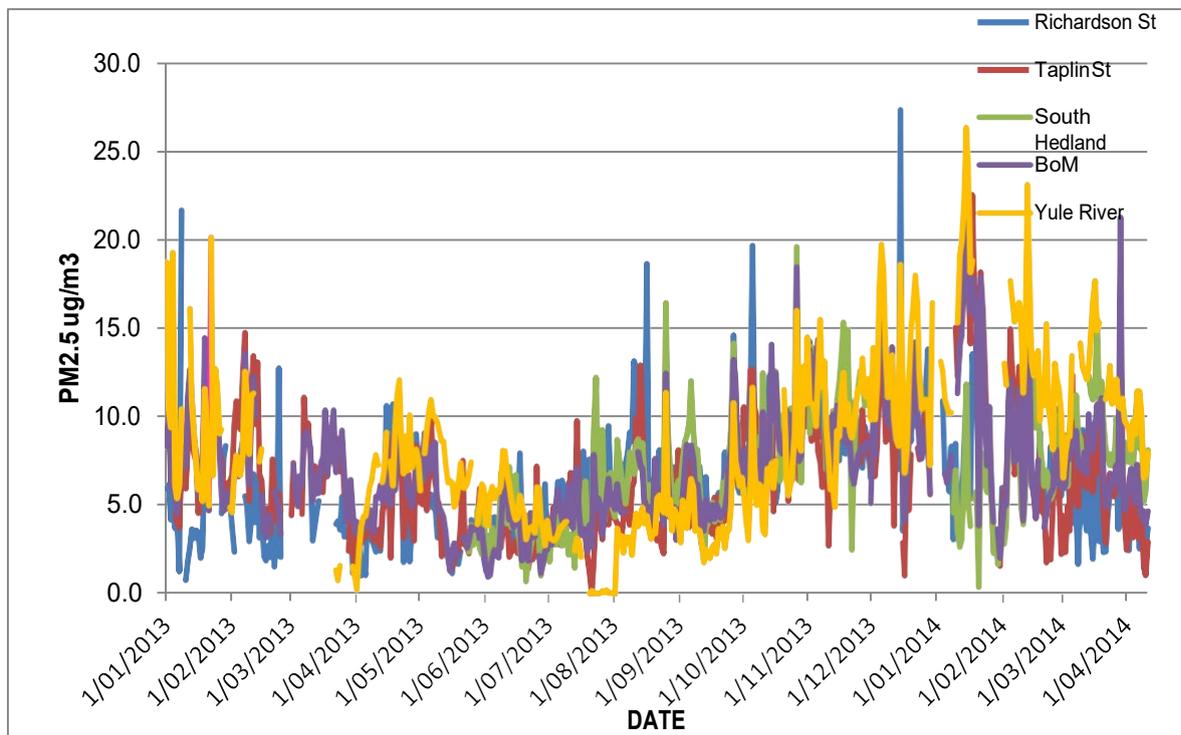
Figure 9 there are numerous exceedances (118 days during the monitoring period) of both the current NEPM standard of $50 \mu\text{g}/\text{m}^3$ and the interim guideline value of $70 \mu\text{g}/\text{m}^3$. In general the data at the two sites are similar although the data from Spoilbank 2 site is higher than that observed at the Spoilbank 1 site in many instances. The high peaks observed on September 19 and 20, 2014 at Spoilbank 2 were not seen at the Spoilbank 1 site. No explanation was provided in the Ecotech report (December 2014) as to the cause of these high levels. The mean and maximum values for the Spoilbank sites were higher than those observed at the other monitoring locations. The mean values were $47 \mu\text{g}/\text{m}^3$ and $51 \mu\text{g}/\text{m}^3$ at Spoilbank 1 and 2 respectively with corresponding maximum values of $175 \mu\text{g}/\text{m}^3$ and $411 \mu\text{g}/\text{m}^3$.

$\text{PM}_{2.5}$ data was collected at 5 sites, Richardson St, Taplin St, South Hedland, BoM and Yule River, during 2012 to April 2014. The data in

Figure 10 shows the PM_{2.5} data collected for all sites. As can be seen from

Figure 10 only 2 exceedances of the NEPM advisory reporting standard of 25 $\mu\text{g}/\text{m}^3$ were recorded during that period – one at Richardson St and one at Yule River. The annual average at all sites during 2013 did not exceed the annual $\text{PM}_{2.5}$ NEPM standard.

Figure 10: PM_{2.5} Data From PHIC Monitoring Sites 2012-2014.



Summary statistics for PM_{2.5} are shown in Table 9:

Table 9: Summary Statistics for PM_{2.5} (µg/m³) 2013-14

	Richardson St	Taplin St	South Hedland
Maximum	27.3	22.6	19.6
Mean	6.4	6.8	6.2
99th percentile	18.5	20.6	16
95th percentile	13.1	14.3	12.6
90th percentile	11.5	12.3	11.5

6.2.1 Adjustment of PM₁₀ data for Scenario modelling

As part of the risk characterisation the health risks associated with reductions in PM₁₀ levels such that the peak levels would be the alternative standards/guidelines considered in the HRA was required. The standards/guidelines to be assessed include the Taskforce guideline of 70 µg/m³ and the NEPM standard of 50 µg/m³. At the time of the preparation of the HRA, NEPC had proposed that the PM₁₀ standard in the NEPM be revised to 40 µg/m³ as a 24-hour average. An assessment against this proposed standard has been included in the HRA.

To derive a PM₁₀ dataset that is representative of each of these scenarios the percentage reduction required to reduce the peak levels below the specific standard/guideline has been determined for each monitoring location. The adjustment factors applied are summarised in Table 10:

Table 10: Adjustment Factors Applied to Derive PM₁₀ datasets for each Scenario assessed (% reduction) 2013 data

	Richardson St (% reduction)	Kingsmill St (% reduction)	Taplin St (% reduction)	Neptune Place (% reduction)	Acacia Way (% reduction)
Scenario 1: meeting 70 µg/m³	69	71	13	62	62
Scenario 2: meeting 50 µg/m³	78	79	38	73	73
Scenario 3: meeting 40 µg/m³	82	83	50	78	78

These adjustment factors were applied to all available data across the whole distribution to derive a daily PM₁₀ dataset. This approach was taken to account for all sources of dust in Port Hedland and assumes that the PM₁₀ levels follow a normal distribution. It also assumes that any actions taken to reduce PM₁₀ levels will be applied for a range of sources and not just apply to one source.

Although sea salt contributes to PM₁₀ levels in Port Hedland the extent to which it influences total PM₁₀ will vary day-to-day based on wind speed and direction. When winds are coming from inland the contribution to total PM₁₀ will be less than on days when the wind is coming from the ocean. Until further information is available on the contribution of sea salt to total PM₁₀ under different meteorological conditions the contribution from sea salt cannot be more accurately accounted for.

An alternative approach to adjust PM₁₀ levels would be to truncate the peak levels at the relevant guideline/standard. This approach assumes that there is one dominant source of PM₁₀ impacting on total PM₁₀ levels and that actions are targeted at exceedance days. This approach ignores broader air quality management actions that should be applied to address all sources of PM₁₀. This approach is likely to lead to an overestimate of the risks posed by PM₁₀ in each of the scenarios and has not been applied in the HRA.

6.3 Risk Characterisation

The results of epidemiological studies have shown that a wide range of health effects are associated with exposure to PM₁₀. Australian studies (NEPC, 2012; EPHC 2006) have found associations between PM₁₀ levels currently experienced in Australian cities and the following health outcomes:

- Increases in daily mortality
- Hospital Admissions
 - Respiratory disease
 - Cardiovascular disease
 - Cardiac disease
 - Pneumonia and bronchitis
- Emergency room attendances for asthma

These health outcomes have been assessed in this health risk assessment for the relevant age groups.

Although no studies investigating the long term effects of exposure to PM₁₀ on health have been conducted in Australia, there have been several international studies that have shown strong associations between long-term exposure to PM₁₀ and increases in mortality. On the basis of the findings of these studies long-term mortality has also been assessed.

Baseline health statistics were obtained from the DoH epidemiology unit. Given the small population in Port Hedland, data was not available for all age groups for all outcomes. No statistics were available for the indigenous communities therefore they were unable to be considered as a separate vulnerable group. Studies on the health effects of smoke in Darwin have shown that indigenous populations appear to be more vulnerable to the effects of PM₁₀ than non-indigenous communities.

For most outcomes health data was only available for Port Hedland town as a whole and South Hedland which does not allow specific analysis of the effects in the west compared to the east end or South Hedland. To account for this the risk to the whole population of Port Hedland using both Richardson St and Taplin St data has been calculated and compared to the risk to the population of South Hedland. For all-cause mortality data was available for the west end and east end as well as South Hedland so this analysis has been undertaken to compare the risk in the separate areas.

The health effects for which data was available and for which risks have been calculated are:

- All-cause mortality all ages – long-term and daily
- Daily cardiovascular mortality all ages
- Hospital admissions for respiratory disease – 15-64 years, 65+ years
- Hospital admissions pneumonia and bronchitis 65+ years

The baseline health statistics for each area have been combined with the relevant health statistics, concentration response functions per µg/m³ of PM₁₀ and the air pollution data for each area as monitored at the PHIC monitoring sites.

The increase in risk per 100,000 population due to PM₁₀ has been calculated using the following equation:

$$\text{Increase in risk for each health outcome} = \frac{\text{exposure response function}/\mu\text{g}/\text{m}^3 \text{ PM} \times \text{PM concentration} \times \text{baseline incidence rate}}{100,000 \text{ population}}$$

To calculate the number of attributable cases the risk per 100,000 was multiplied by the actual population as a fraction of 100,000. The number of cases for each outcome was calculated for the population represented by each monitoring location. For short-term effects associated with daily

changes in PM₁₀ the number of cases for each day of the year were calculated and then summed to give the annual total. For the assessment of long-term mortality, the annual average concentrations were used in the calculations. Sample calculations are shown in Appendix C and baseline health data shown in Appendix B.

The increase in risk for each health outcome has been calculated using the 2013 monitoring data collected at the Richardson St, Taplin St and South Hedland (Acacia Way) monitoring sites. The 2013 data was used as it was the most complete dataset available for inclusion in the HRA. For the short-term effects the data for every day of the year (where available) was used. It should be noted that there were days where data was not available due to issues with the data collection. For long-term effects the annual average concentration was calculated from the available daily data was calculated. In all cases the data collection for the year was greater for 75% enabling an annual average concentration to be calculated. The risks have been expressed as the potential increase in health outcome per 100,000 of population to account for the differences in population in each location and allow meaningful comparisons across all areas. The actual number of people affected will be lower as the population of Port Hedland is less than 100,000. Risks have been calculated for current monitored PM₁₀ levels, PM₁₀ meeting 70 µg/m³ and PM₁₀ meeting 50 µg/m³. In adjusting the PM₁₀ data to estimate risk from the latter two scenarios, the whole distribution of the monitored PM₁₀ was adjusted to meet either the 70 µg/m³ guideline or 50 µg/m³ standard. The results for PM₁₀ are shown in **Table 11**.

Table 11: Increase in health outcome attributable to PM₁₀ per 100,000 population

Outcome*	Port Hedland using Richardson St data current	Port Hedland using Richardson St data meeting 70 µg/m ³	Port Hedland using Richardson St data meeting 50 µg/m ³	Port Hedland using Taplin St data current	Port Hedland using Taplin St data meeting 70 µg/m ³	Port Hedland using Taplin St data meeting 50 µg/m ³	South Hedland current	South Hedland meeting 70 µg/m ³	South Hedland meeting 50 µg/m ³
Long-term all-cause mortality	29	12	6	26	23	16	15	5	3
Daily all-cause mortality	13	4	3	12	11	8	5	2	1.6
Daily Mortality cardiovascular causes	6	2	1	5	5	3	2	0.5	0.4
Hospital Admissions Respiratory Disease 65+ years	1682	521	370	1581	1375	980	1435	390	278
Hospital Admissions Pneumonia and Bronchitis 65 + years	729	223	160	685	596	425	185	50	36
Hospital Admissions Respiratory Disease 15-64 years	80	25	18	76	66	47	122	33	24

The results in **Table 11** show that the resultant increase in health outcomes/100,000 are higher for Port Hedland using either the Richardson St or Taplin St data compared with the risks in South Hedland. There is a substantial reduction in risk if either the 70 $\mu\text{g}/\text{m}^3$ or 50 $\mu\text{g}/\text{m}^3$ guidelines could be met. The benefit of reducing PM_{10} levels in the Taplin St data is not as great as the reduction in risk associated with decreases in PM_{10} at both Richardson St and South Hedland. This is probably due to the variability in the data not being as great at Taplin St compared to the other locations. This would suggest that there is a source impacting on Taplin St that has a more constant rather than intermittent (large spikes in the data) impact.

To enable an assessment of what the risk means for actual cases per year, the risk calculation have been converted to the actual number of outcomes for the current population of Port Hedland and a predicted future population. According to the Town of Port Hedland publication *Pilbara Port City Growth Plan 2012* the predicted population for both Port Hedland and South Hedland combined is 50,000 people with one third of this total in Port Hedland and two thirds in South Hedland. This translates to a predicted population for Port Hedland of approximately 17,000 people. This figure has been used to calculate the potential number of adverse health outcomes for each of the scenarios assessed. Included in these calculations is an assessment against the proposed changes to the PM_{10} NEPM standard to 40 $\mu\text{g}/\text{m}^3$ as a 24-hour average. It should be noted that no formal decision has been made by NEPC at this time to adopt this standard however it has been included in this assessment for completeness. The results of these calculations using the Richardson St data are shown in

Table 12.

Table 12: Annual number of Health Outcomes Attributable to PM₁₀

Outcome	Port Hedland Richardson St data current	Port Hedland Richardson St current 17,000 population	Port Hedland Richardson St data meeting 70 µg/m ³	Port Hedland Richardson St data meeting 70 µg/m ³ 17,000 population	Port Hedland Richardson St data meeting 50 µg/m ³	Port Hedland Richardson St data meeting 50 µg/m ³ 17,000 population	Port Hedland Richardson St data meeting 40 µg/m ³	Port Hedland Richardson St data meeting 40 µg/m ³ 17,000 population
Long-term all-cause mortality	1.3	5	0.4	1.5	0.3	1	0.2	0.8
Daily all-cause mortality	0.6	2	0.2	0.7	0.1	0.5	0.1	0.4
Daily Mortality Cardiovascular Disease	0.3	1	0.08	0.3	0.06	0.2	0.05	0.2
Hospital Admissions Respiratory Disease 65+ years	2	7	0.6	2.2	0.4	1.6	0.4	1.3
Hospital Admissions Pneumonia and Bronchitis 65 + years	0.9	3	0.3	1	0.2	0.7	0.2	0.6
Hospital Admissions Respiratory Disease 15-64 years	2.7	10	0.9	3	0.6	2.2	0.5	1.8

Table 13 shows that in terms of actual numbers there would be 1 additional death per year due to long-term exposure to PM₁₀ in Port Hedland at the current PM₁₀ levels. If the population increased to 17,000 as planned, this would increase to 5 additional deaths in the community. If the 70µg/m³ interim guideline could be met then the number of additional deaths due to PM₁₀ would be 4 in 10 years for the current population. Increasing the population to 17,000 increases that number to 1.5 per year (15 in 10 years). For hospital admissions a similar picture is found. For admissions for respiratory disease in people over 65 years of age current PM₁₀ levels would lead to 2 additional admissions per year which would increase to 7 per year if the population is increased to 17,000 people. Reducing PM₁₀ levels such that the interim guideline of 70µg/m³ would lead to a significant public health benefit with reductions in both deaths and hospitalisations predicted. These benefits are also observed for reductions in PM₁₀ to meet the 50µg/m³ or proposed 40µg/m³ NEPM standards. **Table 13** shows the number of adverse health outcomes that could be avoided if these standards were achieved.

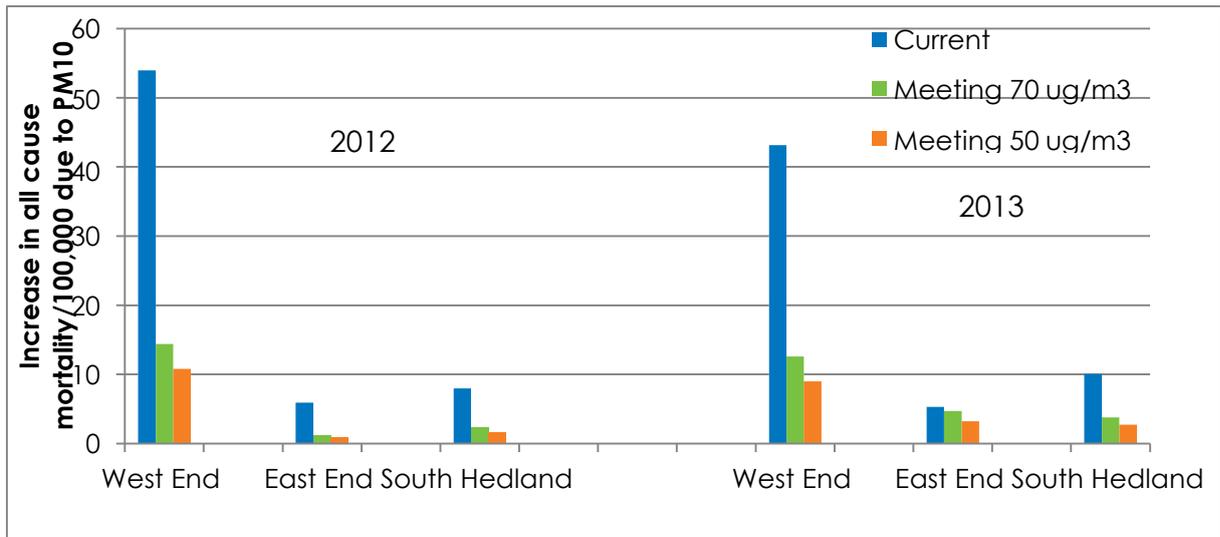
Table 13: Annual Health Outcomes Avoided on Meeting Alternative Standards

Outcome	Port Hedland Richardson St data meeting 70 µg/m ³	Port Hedland Richardson St data meeting 50 µg/m ³	Port Hedland Richardson St data meeting 40 µg/m ³	Port Hedland Richardson St data meeting 70 µg/m ³	Port Hedland Richardson St data meeting 50 µg/m ³	Port Hedland Richardson St data meeting 40 µg/m ³

				17,000 population	17,000 population	17,000 population
Long-term all-cause mortality	0.9	1	1.1	3.5	4	4.2
Daily all-cause mortality	0.4	0.5	0.5	1.3	1.5	1.6
Daily Mortality Cardiovascular Disease	0.22	0.24	0.25	0.7	0.8	0.8
Hospital Admissions Respiratory Disease 65+ years	1.4	1.6	1.6	4.8	5.4	5.7
Hospital Admissions Pneumonia and Bronchitis 65 + years	0.6	0.7	0.7	2	2.3	2.4
Hospital Admissions Respiratory Disease 15-64 years	1.6	2.1	2.5	7	7.8	8.2

Figure 11 shows the increased risk in all-cause mortality/100,000 population due to PM₁₀ for the west end, east end and South Hedland. The data shows that the risk/100,000 population is greater for the west end using Richardson St data compared with the east end (using Taplin St data) and South Hedland. There is a clear reduction in risk if the 70 µg/m³ or 50 µg/m³ could be met at all locations while reducing the concentration from 70 µg/m³ to 50 µg/m³ results in a comparatively smaller risk reduction.

Figure 11: Increase in health outcome attributable to PM₁₀ per 100,000 population



The health effects attributable to PM_{2.5} are shown in

Table 14. As can be seen from

Table 14 the risk from exposure to PM_{2.5} is similar using both Richardson St and Taplin St. The risks are much lower at South Hedland than in Port Hedland. The risks due to exposure to PM_{2.5} are much lower than those attributed to PM₁₀.

Table 14: Increase in health outcome attributable to PM_{2.5} per 100,000 population

Outcome	Port Hedland using Richardson St data current	Port Hedland using Taplin St data current	South Hedland current
Long-term all-cause mortality	8	8	2
Long term Lung cancer mortality	4	4	1
Long term Cardiovascular mortality	9	8	1
Daily all-cause mortality	3	3	2
Daily Mortality cardiovascular causes	1	1	0.2
Hospital admissions Respiratory Disease 65+ years	401	366	297
Hospital admissions pneumonia and bronchitis 65 + years	501	458	110
Hospital admissions Respiratory Disease 15-64 years	14	13	19

6.3.1 Health Effects Attributable to Coarse Particles - PM_{2.5-10}

Given that the coarse component of PM₁₀ is the predominant component in dust in Port Hedland a preliminary analysis of the health risk for PM_{2.5-10} has been undertaken. Using the ratio of PM_{2.5} to PM₁₀ at Richardson St, Taplin St and South Hedland the percentage of PM_{2.5-10} for each location was calculated. The resultant PM_{2.5-10} percentages are:

- Richardson St 82%
- Taplin St 80%
- South Hedland 92%

The higher percentage of coarse particles at South Hedland may reflect a greater contribution from combustion sources in Port Hedland which would be expected. These percentages were applied to the monitored PM₁₀ data to calculate the daily PM_{2.5-10} dataset.

A review of the epidemiological literature on the health effects of coarse particles was undertaken to identify exposure-response relationships that could be used to quantify the effects of coarse particles on particular health outcomes. Two meta-analyses were identified: (1) Southern Europe (Samoli et al., 2013) which is impacted by Saharan Desert dust, and (2) one from the US (Zanobetti and Schwartz., 2009) that studied 47 cities. The use of data from meta-analyses is consistent with the recommendations of NHMRC (2006) and NEPC (2011).

The exposure response functions for all-cause mortality were available from both studies. For respiratory and cardiovascular mortality exposure response functions were only available from the US study. The US study also conducted the analysis for cities with specific climatic conditions including hot continental summers. This data has been used in the analysis. The exposure response functions from the European study are very similar to those for the hot continental cities studied in the US.

For 2008-2012 for Port Hedland due to low numbers respiratory deaths not be included. The numbers of deaths attributable to coarse particles was undertaken for cardiovascular and all-cause mortality. The results are presented in **Table 15**. These results are lower than those attributed to total PM₁₀ but should only be considered as indicative. The reliability of the exposure response relationship for PM_{2.5-10} is not as strong as that for PM₁₀.

Table 15: Attributable deaths due to PM_{2.5-10} (per 100,000 population)

Outcome	Port Hedland using Richardson St data	Port Hedland using Taplin St data	South Hedland
Daily All-Cause Mortality	4.7	4.5	5
Daily Cardiovascular Mortality	1.7	1.6	1.8

6.3.2 Sea Salt

Sea salt in coastal communities is known to be a substantial component of dust. The amount of sea salt aerosol present is dependent on prevailing winds in the area. In Port Hedland as well as the presence of salt aerosol from the ocean, there is an industrial source of salt aerosol from Dampier Salt works. The composition if the salt from industrial sources will be the same as sea salt aerosol as the

source of the salt is the same – the ocean. The scope of the HRA identified consideration of sea salt in determining the health effects associated with PM₁₀ monitored in Port Hedland.

A review of the scientific literature in respect to sea salt and associated health effects is limited. Some studies that have used the results of source apportionment studies in epidemiological studies have found that sea salt is associated with an increased risk of hospitalisation for respiratory disease while others have found that there is no impact on any of the health outcomes studied. In these studies the health effects observed were linked with particle mass not individual components of the particles.

Based on the current literature it is not possible to conclude whether sea salt has an independent health effect apart from the effects associated with particle mass alone. Conversely it is not possible to conclude that it does not have an independent effect. Therefore in the HRA no adjustment has been made for sea salt in the assessment of potential health risk. It has been assumed that sea salt particles in the PM₁₀ size fraction will be the same as other particles based on particle size alone.

From an exposure perspective, the preliminary source apportionment study conducted by the WA ChemCentre (Muir, 2014) has identified that sea salt comprises 20-40% of the measured PM₁₀ in Port Hedland including South Hedland. The source apportionment could not differentiate between industrial sources and natural sources of salt.

6.3.3 Summary

The risk assessment for PM₁₀ has shown that there are substantial health risks associated with dust in Port Hedland. The risks per 100,000 population are higher when the data from Richardson St are used in the analysis. Both the Richardson St and Taplin St data lead to higher health risks than that calculated for South Hedland. Reducing the PM₁₀ levels to meet either the interim 70 µg/m³ guideline or the 50 µg/m³ NEPM standard leads to a statistically significant reduction in risks to the Port Hedland community.

Analysis of the risk of all-cause mortality attributable to PM₁₀ in the west end, east end and South Hedland shows that the risk per 100,000 population is higher in the west end of Port Hedland compared to the other locations. The increased risk of mortality attributable to PM₁₀ in the west end in 2013 was 42 per 100,000 population. This is higher than that calculated for greater Sydney which is 10 per 100,000 population.

It should be noted that the risk calculations have been conducted using only one year of data which was the only data available for the HRA. Using additional years of data would provide a more precise estimate of the risk posed by PM₁₀ in Port Hedland, however, if the PM₁₀ data does not vary significantly year to year it is unlikely to change the overall outcomes of this HRA.

7 HEALTH RISK ASSESSMENT METALS

In the scoping of the HRA four metals were identified to be potential pollutants of concern. These are Chromium (III), (Cr III), Chromium IV (Cr IV), Manganese (Mn) and Iron Oxide. Monitoring of these metals, along with a range of other metals including aluminium, arsenic, boron, barium, calcium, cadmium, cobalt, potassium, lithium, molybdenum, nickel, lead, sulphur, selenium, vanadium and zinc, was undertaken at nine locations operated by the PHIC monitoring network and industry monitoring sites:

- BoM site
- Richardson St
- Taplin St
- Moore St
- South Hedland
- Wedgefield
- Yule River
- Utah East
- Utah North

Yule River is considered to be a regional background site and provides data on levels of metals that are generally found in the dust in the Pilbara. South Hedland has been used as an urban background site that is not directly impacted by activities at the Port. Utah East and Utah South are industry monitoring sites and are located at the Port. Richardson St is representative of exposures in the west end of Port Hedland and Taplin St is considered representative of the east end.

Data was also made available from analysis conducted by the WA ChemCentre for which a number of additional metals were examined including: aluminium, arsenic, boron, barium, calcium, cadmium, cobalt, potassium, lithium, molybdenum, nickel, lead, sulphur, selenium, vanadium and zinc. The levels of most of these metals were below the level of detection of the analysis method.

7.1 Hazard Assessment

7.1.1 MANGANESE

Manganese is an essential nutrient however it does exhibit toxic effects if exposure is excessive or prolonged. Inhaled manganese enters systemic circulation directly, making the manganese available for distribution to and accumulation in the body's tissues, including the brain (Health Canada 2010). The health effects of manganese (Mn) have been reviewed by several agencies including the WHO (2000), USEPA (1996), OEHHA (2008) and the ATSDR (2012).

7.2 Epidemiological Studies

The main evidence of adverse effects of manganese in humans comes from long-term (chronic) exposure studies.

7.2.1 Neurological Effects

The main health endpoints of chronic exposure to manganese are neurobehavioral and neurological effects which are initiated by accumulation of Mn in the brain and can eventually lead to manganism (ATSDR 2012, OEHHA 2008). Manganism is defined as a progressive condition that usually begins with relatively mild symptoms, but evolves to include dull affect, altered gait, fine tremor, and sometimes psychiatric disturbances. Some of these symptoms also occur with Parkinson's disease, which has resulted in the use of terms such as "Parkinsonism-like disease" and "Mn -induced Parkinsonism" to describe those symptoms observed with Mn poisoning (ATSDR 2012, OEHHA 2008, WHO 2000).

Pathologically manganism is characterised by diffuse lesions found mainly in the pallidum, caudate nucleus, the putamen, and even the cortex with no effects on the substantia nigra and no Lewy bodies (Pal et al. 1999; Perl and Olanow 2007). Mn appears to affect pathways that are post-synaptic to the nigrostriatal system, most likely the globus pallidus (Chu et al. 1995). MRI of the brain reveals accumulation of Mn in cases of manganism and fluorodopa positron emission tomography (PET) scans come back normal in cases of manganism (Calne et al. 1994). Other studies suggest that Mn produces a syndrome described as Parkinsonism, distinct from Parkinson's disease or manganism (Lucchini et al. 2007, Racette et al. 2005).

The mechanism of brain accumulation has been discussed by Gavin et al 1999 who investigated the implications of Mn toxicity in the mitochondria specifically those found in the liver and brain where Mn is thought to accumulate (OEHHA 2008). The authors reported Mn is sequestered via the mitochondrial calcium uniporter where calcium binds to the external activation site increasing the velocity of uptake of both calcium and Mn. Over 97% of the Mn in the mitochondrial matrix is bound to the membrane or to a matrix protein. Mn transport out of the mitochondria is via the slow sodium independent efflux mechanism (the dominant efflux mechanism of the heart and brain mitochondria) however it was not substantially transported out.

Mn inhibited the efflux of calcium increasing the probability of the mitochondria undergoing the Mitochondrial Permeability Transition (MPT). Intra-mitochondrial Mn also inhibits state 3 mitochondrial respiration. The study data suggests that Mn depletes cellular energy supplies by interfering with the oxidative phosphorylation which could possibly lead to apoptosis in active neurons. Malecki 2001 investigated the direct effects of Mn exposure on striatal neurons and reported that 48 hours after exposure, neurons showed dose dependant (5, 50, 500 micronM) losses of mitochondrial membrane potential and complex II activity (50, 500 micronM). Neurons exposed to the lowest dose also exhibited DNA fragmentation and decreases in Microtubule Associated Protein (MAP-2). Similar to the Gavin et al 1999 study, the results indicate that Mn may trigger apoptotic like neuronal death secondary to mitochondrial dysfunction. HaMai et al 2001 further investigated the mechanism that may lead to neural apoptosis and found that the oxidative properties of Mn promoted formation of Reactive Oxygen Species (ROS) within the cortical mitochondrial-synaptosomal (P2) fraction which can cause oxidative stress. Oxidative stress has been linked to apoptosis of neurons in the animal studies.

Several conclusive studies in humans presenting evidence of manganism after high level Mn inhalation exposure are discussed below. Initial clinical signs of exposures to high levels of Mn are subjective and involve generalized feelings of weakness, heaviness or stiffness of the legs, anorexia, muscle pain, nervousness, irritability, and headache (Mena et al. 1967; Nelson et al. 1993; Rodier 1955; Tanaka and Lieben 1969; Whitlock et al. 1966). These are usually accompanied by apathy and dullness along with impotence and loss of libido (Abdel-Hamid et al. 1990; Emara et al. 1971; Mena et al. 1967; Nelson et al. 1993; Rodier 1955; Schuler et al. 1957). Early clinical symptoms include a slow or halting speech without tone or inflection, a dull and emotionless facial expression, and slow and clumsy movement of the limbs (Mena et al. 1967; Nelson et al. 1993; Rodier 1955; Schuler et al. 1957; Shuqin et al. 1992; Smyth et al. 1973; Tanaka and Lieben 1969). As the disease progresses, walking becomes difficult and a characteristic staggering gait develops. Muscles become hypertonic, and voluntary movements are accompanied by tremor (Mena et al. 1967; Rodier 1955; Saric et al. 1977a; Schuler et al. 1957; Smyth et al. 1973). These symptoms are largely thought to be irreversible however there has been some evidence suggesting that recovery may occur when exposure ceases (Smyth et al. 1973).

Psychomotor excitement has been reported in the high exposure occupational setting, mainly Mn mining. The behaviour is known as "Mn madness" and includes nervousness, irritability, aggression, and destructiveness, with bizarre compulsive acts such as uncontrollable spasmodic laughter or crying, impulses to sing or dance, or aimless running (Emara et al. 1971; Mena et al. 1967; Mena 1979; Rodier 1955; Schuler et al. 1957). Cases of Frank Manganism in workers have clearly indicated that the onset of manganism results from chronic exposure to high concentrations (2 – 22 mg Mn/m³) of the metal (Rodier 1955; Schuler et al. 1957; Smyth et al. 1973). Based on these studies, it appears that the frequency of manganism cases increased with prolonged exposure, suggesting that the seriousness

of the symptoms presented increases with cumulative exposure. Rodier (1955) reports that the highest percentage of manganism cases (28 or 24.4%) occurred in miners with 1–2 years' experience. Only six cases of manganism (5.2%) were reported in males with 1–3 months exposure, and 68% of the cases reported occurred after exposures >1–2 years in length. The study though suggestive of a cumulative effect of manganism neurotoxicity, the findings should be interpreted with caution as no statistics on the number of men in the mine who were employed for comparable durations who did not suffer from manganism were reported.

Studies investigating low level environmental Mn exposure on populations living in close proximity to Mn plants have also been presented. Mergler et al 1999 investigated the nervous system effects of Mn exposure in individuals living in close proximity to a former Mn production plant. Similar to their 1994 a battery of tests used to profile the nervous system function in relation to blood levels (2.5 – 15.9 µg/l) were used. Motor skills and coordination, learning and recall, visual perception and speed, verbal naming and cognitive flexibility were also assessed. Neurobehavioral deficits were much stronger in men than women with pronounced effects seen in older subjects suggesting that Mn neurotoxicity can be viewed as a continuum of dysfunction with progressively severe neurological disorders observed at higher exposure levels (OEHHA 2008).

Riojas-Rodríguez et al. (2010) investigated intellectual function with the revised Wechsler Intelligence Scale for Children and the Progressive Matrices of the Raven test for maternal intelligence in a population with an average Mn exposure of 0.13 µg/m³ for at least 5 years. Children in the exposed communities had significantly elevated mean blood (9.71 µg/L) and hair (12.13 µg/g) Mn concentrations compared with controls (8.22 µg/L and 0.57 µg/g, respectively). Statistically significant (p<0.05) negative associations were found between hair Mn concentrations and verbal and full scale scores. Blood Mn concentration was inversely, but non-significantly, associated with verbal and full scale scores. After adjusting for age and sex, the strongest inverse association between hair concentration and intellectual function was in young girls, with little evidence of associations in boys at any age. Associations with blood concentration were not modified by sex, but age adjustment suggested that the inverse relationship was limited to younger participants. These findings suggest that air-borne Mn exposure is inversely associated with intellectual function in young school-age children. Hernández-Bonilla et al. (2011) evaluated motor impairments (manual dexterity, (fine) motor coordination, and motor speed (using the grooved pegboard, finger tapping, and Santa Ana tests)) in the same children as those assessed in the Riojas-Rodríguez study. The authors found significant inverse relationship between execution of the finger tapping test with blood Mn concentration, but not hair Mn. Additionally, exposed children made substantially more errors in the grooved pegboard test than controls, but this effect was not associated with blood or hair Mn levels. There was no correlation between Mn concentration in blood and hair in any of the other motor function tests and only subtle evidence of adverse effects on motor speed and coordination were reported. Menezes-Filho et al. (2011) also assessed intellectual function in children and their caregivers (Wechsler Intelligence Scale for Children, version III. To assess intellectual function in primary caregivers (94% mothers), the Raven Progressive Matrix was administered) and reported mean blood and hair Mn concentrations in children were 8.2 and 5.83 µg/L, respectively and a negative association between hair Mn levels in children and their verbal and full scale scores. In addition, after adjusting for education years, family income, and age, there was a statistically significant (p<0.05) negative association between caregiver's hair Mn levels and performance on the Raven Progressive Matrix. High Mn exposure, had detrimental effects on cognition in both adults and children, especially in the verbal domain. However, poor cognitive development in children may also be due in part to lower caregiver IQs (ATSDR 2012).

Standridge et al. (2008) evaluated postural balance in residence from a Mn exposed community using postural sway analysis. The residents were exposed to an average of 0.1 and 2.0 µg/m³ Mn over 3 years and the analysis showed postural analysis measures of Mn -exposed residents were significantly larger than controls in five out of eight postural balance outcomes (sway area for eyes open on the platform, sway area for eyes open or closed on foam, sway length for eyes open or closed on the foam). After adjustment for covariables, a statistically significant positive association was found between hair Mn

levels and sway area and length (eyes open or closed on the platform). These preliminary findings suggest subclinical impairment in postural balance in Mn -exposed residents. Kim et al. (2011) evaluated motor function (Unified Parkinson's Disease Rating Scale, a postural sway test, and a comprehensive questionnaire) in the same population and found no statistically significant differences between the exposed and comparison groups in regards to Mn blood levels, demographics, or major health outcomes. However, when adjusted for covariates (presence of other neurotoxic metals, factors aggravating susceptibility to Mn or motor performance, demographics), the Mn -exposed residents had a significantly increased risk of abnormal performance on the Unified Parkinson's Disease Rating Scale and showed significantly higher postural sway scores. These findings may reflect early subtle effects of chronic, low-level Mn exposure, but alternatively might be due to chance due to the cross-sectional study design, the small to medium effect size, and the lack of association between air or blood Mn levels and motor function performance (ATSDR 2012).

Rodríguez-Agudelo et al. (2006) examined neurobehavioral end points ("Esquema de Diagnóstico Neuropsicológico" Ardila and Ostrosky-Solís's neuropsychological battery to evaluate motor functions; a Spanish adaptation of Luria diagnostic procedures were administered) in men and women from eight communities at various distances from Mn extraction or processing plants exposed to a range of 0 to 5.86 $\mu\text{g Mn /m}^3$. No associations were found between neuromotor performance and blood levels of Mn. The study concluded that there is an incipient motor deficit in the population environmentally exposed to large Mn levels. Solís-Vivanco et al. (2009) evaluated the same group of individuals with a battery of neuropsychological tests for cognitive function (general cognitive state, attention, semantic and phonological fluency, construction, verbal memory, visual memory coding and recall, and depression) and found no risk of poor performance with a 0.05 $\mu\text{g/m}^3$ cut-off point. When using a 0.1 $\mu\text{g/m}^3$ cut-off point, only 1 of 10 cognitive measures had a significantly increased risk of poor performance (attention as measured by the digit span test). The attention impairments associated with high levels of air Mn exposure are evidence of cognitive impairment in the exposed population. However, similar to the study by Rodríguez-Agudelo et al. (2006), the finding on this one measure could be due to chance, as there was no association between blood Mn levels and cognitive performance (ATSDR 2012).

Bowler et al 2012 investigated the occurrence of anxiety in a population living in an area with elevated Mn in air. A random cohort of residents of Marietta Ohio exposed to an average on 0.18 $\text{g}/\mu\text{m}^3$ Mn were administered the Unified Parkinson's Disease Ratings Scale (UPDRS), motor efficiency and mood tests along with a comprehensive questionnaire. Their blood Mn levels were also measured and found to be similar to the control group. This cohort reported generalized anxiety related to the cumulative exposure index ($p=0.002$) based on the modelled Mn air concentration and length of residence. The study findings suggested an association between environmental Mn exposure and anxiety states however whether the association was due to neurotoxic effects of Mn in air or a concern about health effects of air pollution remains an open question.

In another study by Kim et al 2011 motor function in adults from a Mn exposed community in Ohio was evaluated using the UPDRS test, a postural sway test and a comprehensive questionnaire. No statistically significant differences between the exposed and comparison groups were evident and the risk of abnormal UPDRS performance was increased in the exposed group and high postural sway scores under eyes open conditions were reported. No participants however were diagnosed with clinical manganism by neurological examination. These findings may possibly reflect early subtle effects of chronic low-level Mn exposure however the study is weak and therefore there are limitations to the causal relationship.

Torres-Agustin et al 2013 assessed the effect of Mn exposure on verbal memory and learning in 7- to 11-year-old children from a mining town using the Children's Auditory Verbal Learning Test (CAVLT). Blood and hair samples were also obtained to determine Mn concentrations. The exposed group presented higher hair and blood Mn ($p<0.001$) than the non-exposed group (median 12.6 vs. 0.6 $\mu\text{g/g}$, 9.5vs. 8.0 $\mu\text{g/L}$ respectively), as well as lower scores ($p<0.001$) for all the CAVLT subscales. Hair Mn was inversely associated with most CAVLT subscales, mainly those evaluating long-term memory and

learning ($\beta=-0.47$, 95% CI -0.84, -0.09). Blood Mn levels showed a negative but non-statistically significant association with the CAVLT scores. The results suggest that Mn exposure has a negative effect on children's memory and learning abilities.

Overall new evidence shows that there is a need to characterise preclinical effects of manganese exposure especially in communities living in proximity to areas with high Mn-air concentrations and further work is required to characterise the effects, neonatal and post-natal, in children they are a susceptible population for manganese exposure.

7.2.2 Hematopoietic Effects

There have been inconsistent results reported regarding the effects of Mn exposure on erythrocyte superoxide dismutase activity. Yiin et al 1996 investigated whether plasma Mn concentration is associated positively with the product of lipid peroxidation and whether lipid peroxidation is associated negatively with the activities of antioxidants in exposed workers. 22 exposed Mn smelter workers and 45 controls had their blood collected and plasma separated for analysis. Malondialdehyde (a product of lipid peroxidation) was used as a biomarker and measured while antioxidants superoxide dismutase (SOD), Glutathione peroxidase (GPX) and Catalase (CAT) activities were determined to establish indices of lipid peroxidation and reactive oxygen metabolites. The activities of SOD and concentration of malondialdehyde were elevated in exposed workers and these had a strong correlation with Mn plasma levels. Li et al 2004 determined Mn levels and subsequent oxidative stress status in the body fluids of welders exposed to an average of 1.45 mg/m³ Mn. They reported 4.3 fold increase of Mn in the serum of welders when compared to controls, a 24% decrease in erythrocytic SOD activity and a 78% increase in serum malondialdehyde levels. Though the SOD activity was conflicting between both studies, the increased malondialdehyde was consistent and as such indicated that a possible mechanism of Mn toxicity could be through lipid peroxidation.

7.2.3 Immunological Effects

Boshnakova et al 1989 subjected 74 welders to immunological screening and measured serum immunoglobulins (IgG, IgA, IgM) and total and active E-rosette-forming cells (E-RFC). The authors reported suppressed T and B lymphocyte immune systems, expressed by decreased levels of serum IgG and total E-RFC. These findings however were not conclusive because the welders were exposed to other compounds that may have contributed to the observed effect.

7.2.4 Reproductive Effects

Impotence and libido in men are some reported symptoms of manganism and are attributed to high Mn exposure for 1 – 21 years. These symptoms subsequently lead to reduced reproductive success however the evidence is conflicting and of the few studies (Wu et al 1996, Jiang et al 1997a, Chandra et al 1973, Seth et al 1973) that support this outcome, none have established a dose response relationship or effect threshold. Wu et al 1996 examined 211 workers (miners/ ore processors, electric welders in mechanical fields and in ship building) exposed to 0.14 to 82.3 mg Mn/m³ for ≥ 1 year and found that they exhibited increased semen liquefaction time and decreased sperm count and viability. Though Mn concentrations in the semen were increased, so were the concentrations of other metals such as copper, nickel, chromium and iron. As such the results of this study are not conclusive. Jiang et al 1997a examined men from a Mn plant exposed to an average on 0.145 mg Mn /m³ for up to 35 years. No statistically significant reproductive outcomes were reported however reports of impotence and lack of sexual desire were prevalent. ATSDR suggests that the reported effects may occur as a secondary result of neurotoxicity rather than direct effects of high Mn exposure. Chandra et al 1973 and Seth et al 1973 reported severe degenerative changes in the seminiferous tubules leading to sterility in rabbits given a high dose of Mn (158 mg/kg) by intra-tracheal instillation. The effects were not immediate but developed over 4 to 8 months.

Direct damage to the testis has not been reported in occupational human studies suggesting it may not be of concern to humans however it remains unclear whether such studies have been carried out

in humans. It is important to note that no information regarding reproductive effects in women was found.

7.2.5 Developmental Effects

There is very limited information on developmental effects after Mn inhalation exposure. Hernández-Bonilla et al 2011 reported that children living in a Mn mining area had higher Mn hair concentrations than children from a non-mining area, but did not show clear performance deficits on several tests of motor skills when compared with the control group of children. These results suggest an association between environmental exposure of children to Mn and impaired cognitive abilities, but are inadequate to establish causal relationships due to the cross-sectional design and inability to control for possible confounding factors. OEHHA found a correlation between early life exposure to high levels of Mn and impaired neurodevelopment based on the following studies. Takser et al 2003 found an inverse correlation between cord blood Mn at birth and three subscales of psychomotor development (McCarthy scales of children's abilities) measured at 3 years of age i.e. attention (partial $r = -0.33$, $p < 0.01$), nonverbal memory (partial $r = -0.28$, $p < 0.01$), and hand skills (partial $r = -0.22$, $p < 0.05$). The adverse effects of manganese on neurodevelopment in these children persisted after adjustment for gender and maternal education, although the effects of manganese on hand skills were only observed in boys. Ericson et al 2007 measured the amount of Mn in developing teeth as an indicator of gestational exposure to Mn and found a positive correlation between exposure and behavioural disinhibition at 3 and 4.5 years old. The results show that high prenatal Mn exposure may adversely affect behaviour expressed postnatally. Collip et al 1983 and Zhang et al 1995 further support these study outcomes as they both found an association between elevated hair Mn levels (0.434 $\mu\text{g/g}$ and 1.242 $\mu\text{g/g}$) and hyperkinetic and exhibited learning disabilities in children.

7.2.6 Endocrine Effects

Alessio et al 1989 evaluated neuroendocrinal tests (analysing FSH, LH, prolactin, and cortisol) in 14 male workers exposed to 0.04 – 1.1 mg Mn/m³ (particulate matter) and 0.05 – 0.9 mg Mn/m³ (fumes) over 10 years. They reported elevated prolactin and cortisol levels but no changes in the Follicle Stimulating Hormone (FSH) and Luteinising Hormone (LH) levels.

7.3 Toxicological Studies

7.3.1 Neurological effects

Studies discussing the effects and subsequent accumulation of Mn in the brain after acute exposure have been identified (OEHHA, 2008). Newland et al 1987 investigated the clearance of manganese chloride (⁵⁴MnCl₂) in three macaque monkeys. The authors exposed two monkeys to trace amounts of ⁵⁴MnCl₂ by inhalation for 30 minutes and monitored their chest, head and faecal radioactivity over a year. Head levels peaked 40 days after exposure and remained elevated for the year. The kinetic analyses suggested that the long half-times of Mn in the head following inhalation reflected both slow disappearance from the head and replenishment from other depots.

Brenneman et al 2000 investigated the direct olfactory transportation of ⁵⁴MnCl₂ (0.54 mg Mn/m³; MMAD 2.51 μm) by inhalation after a single 90 minute intranasal instillation in rats. High levels of ⁵⁴Mn were observed in the olfactory bulb and tract/tubercle demonstrating that the olfactory route contributes up to 90% of the ⁵⁴Mn found in the olfactory pathway but not in the striatum of the rat brain up to 8 days following single instillation.

Dorman et al 2002 further investigated olfactory transport of Mn to the brain and evaluated the olfactory uptake and direct brain delivery of inhaled manganese phosphate (⁵⁴MnHPO₄ (0.39 mg Mn/m³; MMAD 1.68 μm)) in male rats after a 90 minute instillation.

The olfactory pathway, striatum, cerebellum and rest of the brain were evaluated immediately after exposure and 1, 2, 4, 8 and 21 days post exposure. Mn was detected in the olfactory bulb and striatum

with increased activity noted in the olfactory bulb and tubercle. The study findings demonstrate that the olfactory route contributes to Mn delivery to the rat olfactory bulb and tubercle. Though the pathway did not significantly contribute to striatal Mn concentrations post exposure. OEHHA concluded that pulmonary oedema, pulmonary impaired function and Mn accumulation through the olfactory pathway occur as a result of acute inhalation exposure to Mn.

In an animal study by Lown et al 1984, dams and non-pregnant mice were exposed to an average of 61 mg Mn/m³ for 16 weeks prior to conception, then exposed to air or Mn post conception irrespective of preconception exposure. The pups were nursed in the absence of Mn exposure and on evaluation on postpartum day 7 weight gain and gross locomotor activity and on day 45 for different behavioural parameters and learning performance, pups from mothers exposed to Mn pre conception and air post conception had reduced weights compared to pups from mother only exposed to air pre and post conception. There was no observable difference in activity between pups who had been exposed to Mn in utero and those that had not. Therefore, the data did not provide evidence that Mn exposure resulted in adverse neurological developmental effects.

7.3.2 Respiratory Effects

ATSDR (2012) report that people exposed under occupational settings after acute Mn exposure may exhibit a lung inflammatory response characterised by increased macrophage and leukocytes presumably in Broncho alveolar fluid (BALF) and some lung tissue damage. This is based on the study by Bergstrom 1977 who exposed male and female guinea pigs to 22 mg/m³ manganese dioxide (MnO₂) aerosol over a 24 hour period and observed a rapid clearance of MnO₂ from the lungs, a significant decrease in the number of macrophages immediately after exposure, an increase in leukocyte counts 1 and 3 days post exposure and increased phagocytic capacity of the population of alveolar macrophages. These results indicate a primary inflammatory reaction occurs in the respiratory tract after acute exposure to MnO₂. Such exposure subsequently increases lung susceptibility to infection by bacterial pathogens as reported by Maigetter et al 1976. The latter authors exposed mice to MnO₂ aerosol for single and multiple 3-hour-long exposures for 3 to 4 days and challenged their immune systems by exposing them to airborne *Klebsiella pneumoniae* within 1 hour and 5 hours post exposure. The mice had increased mortality rates, reduced survival times and altered resistance to infection. Bredow et al 2007 reported an increase in pulmonary levels of mRNA for Vascular Endothelial Growth Factor (VEGF) – a proliferation regulator- when female GVB/N mice were exposed to 2 mg manganese/m³ as manganese chloride aerosols 6 hours/day for 5 consecutive days.

OEHHA (2008) identified pulmonary oedema and impaired function as the main endpoints of continued MnO₂ dust exposure based on the Shiotsuka 1984 sub-chronic dose response study. These findings were supported by the outcomes of two acute studies; Adkins et al 1980 who exposed female mice to manganese oxide aerosols over a 2 hour period and observed general systemic distribution of Mn and respiratory effects (oedema) with a NOAEL of 2.9 mg/m³ and the Bergstrom 1977 study as described in ATSDR (2012) reported reversible respiratory inflammation and pulmonary dysfunction as intermediate respiratory effects of Mn exposure based on 2 studies. First, Dorman et al 2004 who reported reversible inflammation in the nasal respiratory epithelium of rats exposed to 0.01, 0.1 and 0.5 mg Mn/m³ for 13 weeks. 45 days post exposure, no respiratory lesions were observed indicating their transient nature. Second, Dorman et al 2005 found an association between Mn exposure and pulmonary dysfunction when they exposed a set of male rhesus monkeys to manganese sulphate (MnSO₄) (0.06, 0.3, 1.5 mg Mn/m³) for 13 weeks. A second set of monkeys were only exposed to the highest exposure and held for 45 or 90 days post exposure while a third set was exposed to the higher dose and held for 15 or 30 days post exposure. Histopathological assessments of the lungs were carried out as well as Mn content determination of lungs and olfactory epithelium. For the first group of monkeys, Mn levels in the olfactory bulb were elevated at all exposures and in the lungs at ≥ 0.3 mg/m³. Significant bronchiolitis, alveolar duct inflammation, increased bronchus-associated-lymphoid tissue and elevated Mn levels in the lungs and olfactory bulb were reported in monkeys exposed to the higher dose however all these effects were reversed 45 to 90 days post exposure.

Reversible inflammatory changes were only reported in the higher exposure groups and as such the authors suggest the lungs are a less sensitive target for Mn toxicity when compared to the central nervous system. Though the findings of Dorman et al 2004; 2005 are consistent with an inflammatory response in respiratory tissues, ATSDR notes that the responses are not unique to Mn containing particles but are characteristic of nearly all inhalable particulate matter.

7.3.3 Iron Oxide

Iron oxide is of concern with respect to the potential exposure with air in the Port Hedland area, given that iron ore is the major export from the port. It is recognised that there is information available concerning elemental iron, carbonyl iron as examples; however, the available literature suggests that the behaviour of these other species differs from that of iron oxide. Iron oxide has been noted to generate reactive oxygen species in the oxygen rich lung, which is the source of its potential effects via inhalation (e.g., inflammation). An additional confounder in understanding and describing the potential effects related to the inhalation of iron oxide, is the source of iron on which each of the available toxicological/epidemiological studies are based. Many have examined sources such as smelters (Zhou et al., 2001 – EPA 2012 PM update doc), production of iron/steel (Chan et al., 2010; Kamal et al., 2011; Rohr et al., 2011 EPA 2012 PM update doc), underground train stations (Seaton et al., 2005), which are unrelated to iron oxide as represented in Port Hedland. Additionally, there is concomitant exposure to other metals in each of the studies, as a result caution must be taken in interpreting the results of these studies with respect to the potential effects related only to iron oxide.

There were two major reviews related to exposure to iron oxide that were identified. The first review is via the US EPA's Integrated Science Assessment for Particulate Matter (US EPA, 2009), which examined the effects of various sources of iron and different species of iron particulate. The other is the Chemical Safety Report (CSR) year compiled under the European REACH program which examined the potential effects due to carbonyl iron, described as an "inert dust", which is not soluble when it came to inhalation exposures. The inert nature of the particles of carbonyl iron was suggested to be due to the fact that they may be protected from chemical reactions due to them being available in a coated form. As a result, this model would not be relevant for iron oxide at Port Hedland. Given the different form of iron examined in the CSR, it is not appropriate to rely on the studies examined or its conclusions with respect to the potential effects on human health, and as such the report has not been considered further in this review.

Prolonged inhalation of high concentrations of fine particles of metallic iron, or iron compounds in an occupational situation causes pulmonary siderosis (ACGIH, 2006). This is a relatively benign pneumoconiosis, characterised by a large accumulation of inorganic containing macrophages in the lungs with minimal reactive fibrosis. In its pure form (i.e. due to iron oxide exposure only) the condition probably does not progress to true nodulation, as seen with silicosis and is usually asymptomatic, it does however show up as abnormal changes on X-rays (McLaughlin et al. 1945, Teculesu and Albu 1973, Morgan 1978, Brooks 1986, Sentz and Rakow 1969).

When iron is inhaled with other fibrogenic mineral dusts pulmonary fibrosis can be induced (ACGIH 2006). This is called mixed dust pneumoconioses or silicosiderosis. Haematite pneumoconiosis occurs in iron miners who are exposed to iron oxide in combination with free silica and silicates. It is characterised by a brick red coloured lung surface and has been likened to a simple form of coal workers' pneumoconiosis (Brooks 1986).

The best review of the available information concerning the mechanisms of action and effects following exposure to iron oxide have been reviewed by the ACGIH (2006) in deriving their Threshold Limit Value. The following provides a summary of the information provided in the supporting document from the ACGIH. In a study by Keenan et al. (1989), a mild inflammatory response was noted with the instillation of 3 mg of iron oxide into the lungs of hamsters. In a study by Das et al., (1983) the authors

noted that was no evidence from either single or repeated exposures of iron oxide that it produced irreversible changes in the lungs (doses up to 50 mg in guinea pigs), however, alveolar macrophages and both intracellular and extracellular iron oxide particles were observed. Alveolar macrophages lavaged from the lungs of hamsters exposed to iron oxide aerosols at a concentration of 274 mg/m³ for 3 hours were increased in numbers and exhibited increased rates of phagocytosis (Kavet et al., 1978). As study by Grant et al. (1979) also confirmed that results from the study of Kavet et al. (1978). In study examining the inflammatory response at lower concentrations (20 mg/m³ for 2 hours), iron oxide did not cause a significant change in the number of alveolar macrophages of PMNs, although the authors noted that phagocytic activity of the macrophages was enhanced (Lehnert et al., 1985).

In examining the case reports and studies outlined in the review by the ACGIH (2006), it is noted that while the studies outlined examine workers mainly exposed to iron oxide, they were also exposed to other compounds, which may also have contributed to the effects observed. As a result it is difficult to draw a conclusion regarding the relationship between exposure to iron oxide and the observed effects, which are described as generalised discrete densities in chest x-ray films (e.g., pulmonary siderosis) and pneumoconiosis. In addition to effects on the lungs, some incidences of contact dermatitis have been infrequently reported in the handling of pure iron oxide (Zugerman, 1985; Saxena et al., 2001; Motolese et al., 1993).

A number of epidemiological studies have examined the prevalence of adverse health effects in association with long-term occupational exposure to iron oxide. In a study by Kleinfeld et al. (1969) they reported that they found evidence of siderosis in 8 of 25 welders who were exposed to iron oxide for an average period of 18.7 years. Exposures to iron oxide measured during the study ranged from 30 to 47 mg/m³ in the breathing zone samples. In a study by Faulds (1957) it was reported that some hematite miners exposure to mixed dust developed massive pulmonary fibrosis. It was also reported that there was an increased incidence of lung cancer in this group, with the author suggesting that inhalation of iron oxide was a factor leading to the development of lung cancer. Similar results were reported in several other studies (Boyd et al., 1970; Braun et al., 1960; Jorgensen, 1973), however it should be noted that in these studies mixed exposures occurred that included silica, radon gas, and diesel exhaust.

A study by Chen et al. (1989; 1990) investigated the rate of non-malignant respiratory disease and the mortality experience of hematite mine workers in China. The results of the study demonstrated that the presence of respirable crystalline silica and not hematite was the cause of the silicosis. On this basis, the results of this study with respect to the relevance to the effects of hematite exposure are unknown. A similar result was noted in two studies undertaken by Koskela et al. (1976) and Gibson et al. (1977) in that an increase in lung cancer incidence was noted in workers, however there was concomitant exposure to several other compounds such as silica. A retrospective cohort study by Lawler et al. (1985) followed 10430 iron oxide workers in Minnesota. The authors noted significant deficits in overall mortality rates and for a number of specific causes including respiratory diseases. The authors reported no excesses of lung cancer. It is of note that the study controlled for a number of confounders identified in many other studies of iron oxide workers such as smoking, level of radon, diesel exhaust, as well as silica. On the basis of the available studies regarding the carcinogenic potential of iron oxide, IARC have noted that it is "not classifiable" as a human carcinogen.

In examining some of the earlier studies where exposure was to iron oxide dust alone, two (Teculescu and Albu 1973, Sentz and Rakow 1969) contain information on exposure concentrations. In the first (Teculescu and Albu 1973), subjects were male workers in a plant manufacturing pure red iron oxide ('rouge'). Dust concentrations (30% was <1 µm, 45% 1-3 µm, 23% 3-5 µm, and 2% 5-10 µm in diameter) varied according to the place and phase of the production process. They were 10 to 15 mg/m³ in the chemical reaction and filter room, 45 to 700 mg/m³ in the drying and mill room, 306 to 770 mg/m³ in

the calcination room, and 330 to 500 mg/m³ in the packing room. The silica content was negligible (<1%). Clinical and X-ray investigations were made in 1965, and repeated in 1967 and again in 1969. A high prevalence of respiratory symptoms was found related to the smoking habits of subjects, but X-ray changes were also found. In the last survey (Sentz and Rakow, 1969), 38 of the 113 workers had opacities on their standard chest film. Comparison with earlier films revealed progression in 41%, regression in 20% and no change in the rest over a 3-year interval. Fourteen subjects of those with nodular shadows, who had not been exposed to other dusts or noxious gases, were selected to undergo pulmonary function tests. It is not stated in the paper which exposure group these subjects belonged to (i.e. chemical reaction, drying, calcination, or packing room concentrations). They had been exposed to iron oxide dust for 4-13 (mean 10) years. The group included four smokers, three ex-smokers and seven non-smokers. The authors found no restrictive ventilatory impairment in pulmonary function tests and the static lung compliance was normal. The only effects observed were slight hypoxemia at rest in one subject and a fall in the transfer coefficient in another; these were attributed to chronic bronchitis and recent respiratory disease, respectively. This study indicates long-term exposure to respirable particles (<10 µm) of pure iron oxide dust between 10 and 770 mg/m³ is associated with minimal changes on X-ray diagnosis that are potentially reversible, but not with decrements in pulmonary function. This is consistent with another study (Sentz and Rakow 1969), in which electric arc and powder-burning workers exposed to iron oxide fume well over 10 mg/m³ had no discernible changes in their chest X-rays. It is unknown if this study investigated pulmonary function.

In examining more recent studies concerning the mechanism of action and adverse effects related to iron oxide exposure, in a study by Beck-Speier et al. (2009) they investigated the modulation of PM-induced inflammation by leached off metals through examination of the intracellular solubility of radio-labelled iron oxide particles (0.5 – 1.5 µm geometric mean diameter). The authors noted that alveolar macrophages from Wistar rats exposed to 1.5 µm particles (10 µg/ml) for 24 hours increased IL-6 release and also PGE2 synthesis. Inhibition of PGE2 synthesis by indomethacin caused a pro-inflammatory phenotype as noted by increased IL-6 release from alveolar macrophages exposed to 0.5 µm particles. In the rat lungs, 1.5 but not 0.5 µm particles (4.0 mg/kg) induced neutrophil influx and vascular permeability. The authors concluded that iron oxide particle-induced neutrophilic inflammatory cytokine release in vivo and pro-inflammatory cytokine release in vitro might be modulated by intracellular soluble iron via PGE2 synthesis.

Lay et al. (2001) postulated that inhaled iron oxide particles with associated amounts of soluble iron should induce mild pulmonary inflammation and lead to altered alveolar epithelial integrity and altered gas exchange. The authors noted that pulmonary inflammation secondary to oxidant generation catalysed by transition metals associated with inhaled particles is one factor believed to underlie the acute health effects of particulate air pollution. On this basis, the authors investigated the effects of inhaled iron oxide particles on alveolar epithelial permeability. Sixteen health subjects inhaled aerosols of iron oxide (1.5 µm mass median aerodynamic diameter) having either high or low water-soluble iron content. (3.26 and 0.14 µg soluble iron/mg of particles, respectively) for 30 minutes at an average mass concentration of 12.7 mg/m³. Alveolar epithelial permeability was assessed by measuring the pulmonary clearance of an inhaled radiolabeled tracer molecule (^{99m}Tc-DTPA, diethylene triamine pentaacetic acid) using a gamma camera at ½ hour and 24 hour post particle exposure. Carbon monoxide lung diffusing capacity (DL CO) and spirometry were also performed before and after breathing the iron oxide. As a control, on a separate day, the procedures were duplicated except that the subject breathed particle-free air. The results noted that those subjects breathing aerosols with high soluble iron, there was no significant difference in DTPA clearance between the exposed and controls at 30 mins or 24 hours post inhalation. In the case of the low soluble iron content the authors noted that there was also no significant difference in DTPA. With respect to spirometric measurements, only minor differences were noted and were not statistically different. Based on the above, the authors concluded that the inhalation of iron oxide particles at a

concentration of 12.7 mg/m³ did not cause an appreciable alteration of alveolar epithelial permeability, lung diffusing capacity, or pulmonary function in healthy subjects.

In a study by Pauluhn (2009), they examined the pulmonary toxicokinetics and toxicodynamics of synthetic iron oxide black (pigment grade in Wistar rats). The fate of the particles was studied during a 3-6 post-exposure period (1.5µm mass median aerodynamic diameter). The results of the study noted that there is strong evidence that pulmonary toxicity (characterised as inflammation) corresponds best with the mass-based cumulative lung exposure dose, as evidenced by the increase in relative and absolute counts of neutrophilic granulocytes, as well as the total cell counts in the bronchoalveolar lavage.

In a follow up study from Pauluhn (2012), Wistar rats were exposed to pigment-sized iron oxide dust in a subchronic 13-week inhalation study according to OECD testing guidelines TG#413 and GD#39. Animals were exposed 6 hours per day, 5 days per week for 13 consecutive weeks at actual concentrations of 0, 4.7, 16.6 and 52.1 mg/m³ (mass median aerodynamic diameter ~1.3 µm, geometric standard deviation = 2). The exposure to iron oxide dust was tolerated without mortality, consistent changes in body weights, food and water consumption or systemic toxicity. With respect to hematology, minimally increased differential neutrophil counts in peripheral blood were noted. The author noted that elevations of neutrophils in bronchoalveolar lavage (BAL) appeared to be the most sensitive endpoint examined. Histopathology demonstrated responses to particle deposition in the upper respiratory tract (goblet cell hyper- and/or metaplasia, intraepithelial eosinophilic globules in the nasal passages) and the lower respiratory tract (inflammatory changes in the bronchiole-alveolar region). Consistent changes suggestive of pulmonary inflammation were evidenced by BAL, histopathology, increased lung and lung-associated-lymph node (LALN) weights at 16.6 and 52.1 mg/m³. Increased septal collagenous fibers were observed at 52.1 mg/m³. Particle translocation into LALN occurred at exposure levels causing pulmonary inflammation. In summary, the retention kinetics iron oxide reflected that of poorly soluble particles. The empirical no-observed-adverse-effect level (NOAEL) and the lower bound 95% confidence limit on the benchmark concentration (BMCL) obtained by benchmark analysis was 4.7 and 4.4 mg/m³, respectively, and supports an OEL (time-adjusted chronic occupational exposure level) of 2 mg/m³ (alveolar fraction).

Investigation of the effects of acute inhalation exposure to iron oxide nanoparticles (15 – 20 nm particle size) was undertaken by Srinivas et al. (2012). The study examined the effects of a continuous 4 hour inhalation exposure of only the head and nose to a concentration of 640 mg/m³. Markers of lung injury and proinflammatory cytokines (interleukin-1β, tumor necrosis factor-α, and interleukin-6) in bronchoalveolar lavage fluid (BALF) and blood, oxidative stress in lungs, and histopathology were assessed on 24 hour, 48 hour, and 14 days of post-exposure periods. The authors noted a significant decrease in the cell viability, with the increase in the levels of lactate dehydrogenase, total protein, and alkaline phosphatase in the BALF. Total leukocyte count and the percentage of neutrophils in BALF increased within 24 hours of post-exposure. Immediately following acute exposure, rats showed increased inflammation with significantly higher levels of lavage and blood proinflammatory cytokines and were consistent throughout the observation period. Iron oxide nanoparticles exposure markedly increased malondialdehyde concentration, while intracellular reduced glutathione and antioxidant enzyme activities were significantly decreased in lung tissue within 24-hours post exposure period demonstrating the generation of reactive oxygen species (e.g., oxidative stress). On histological observation, the lung showed an early activation of pulmonary clearance and a size-dependant biphasic nature of the iron oxide nanoparticles in causing the structural alteration. Collectively, our data illustrate that iron oxide nanoparticle inhalation exposure may induce cytotoxicity via oxidative stress and lead to biphasic inflammatory responses in Wistar rat.

In another study conducted by Szalay et al. (2012), the potential adverse effects due to exposure to iron oxide nanoparticles were investigated. In *in vivo* experiments the effects of nanoparticles were monitored in adult male Wistar rats following a single intra-tracheal instillation. The rats were exposed to a physiological saline solution containing 1 and 5 mg/kg bw of iron oxide nanoparticles. Lungs and internal organs underwent histopathological examination following 1, 3, 7, 14 and 30 days. The mutagenic effects of the iron oxide nanoparticles was evaluated by bacterial reverse mutation assay on *Salmonella typhimurium* TA98, TA100, TA1535 and TA1537 strains and on *Escherichia coli* WP2uvrA strain both with and without metabolic activation. The authors noted that there were no pathological changes in examined internal organs, except a very minor pulmonary fibrosis developing by the end of the first month in the treated rats. While *in vitro* the MTT assay showed a moderate cytotoxic effect, the iron oxide nanoparticles did not demonstrate a mutagenic effect in the bacterial systems tested.

A study by Ghio and Cohen (2005) noted effects Fe(3+) has a high affinity for oxygen-donor ligands and will react with these groups at the particle surface. Retained particles accumulate metal from available sources in a cell and tissue and this was postulated by the authors that this complexed iron mediates oxidant generation. The authors noted that there are several other ways by which metal homeostasis in the lower respiratory tract can be disrupted following exposure to ambient air pollution particles to affect an oxidative stress. The authors concluded an association between metal equilibrium in the lower respiratory tract and biological effect in the lung could explain the observed differential toxicity of ultrafine, fine and coarse particles and disparities in host susceptibility.

7.3.4 Copper

Copper is an essential nutrient that is incorporated into a number of metallo-enzymes involved in haemoglobin formation, drug/xenobiotic metabolism, carbohydrate metabolism, catecholamine biosynthesis, the cross-linking of collagen, elastin, and hair keratin, and the antioxidant defence mechanism. The USEPA (1991) concluded that copper and copper compounds was not classifiable in relation to carcinogenicity due to lack of human studies and inadequate animal studies. Most health effects associated with exposure to copper are gastrointestinal system such as nausea, vomiting, and/or abdominal pain. It also affects other organs to a lesser extent i.e. respiratory, hepatic and immune system (ATSDR 2004). The health effects of Copper (Cu) has been reviewed by the RIVM (2001) and OEHHA (2008) and in more detail by the ATSDR (2004).

7.3.4.1 Acute Effects

There were very few studies on the acute effects of Cu by inhalation exposure. Whitman et al 1957 and 1962 reported acute effects of Cu dust exposure in workers exposed to 0.02 – 3.0 mg/m³ Cu. Subjects exhibited reactions including metallic or sweet taste, upper respiratory tract irritation, and nausea. Upper respiratory irritation has also been reported, in addition to fever, dyspnea, chills, headache, nausea, myalgia, cough, shortness of breath, a sweet metallic taste, and vomiting, in factory workers exposed to Cu fumes for 1 to 10 hours as a result of cutting pipes known to contain Cu (Armstrong et al., 1983).

Several animal studies on acute Cu exposure were identified by OEHHA (2008) and ATSDR (2004). Drummond et al (1986) studied the potential of Cu to induce respiratory effects in mice and hamsters exposed to 0.12 mg Cu/m³ and 3.3 mg Cu/m³ respectively. The hamsters showed decreased cilia beating after a 3 hour exposure whilst the mice showed increasing alveolar thickening after a 3 hours/day 5 days/week for 1–2 weeks exposure.

Skornik and Brain, (1983), reported on the effects of copper sulphate (and other metal sulphates) aerosols on the respiratory defence mechanisms in male hamsters. Hamsters were exposed to a single 4-hour inhalation exposure of 0, 0.3, 3.2, 4.0, 5.8 and 7.1 mg Cu/m³. *In vivo* uptake of radioactive colloidal gold 1, 24, or 48 hours after exposure was used to measure pulmonary macrophage

phagocytic rates. Hamsters exposed to doses greater than 3.2 mg Cu/m³ showed a significant, dose dependant, reduction in macrophage endocytosis however this returned to control levels after 48 hours.

Immunological and Lymphoreticular effects were reported by Drummond et al. 1986. The author exposed mice to 0.56 mg Cu/m³ for 3 hours or 0.13 mg Cu/m³ for 3 hours/day, 5 days/week for 2 weeks followed by an aerosol of *Streptococcus zooepidemicus* which resulted in an impaired immune response. When mice were exposed to 3.3 mg Cu/m³ for 3 hours or 0.12 mg Cu/m³ for 3 hours/day, 5 days/week for 2 weeks following exposure to an aerosol of *Klebsiella pneumonia*, decreased bactericidal activity of alveolar macrophages was also observed.

7.3.4.2 Intermediate Effects

The ATSDR (2004) and OEHHA (2008) identified several reports of occupational diseases after both intermediate and chronic exposure to Cu. Metal fume fever characterised by chills, fever, aching muscles, mouth and throat dryness and headaches has been reported by several authors. Gleason (1968) noted that in the lapping (polishing) of Cu plates, unexpected exposures to Cu dust were found to occur and the symptoms of metal fume fever were observed in workers exposed for an unspecified number of weeks to 0.075 – 0.12 mg/m³.

Vineyard Sprayers Lung is another occupational disease associated with Cu inhalation exposure. Cortez Pimentel and Marques (1969) and Plamenac et al. (1985) published case reports with no concentration-response information of findings from the alveolar lavage and biopsy of these workers. Common findings include intra-alveolar desquamation of macrophages, formation of histiocytic and noncaseating granulomas containing inclusions of Cu, and healing of lesions in the form of fibrohyaline nodules, very similar to those found in silicosis. Higher incidences of abnormal columnar cells, squamous metaplasia without atypia, Cu containing macrophages, eosinophilia, and respiratory spirals were also reported in the sputa of smoking and non-smoking vineyard sprayers, as compared to rural workers from the same geographic region who did not work in the vineyards

7.3.4.3 Chronic Effects

Cu has been identified as a respiratory irritant characterised by coughing, sneezing, thoracic pain and runny nose. Askergren and Mellgren (1975) studied 11 sheet metal workers exposed to Cu salt dust and observed increased vascularity and superficial epistatic vessels in the nasal mucosa. These workers also reported eye irritation.

Suciu et al (1981) reported respiratory effects along with endocrine, gastrointestinal, hepatic, neurological and reproductive effects associated with inhaled Cu in 75 – 100 workers involved in Cu grinding and sieving exposed to 111 mg Cu/m³ – 434 mg Cu/m³ over a 3 year period. The workers lung radiographs showed linear pulmonary fibrosis and nodulation. Observed hepatic effects included Hepatomegaly while endocrine effects included enlargement of the sella turcica, non-secretive hypophyseal adenoma (Cushing's syndrome), accompanied by obesity, arterial hypertension, and "red facies". The significance of these effects however could not be determined. Workers reported anorexia, nausea and diarrhoea however these effects are thought to be more likely due to oral exposure as a result of muco-ciliary clearance of Cu particles deposited in the respiratory tract. They also reported headaches, vertigo, drowsiness and sexual impotence however once again the significance of these findings could not be established.

Haematological effects were reported by Finelli et al (1981) who investigated anaemic effects in male workers exposed to 0.64 – 1.05 mg/m³ Cu. They found decreased haemoglobin and erythrocyte levels however this could not fully be attributed to Cu as hair analysis revealed the workers were exposed to other metals.

Certain predisposed conditions have been identified for Cu exposure sensitivity. Persons with Wilson's disease, glucose-6-phosphate dehydrogenase deficiency, anaemic, allergic, and liver or kidney

conditions may be susceptible to the effects of Cu exposure. Persons exposed to molybdenum might be less sensitive to Cu, since molybdenum is antagonistic to Cu toxicity. Infants and children less than 1-year of age may be more sensitive to the effects of Cu exposure because homeostatic mechanisms for clearing Cu from the body are not yet developed (OEHHA 2008).

Although Cu homeostasis plays an important role in the prevention of Cu toxicity, exposure to excessive levels of Cu can result in a number of adverse health effects including liver and kidney damage, anaemia, immune toxicity, developmental toxicity, cancer progression, cardiovascular disease, atherosclerosis, diabetes and neurological disorders. It is well known that Cu promotes oxidative damage in the conditions of increased Cu levels in the liver and brain and as such many of the observed effects of Cu toxicity have been consistent with oxidative damage to membranes or macromolecules (Boveris et al 2012, Tepe 2014). Absorption and excretion feedback mechanisms normally prevent chronic Cu toxicity in humans. However, an accumulation of Cu in body tissues resulting in dyshomeostasis can occur in rare cases of Wilson's disease (Tepe 2014). Wilson's disease (ATP7B mutation) is an autosomal recessive disorder linked to the Cu translocase expressed in hepatocytes. This enzyme is critical in the distribution and elimination of excess Cu from the organism (Boveris et al 2012).

Liu et al (2009) evaluated the toxicity of nasal instilled nanoscale copper particles (23.5 nm) in comparison to the macro sized Cu particles (17 µm) in mice. When 40 mg/kg body weight was instilled three times in one week, body weight of mice was retarded and significant pathological changes were observed. There were hydropic degeneration around the central vein and the spotty necrosis of hepatocytes in the liver and swelling in the renal glomerulus, while, severe lesion associated with the decreased number of olfactory cells and the dilapidated laminated structure were also observed in the olfactory bulb. The serum biochemical assay also indicated the sign of renal and hepatic lesion. Retention and distribution of copper in various tissues show that the liver, kidneys and olfactory bulb are the main accumulated tissues for copper particles. This study indicated that nasal inhaled copper particles at very high dosage can translocate to other organs and tissues and further induce certain lesions.

The majority of the literature on the neurotoxicity of Cu centres around nutritional deficiency and its effect on brain. There is evidence of neurotoxicity when Cu is found in excess in the brain. Excess brain Cu is a common finding in neurodegenerative diseases such as Alzheimer's disease and Wilson's disease, with the presenting complaint for this genetic disorder frequently including neurobehavioral changes resembling schizophrenia. These neurologic findings may even precede other findings such as liver disease (Wright and Baccarelli 2007). In an animal study, Zhang et al 2012 aimed to quantify the neurological effects of Cu inhalation in mice by intranasal instillation based on neurotransmitter secretion. Cu nanoparticle-exposed mice exhibit pathological lesions at different degrees in certain tissues and especially in lung tissue. The liver, lung and olfactory bulb were identified as the main tissues in which the Cu concentrations increased substantially after exposure to a higher level of Cu nanoparticles (40 mg/kg of body weight). The secretion levels of various neurotransmitters changed as well in some brain regions, especially in the olfactory bulb. These results indicate that the intranasally instilled Cu nanoparticles (23.5 nm) not only cause the lesions where the Cu accumulates, but also affect the neurotransmitter levels in the brain.

Barchowsky (2010) investigated the impacts of Cu on human cardiovascular diseases in an attempt to elucidate the biological mechanisms for affecting the heart and vascular tissues. Wilson's disease has been additionally characterised by not only hepatic and neuronal copper overload with oxidant stress, but left ventricular remodelling and a relatively high frequency of benign supraventricular tachycardia's and extra systolic beats in the cardiovascular system. The mechanism of action can either be direct or indirect. Cu (depending on the oxidation state) readily reacts with sulfhydryl, carbonyl, or phosphate groups and when bound is capable of enzyme inactivation through redox cycling and ROS generation. Cu has several unique mechanisms for promoting disease through

oxidant stress. As indicated, Cu directly generates reactive oxygen species by redox cycling and increased Cu levels in plasma correlate with increased levels of oxidized lipoproteins; although the mechanism for this increase is unclear and Barchowsky noted that it was not simply defined by direct redox catalyzed reactions. Similarly, copper increases the levels of the known endogenous cardiovascular toxicant, homocysteine, which may contribute to its association with oxidative vascular dysfunction and increased peripheral vascular disease.

Arteriosclerosis, and especially atherosclerosis, or occlusive disease are the most common pathologic process underlying cardiovascular diseases and can be systemic or confined to individual organs. Accumulation of Cu may play a role in atherogenesis and the etiology of atherosclerosis related to aging. There is strong evidence that levels of Cu that is normal and adequate in reproductive years become clear risks for age-related atherosclerosis (ATSDR, 2004). Elevated labile Cu is a well-established risk for atherogenesis, as well as coronary artery disease. Molecular studies are generally supportive of a relationship of Cu to the atherogenic process and mechanistic studies find elevated levels of Cu in human atherosclerotic plaques. These studies also find that copper redox reactions oxidize LDL and that apolipoprotein E may owe its antioxidant effects to inhibiting Cu oxidation of LDL. A substantive portion of copper-related ischemic disease results from effects on and interactions with homocysteine (ATSDR, 2004; OEHA, 2008).

Goering and Barber 2010 investigated the hepatotoxicity of Cu and the underlying hepatotoxic mechanisms. Cu acts as a direct cellular toxicant resulting in cell injury and hepatocellular necrosis through redox activity and production of reactive oxygen species and associated injury. Cu overload results in severe hepatic injury, which can be fatal if not treated. Wilson's disease and genetic hemochromatosis (GH) involve heritable defects in the excretion of Cu and absorption of iron, respectively, which lead to progressive toxic accumulation of Cu within hepatocytes. Cu undergoes redox reactions, increasing its propensity to initiate and participate in the generation of tissue-damaging oxidative free radicals that can eventually precipitate hepatocellular injury. Acute over dosage can cause hepatotoxicity but acquired chronic Cu hepatotoxicity has not been definitively established.

Regarding chronic hepatotoxicity, several disease states attributable to toxic, chronic accumulation of hepatic Cu include Wilson's disease, Indian childhood cirrhosis, idiopathic Cu toxicosis (non-Indian childhood cirrhosis), and vineyard sprayer's lung. Wilson's disease, idiopathic Cu toxicosis and vineyard sprayer's lung are the most relevant disease states after excessive inhalation exposure to Cu. In Wilson's disease and idiopathic Cu toxicosis, inheritance of mutant genes is necessary and sufficient to cause copper hepatotoxicity and lethality independent of the dietary intake of Cu. Vineyard sprayer's lung is perhaps the only disease thus far attributable to acquired chronic Cu hepatotoxicity.

7.4 Exposure Assessment

Metals data was available from the PHIC monitoring stations from October 2011 to November 2013. Monitoring was undertaken for the following periods:

- 2011 – daily for the period of October – December
- 2012 – daily for the period of January to mid-February and mainly on a weekly frequency for the remainder of 2012
- 2013 – sampling every 3 days for the year up to the end of November

Monitoring was undertaken for metals in the PM₁₀ fraction.

Additional monitoring undertaken by the WA ChemCentre. Data was available for the following a frequency of every 3 days from 31 October, 2012 through to 12 May, 2014. Metals were monitored in both the PM₁₀ and PM_{2.5} fractions.

Datasets from all years of monitoring were used within the exposure assessment. With respect to data for chromium III and VI, it has been identified by DoH and PHIC that the data was invalid due to the use of incorrect filters for sampling, as such, chromium III and VI has not been evaluated further within the HRA.

To analyse the available data, the data was examined temporally, examining each monitor for each metal, for each dataset (e.g., 2011, 2012, 2013 & ChemCentre data). In reviewing the data, there did not appear to be any missing data or zero data. In assessing concentrations for metal parameters that were below the detection limit they were treated as zero for the PHIC monitoring. In the case of the ChemCentre monitoring they chose to treat those below detection limit as being present at the detection limit concentration. As the less than signs have been removed from the data it is not possible to distinguish those results that are less than detection and those found at the limit of detection. As a result, this dataset will be treated differently in terms of non-detect concentrations. The effects of this assumption have been examined in the analysis below.

The summary statistics for each of the metals for each site are summarised in Tables 16 - 27.

Table 16: Copper data Richardson St

Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	Chem Centre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	7.23E-03	1.40E-02	1.51E-02	1.27E-02	6.46E-03
90 th Percentile	1.13E-02	2.00E-02	3.04E-02	2.81E-02	1.20E-02
95 th Percentile	1.50E-02	3.22E-02	4.68E-02	4.90E-02	2.13E-02
Maximum	3.60E-02	2.60E-01	2.90E-01	2.90E-01	1.50E-01

Table 17: Iron oxide Richardson St

Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	ChemCentre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	Not sampled	3.26E+00	3.71E+00	3.39E+00	1.59E+00
90 th Percentile		5.47E+00	6.72E+00	5.72E+00	3.43E+00
95 th Percentile		6.01E+00	8.29E+00	7.16E+00	4.50E+00
Maximum		1.40E+01	9.90E+00	9.90E+00	5.80E+00

Table 18: Manganese Richardson St

Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	ChemCentre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	9.40E-02	1.08E-01	9.13E-02	8.44E-02	4.47E-02
90 th Percentile	1.26E-01	1.38E-01	1.53E-01	1.50E-01	7.86E-02
95 th Percentile	2.00E-01	3.40E-01	2.36E-01	2.06E-01	1.20E-01
Maximum	2.50E+00	2.30E+00	1.60E+00	1.60E+00	1.30E+00

Table 19: Copper Taplin St

Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	ChemCentre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	6.90E-03	6.53E-03	5.09E-03	4.06E-03	2.61E-03
90 th Percentile	9.00E-03	9.00E-03	8.00E-03	7.00E-03	4.00E-03
95 th Percentile	9.00E-03	1.19E-02	9.00E-03	9.00E-03	6.00E-03
Maximum	3.20E-02	7.30E-02	1.60E-02	1.60E-02	8.00E-03

Table 20: Iron oxide Taplin St

Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	ChemCentre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	Not sampled	1.53E+00	1.74E+00	1.68E+00	7.06E-01
90 th Percentile		2.64E+00	3.19E+00	3.10E+00	1.60E+00
95 th Percentile		3.24E+00	4.90E+00	4.44E+00	2.10E+00
Maximum		8.50E+00	7.90E+00	7.90E+00	4.70E+00

Table 21: Manganese Taplin St

Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	ChemCentre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	3.54E-02	3.64E-02	3.37E-02	3.17E-02	1.36E-02
90 th Percentile	5.62E-02	6.65E-02	6.16E-02	5.82E-02	3.02E-02
95 th Percentile	7.60E-02	1.10E-01	8.27E-02	7.30E-02	3.82E-02
Maximum	3.40E-01	3.90E-01	2.10E-01	3.20E-01	1.30E-01

Table 22: Copper South Hedland

Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	ChemCentre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	5.40E-03	3.98E-03	3.83E-03	2.51E-03	2.42E-03
90 th Percentile	7.00E-03	5.10E-03	5.00E-03	3.00E-03	2.00E-03
95 th Percentile	7.00E-03	6.00E-03	5.95E-03	4.00E-03	3.00E-03
Maximum	1.60E-02	1.00E-02	3.60E-02	3.60E-02	3.70E-02

Table 23: Iron oxide South Hedland

Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	ChemCentre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	Not sampled	8.76E-01	7.72E-01	7.60E-01	4.28E-01
90 th Percentile		1.30E+00	1.25E+00	1.30E+00	8.00E-01
95 th Percentile		1.79E+00	1.48E+00	1.55E+00	1.00E+00
Maximum		7.50E+00	2.70E+00	2.70E+00	1.90E+00

Table 24: Manganese South Hedland

Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	ChemCentre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	2.03E-02	1.70E-02	1.57E-02	1.58E-02	8.80E-03
90 th Percentile	3.30E-02	2.79E-02	2.83E-02	2.90E-02	1.70E-02
95 th Percentile	3.95E-02	3.20E-02	3.07E-02	3.25E-02	1.96E-02
Maximum	9.20E-02	1.90E-01	6.80E-02	6.80E-02	3.80E-02

Table 25: Copper Yule River

Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	ChemCentre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	Not sampled	1.69E-01	3.53E-02	2.16E-02	1.58E-02
90 th Percentile		2.78E-01	6.13E-02	2.10E-02	1.18E-02
95 th Percentile		2.84E-01	6.27E-02	1.05E-01	1.27E-01
Maximum		2.90E-01	6.40E-02	2.90E-01	1.60E-01

Table 26: Iron oxide Yule River

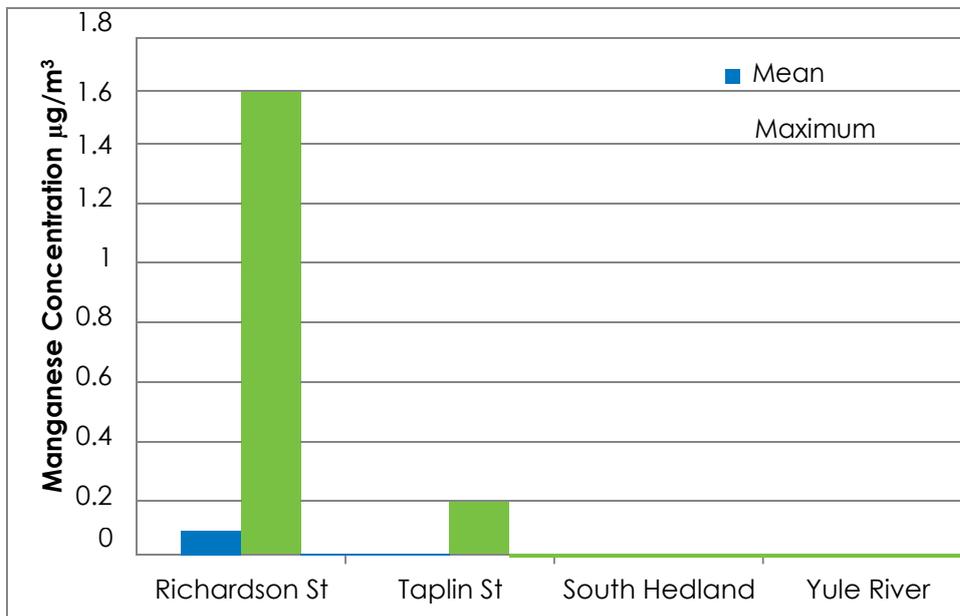
Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	ChemCentre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	Not sampled	1.49E+00	3.48E-01	5.28E-01	2.39E-01
90 th Percentile		2.46E+00	7.49E-01	8.24E-01	5.22E-01
95 th Percentile		4.98E+00	7.70E-01	1.05E+00	5.66E-01
Maximum		7.50E+00	8.80E-01	7.50E+00	1.10E+00

Table 27: Manganese Yule River

Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	ChemCentre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	Not sampled	3.36E-02	1.07E-02	1.28E-02	6.53E-03
90 th Percentile		5.92E-02	1.66E-02	1.72E-02	7.40E-03
95 th Percentile		1.20E-01	1.80E-02	2.04E-02	1.62E-02
Maximum		1.80E-01	1.80E-02	1.80E-01	3.50E-02

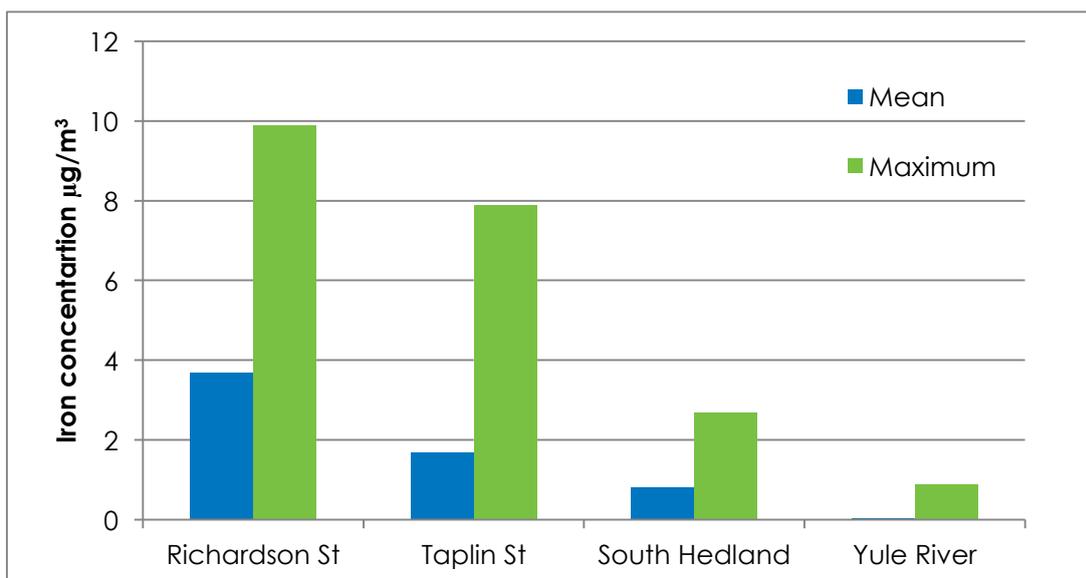
Comparison of mean and maximum values for each of these metals for all sites for 2013 (the most complete data set) is shown in **Figure 14**.

Figure 12: Maximum and Mean Manganese Concentrations 2013 PHIC Data ($\mu\text{g}/\text{m}^3$)



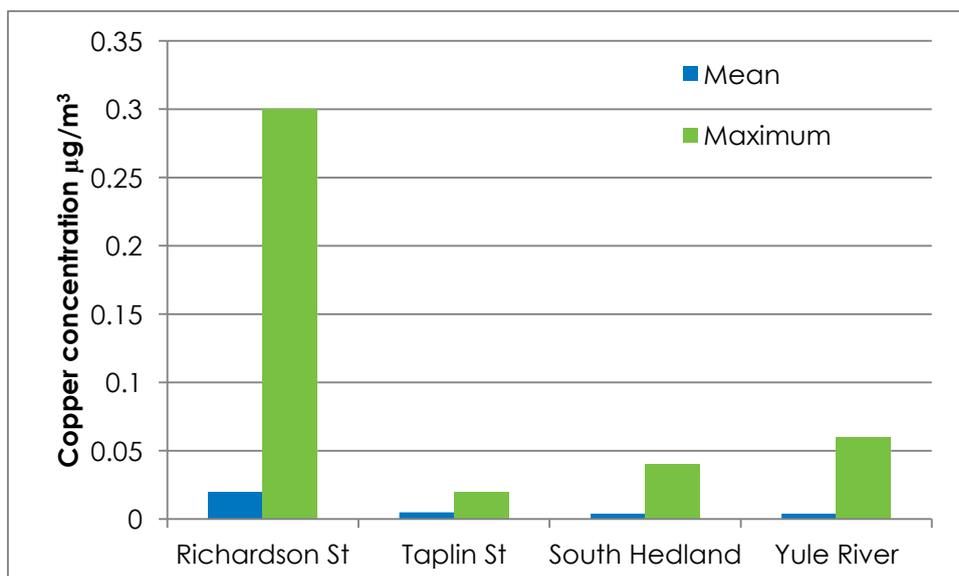
The data shown in Figure 13 shows a clear impact of Port operations on manganese levels at both Richardson St with much lower levels at Taplin St, South Hedland and Yule River. Manganese is stored at both the Port and Finucane Island.

Figure 13 : Maximum and Mean Iron Concentrations 2013 PHIC Data ($\mu\text{g}/\text{m}^3$)



The trend in both maximum and mean iron concentrations shows a clear impact of the Port operations on the West and East End of Port Hedland (represented by data at Richardson St and Taplin St respectively) which decreases at South Hedland and Yule River.

Figure 14: Maximum and Mean Copper Concentrations 2013 PHIC Data ($\mu\text{g}/\text{m}^3$)



The trend in the copper data also shows an impact of Port operations at Richardson St which is not seen at the other sites.

Comparison of data for all sites across each of the years monitored shows very little year-to-year variation. Comparison with the PM_{10} data from the WA Chem Centre monitoring shows very similar results to the PHIC monitoring data.

7.5 Risk Characterisation

The purpose of the risk characterisation is to estimate potential risks associated with inhalation exposure to the metals. For the assessment of non-carcinogenic health effects, the monitored concentration for each COC is compared to the Toxicity Reference Value (TRV) as set out in Table 1. The ratio of the monitored concentration to the TRV is termed the hazard quotient (HQ):

$$HQ = \text{Ambient concentration} / TRV$$

The hazard quotients are estimated for each of the averaging periods relevant to the TRVs for acute and chronic health effects. None of the pollutants that have been assessed are carcinogens therefore a carcinogenic risk assessment is not applicable for this HRA. (Note: Cr VI is a known human carcinogen however no monitoring data was available to enable an assessment the potential risk).

Table 28 summarises the hazard quotients for Manganese, Copper and Iron for Port Hedland based on the PHIC monitoring data. The acute hazard quotients have been based on the maximum 24-hr monitored concentration. The chronic are based on the annual average concentrations.

Table 28: Hazard Quotients for Key Metals

RICHARDSON ST	HAZARD QUOTIENT		
	MANGANESE	COPPER	IRON

Acute		0.003	0.08
Chronic	0.6		
TAPLIN ST			
Acute		0.001	0.07
Chronic	0.2		
SOUTH HEDLAND			
Acute		0.0003	0.03
Chronic	0.1		
YULE RIVER			
Acute		0.0005	0.009
Chronic	0.07		

As can be seen from Table 28 all the hazard quotients are well below 1 which is considered the acceptable level of risk for non-carcinogenic substances. The highest risk is for manganese at Richardson St but this is still below 1.

7.6 Summary

The HRA has shown that for the metals assessed that the health risks associated with current exposure levels in Port Hedland are below the adopted TRVs and are not considered to pose an unacceptable health risk. The monitoring data as shown in Figures 12-14 show a clear impact of Port activities as reflected in higher concentrations recorded for iron, manganese at Richardson and Taplin St compared to the other sites. For iron and manganese there is a clear decrease in ambient concentrations the further the distance from the Port. For manganese the hazard quotient of 0.6 at Richardson St is approaching the acceptable risk level of 1.

8 HEALTH RISK ASSESSMENT RESPIRABLE CRYSTALLINE SILICA

8.1 Hazard Assessment

Silica may either be crystalline or non-crystalline; it is occupational exposure to respirable crystalline silica (RCS) that is most commonly associated with adverse health effects (IARC 1997, CICAD 2000, de Klerk et al 2002). In the occupational setting chronic exposure to crystalline silica is associated with increased incidences of tuberculosis, bronchitis, emphysema, chronic obstructive pulmonary disease, renal diseases, silicosis^a and lung cancer. Of these potential health effects silicosis and lung cancer are the effects of most concern (US EPA 1996, CICAD 2000, de Klerk et al 2002). The California Environmental Protection Agency (OEHHA 2005), the World Health Organization (CICAD 2000) and the US Environmental Protection Agency (US EPA 1996) all judge silicosis as being the most sensitive health end point for which health risks from exposure to silica should be assessed. These agencies consider prevention of silicosis will provide protection against other possible health effects that may be associated with exposure to high levels of airborne crystalline silica in the workplace or ambient air.

Crystalline silica has its fibrogenic effects deep in the lung and it is only particles which are capable of penetrating to the gas exchange region, i.e. the alveoli, that are of concern in determining the hazard to health from crystalline silica (NOHSC 1995, p24 Footnote). With regard to the relationship between silicosis and crystalline silica-induced lung cancer, both the mechanism of toxicity and epidemiological data indicate there are exposures of crystalline silica below which the risk of developing these conditions is very low. Crystalline silica toxicity has been extensively investigated and has led to a widely accepted toxicological mechanism involving chronic inflammation and oxidative stress. Chronic inflammation in the lower respiratory tract is an intrinsic component of the pathophysiological mechanisms that cause many dust-related lung diseases. RCS particles deposited deep in the lung on the alveolar surface are ingested by macrophages which release high levels of a variety of cytokines and initiate local oxidative stress and inflammation. Persistent inflammation, such as occurs when large amounts of RCS are retained in the lungs, leads to the proliferation of fibroblasts, increased collagen production and eventually fibrosis (silicosis). The production of reactive oxygen species by macrophages is thought to lead to mutations in the DNA of dividing pulmonary cells and hence, by indirect genotoxic mechanisms, crystalline silica can also cause lung cancer (US EPA 1996, De Klerk et al 2002, HSE 2003). The induction of lung toxicity, the proliferative cellular changes and DNA damage are related to the relative severity of inflammation. These toxicological mechanisms are consistent with a threshold exposure for both silicosis and lung cancer (HSE 2003). That is an air concentration below which the initiating events for silicosis and lung cancer will not occur.

In 1997 the International Agency for Research on Cancer (IARC 1997) concluded crystalline silica in the form of quartz or cristobalite from occupational sources is carcinogenic to humans. Although silicosis and lung cancer are both likely to stem from a common background of chronic inflammatory lung damage they are distinct disease conditions involving different cell types. This has fuelled debate whether lung cancer can develop independently of silicosis or is a consequence of the latter. Overall,

^a Silicosis is one of the more destructive forms of pneumoconiosis (characterised by scarring of the lungs), which is contracted by prolonged exposure to high levels of fine crystalline silica dust. Pneumoconiosis is a condition characterised by permanent deposition of substantial amounts of particulate matter in the lungs and by the tissue reaction to its presence; depending on the chemical nature of the particulate it may range from relatively harmless forms of tissue hardening to the destructive fibrosis of silicosis. Silicosis is an irreversible and progressive condition in which healthy lung tissue becomes replaced with areas of fibrosis.

where evidence is available concerning the relationship between lung cancer and silicosis it tends to show excess lung cancer mortality in RCS exposed workers is restricted to those with silicosis, the more severe the silicosis, the higher the risk of lung cancer. The implication is that exposures to RCS insufficient to cause silicosis would be unlikely to lead to a significant increase in the risk of lung cancer over and above background levels. However because the power of epidemiological studies to detect small excesses of lung cancer at lower exposure levels of RCS is limited the evidence is not definitive (CICAD 2000, HSE 2003).

A recent study from China examined the long-term exposure to silica dust and the risk of total and cause-specific mortality (Chen et al., 2012). This study followed 74,040 workers who worked at 29 metal mines and pottery factories in China for 1 year or more between January 1960 and December 1974 with follow-up until December 2003 (median follow-up 33 years). This study found that long-term exposure to silica dust was associated with increased mortality in Chinese workers due to respiratory disease and lung cancer as well as cardiovascular disease.

From occupational studies it is known that the severity and incidence of silicosis and silica-induced cancer increases with intensity of dust exposure, with increased cumulative duration of exposure, with increasing peak concentrations of silica and with increased percentage of silica within the respirable dust (de Klerk et al 2003, US EPA 1996). Because both diseases have potential long latencies their incidence also increases with the length of follow-up of exposed workers from date of silicosis diagnosis (CICAD 2000). As exposure concentration or duration of exposure increases early symptoms of silicosis are the first chronic health effects observable from inhalation of crystalline silica (US EPA 1996). Silicosis is also regarded as the critical effect for hazard identification and exposure response assessment for crystalline silica (CICAD 2000, HSE 2003).

In the occupational setting silicosis can occur from relatively short term exposure to very high peak concentrations of airborne crystalline silica^a. However in the absence of such high exposures this disease results from longer term exposure to relatively low concentrations and the consequent accumulation of crystalline silica in the lungs. Exposure is usually estimated as being cumulative over a number of years (e.g. $\mu\text{g}/\text{m}^3 \times \text{years}$) (de Klerk et al 2003, US EPA 1996, CICAD 2000, HSE 2003). Therefore for estimating health risks to residents from exposures to low airborne concentrations of crystalline silica it is appropriate to use modelling predictions for annual ground level concentrations of respirable particulates rather than 24 hour modelled estimates.

Exposure to RCS has also been linked to autoimmune diseases such as rheumatoid arthritis, systemic sclerosis, antineutrophil cytoplasmic antibodies (ANCA)-related vasculitis and more recently Lupus (WHO, 2006). The results of epidemiological studies examining these outcomes have been supported in animal studies. As discussed above, RCS particles are phagocytosed by alveolar macrophages, leading to cellular activation and the release of soluble mediators such as chemokines, proinflammatory cytokines, lysosomal enzymes and reactive oxygen and nitrogen species. These soluble mediators act to recruit and activate additional inflammatory cells that may lead to increased antigen processing and accelerated antibody production. The effect is not limited to the lung (WHO, 2006). Migration of silica-containing macrophages to the lymph nodes and increased systemic immunoglobulin production has also been shown to occur (Huang et al, 2001; Weissman et al., 2001). In

^a A worker may develop one of three types of silicosis depending on the airborne concentration of respirable crystalline silica: Chronic silicosis – which usually occurs after 10 or more years of exposure to relatively low, but still higher concentrations than that which the general public may be exposed. Accelerated silicosis – which develops 5 to 10 years after the first exposure. Acute silicosis – this develops after exposure to high concentrations of respirable crystalline silica and results in symptoms within a few weeks to 4 or 5 years after the initial exposure (CICAD 2000).

a genetically susceptible murine model of lupus, silica exposure exacerbated development of autoimmune disease (Brown et al., 2003). In a rat model for multiple sclerosis, administration of silica up to one month prior to or concurrent with spinal cord homogenates increased the incidence and severity of and advanced the onset of the disease (WHO, 2006).

8.2 Exposure Assessment

Respirable crystalline silica is present in the ore that is handled in Port Hedland. As such it is a potential contaminant of concern for the HRA. Crystalline silica was monitored in Port Hedland at Richardson St, Taplin St and Neptune from 22 March 2014 to 26 September 2014. Monitoring data was collected every 3 days over that period and analysed by MPL Laboratories. A summary of the data collected is shown in Table 29. The data was collected as the PM₁₀ fraction which corresponds to the inhalable rather than respirable fraction.

Table 29: Summary of Monitoring Data for Respirable Crystalline Silica March – September 2014

Location	Duration of Monitoring	Number of Samples	Average (µg/m ³)	Maximum (µg/m ³)
Richardson St	22/3/14 – 26/9/14	47	0.2	0.6
Taplin St	22/3/14 – 26/9/14	45	0.4	1.3
Neptune	22/3/14 – 26/9/14	49	0.25	0.7

8.3 Risk Characterisation

For assessing the potential health impact of crystalline silica, predicted concentrations are compared to health-based ambient air standards that are established to protect public health. This comparison is performed by calculating a hazard quotient (HQ) which is the ratio of predicted ambient concentration to the to the air quality standard which has been derived to protect public health.

The hazard quotient is calculated using the equation below.

$$HQ = \text{ambient concentration} / TRV$$

Generally if a HQ is less than 1 it is generally accepted that there is no cause for concern. The HQ approach is essentially quite conservative in providing an estimate of risk, since there is a significant margin of safety built into the development of air quality standards for pollutants that are known to have a threshold for effect. When the overall HQ is less than 1, it is generally assumed that risk is within reasonable bounds and that there is no need to undertake a more detailed risk assessment (enHealth, 2012).

The Office for Environmental Health Hazard Assessment (OEHHA), which is part of the Californian EPA, has established an ambient air quality guideline (chronic reference exposure level – REL) for the protection of public health for respirable crystalline silica (OEHHA, 2005). This is based on the protection from developing silicosis. This guideline was adopted has been adopted for use as the applicable TRV for the Port Hedland HRA. This TRV has been derived to apply to the PM_{2.5} fraction not the PM₁₀ fraction as has been monitored. This creates a level of uncertainty in the risk calculations. However, in the absence of other air quality guidelines that apply to the PM₁₀ fraction the OEHHA value has been applied.

A further uncertainty is that the data was only collected over a 6 month period rather than the 12 months to accurately determine an annual average value. Over the time period 45-49 samples were collected which is less than that required to determine an annual average. It is noted however that the samples were collected over the dry period in Port Hedland when dust levels would be expected to be higher. In the absence of other data the risk calculations have been done using this data.

Using the data set out in Section 9.2 and the OEHHA chronic REL of $3\mu\text{g}/\text{m}^3$ the resultant HQ for monitored levels on RCS at the 3 sites in Port Hedland are summarised in **Table 30**.

Table 30: Hazard Quotients for Respirable Crystalline Silica in Port Hedland

Location	Average ($\mu\text{g}/\text{m}^3$)	Air Quality Guideline (annual average) ($\mu\text{g}/\text{m}^3$)	Hazard Quotient
Richardson St	0.2	3	0.07
Taplin St	0.4	3	0.13
Neptune	0.25	3	0.08

The resultant HQ indicates that the risk posed by the in Port Hedland at all sites monitored is very low and is not of concern. Even with the uncertainties in the use of only 6 months of data in the PM_{10} fraction with the calculated hazard quotients being well below 1 it is unlikely that the overall risks would change.

9 HEALTH RISK ASSESSMENT MINERAL ASBESTIFORM FIBRES

9.1 Hazard Assessment

Asbestos poses a human health risk through the inhalation of its fibres. If deposited in the lungs, the fibres can initiate diseases that take many years to result in observed health effects. These effects include asbestosis, lung cancer and the normally rare cancer mesothelioma. These health effects tend to be the result of higher levels of exposure, most often occupational, but mesothelioma can also result from low level exposures. The main health effect of associated with exposure to asbestos is its carcinogenic potential (OEHHA, 2013; IARC, 2011; ATSDR, 2001; WHO, 2000 and USEPA, 2001, 1993).

Exposure to asbestos is not known to produce acute short-term health effects. The most significant effects arise from long-term exposures to asbestos. Asbestos is a generic term for a group of six naturally occurring fibrous silicate minerals. Asbestos minerals exist in two forms – serpentine asbestos and amphibole asbestos. Chrysotile, a serpentine asbestos, has long flexible crystalline fibres that are capable of being woven. Amphibole asbestos includes amosite, crocidolite, and fibrous forms of tremolite, anthophyllite and actinolite. Amphibole asbestos is considered to pose a greater health risk than chrysotile asbestos (IARC, 2011; ATSDR, 2001). In a review by Baur et al., (2012) it was concluded that epidemiological studies showed chrysotile causes less pleural fibrosis and mesotheliomas when compared with other asbestos types. However, based on clinical, animal as well as on in-vitro findings, the inflammatory, toxic, carcinogenic, and fibrosis-inducing effects chrysotile are consistent with amphibole asbestos.

Epidemiological studies of asbestos-exposed workers and supporting animal studies indicate that inhalation of asbestos is the principal route of exposure for public health exposures. Depending largely on the size and shape, deposition on asbestos occurs in the lung tissue (ATSDR, 2001). Some fibres may be removed by mucociliary clearance or macrophages while others may remain in the lung for long periods of time. Inhalation exposure is considered to be cumulative. Studies in humans and animals indicate that inhalation exposure to asbestos fibres may lead to the development of pulmonary disease including asbestosis and/or lung cancer and mesothelioma of the pleura or peritoneum (ATSDR 2001; IARC, 2011; WHO, 2000).

It has been conclusively shown in numerous studies of occupationally exposed workers that inhalation of asbestos can lead to an increased risk of cancer and mesothelioma (ATSDR, 2001; IARC, 2011; WHO, 2000). Asbestos is classified by the IARC and USEPA as a known human carcinogen. The USEPA has calculated that, using a linear no-threshold model, that lifetime exposure to asbestos dust containing 0.0001 fibres greater than 5µm in length per mL of air could result in 2-4 excess cancer deaths (lung cancer plus mesothelioma) per 100,000 (USEPA, 2001).

While lung cancer and mesothelioma are generally associated with chronic exposure to asbestos, there are several studies that show that short-term exposures are also of concern (ATSDR, 2001). The ATSDR cite studies that show that workers exposed to asbestos for only 1-12 months had an increased risk of developing cancer a number of years later. Rats exposed to high concentrations of amosite or crocidolite for only 1 day developed mesothelioma. Although there is uncertainty about the dose response relationship for health risks from short-term exposures to asbestos, the data indicate that these exposures should not be disregarded (ATSDR, 2001).

Asbestos exposure is also linked to an increase in gastrointestinal cancer (IARC, 2011; ATSDR, 2001; OEHHA, 2013) although the evidence is less consistent than for lung cancer and mesothelioma. Studies of workers exposed to asbestos via inhalation have also been shown to have small increased death rates from gastrointestinal cancers. This is presumed to be due to the transfer of inhaled fibres from the lung to the gastrointestinal tract. Other studies of populations with high levels of asbestos fibres in

drinking water may have an increased risk of gastrointestinal cancers. These findings are supported by the findings of animal studies where rats were fed intermediate length chrysotile during their lifetime.

As well as causing cancer asbestos can lead to non-cancer respiratory effects known as asbestosis (ATSDR, 2001). Asbestosis results from a prolonged inflammatory response stimulated by the presence of fibres in the lung. This has been observed in workers with relatively low cumulative exposures to asbestos. Exposure to asbestos can also result in changes to the lining of the chest cavity and the outside of the lungs (pleura) which may affect people's breathing.

There is also some evidence that the effects of asbestos and the tendency to develop asbestosis or mesothelioma may be related to the immune system. Some studies have found that people with depressed immune system develop these diseases where people with non-compromised immune systems do not (ATSDR, 2001). However the available evidence does not enable firm conclusions to be drawn at this stage.

9.2 Exposure Assessment

Mineral asbestiform fibres are present in the ore that is processed at Port Hedland. Monitoring was undertaken at Richardson St, Taplin St and Neptune Place between 3 April 2014 and 16 September 2014. Samples were taken every 3 days during that period.

All of the samples collected were at or below the level of detection of 0.01 fibres/mL. Although some filter papers did contain small numbers of fibres many were not asbestos mineral fibres. Some samples from all sites did contain small numbers of fibres on the filters that were identified as asbestos in the form of actinolite. However these were not present at levels above the level of detection of the method -NIOSH filter membrane method and SEM (scanning electron microscope).

9.3 Risk Characterisation

As all samples were below the level of detection no assessment of the risk to the Port Hedland population was required. The level of detection for the method is well below the WA Department of Health guideline for asbestos of 0.01 fibres/mL and below the EPA Victoria guideline for asbestos fibres in air of 0.05 fibres/m³.

10 HEALTH RISK ASSESSMENT NITROGEN DIOXIDE

10.1 Hazard Assessment

In recent years the health effects of NO₂ linked to ambient exposures have been well studied and reviewed by international agencies (WHO, 2013; NEPC, 2010; USEPA, 2008a; WHO, 2006; California EPA 2001). The critical health outcomes identified in overseas and Australian epidemiology studies resulting from short term exposure to NO₂ are increased respiratory disease and symptoms, especially in asthmatic children, and changes in lung function. The evidence for the effects of long-term exposure to NO₂ is limited, but epidemiological studies of chronic exposures to NO₂ from indoor sources suggested an increased risk of lower respiratory illness in children. There is also evidence to suggest an association between chronic NO₂ exposure and changes to growth in lungfunction.

10.1.1 Short-term exposure

10.1.1.1 Mortality

Results from several large U.S. and European multi-city studies and a meta-analysis study indicate positive associations between ambient NO₂ concentrations and the risk of all-cause (non-accidental) mortality (e.g. APHEA1 and 2; US National Morbidity, Mortality, and Air Pollution Study—NMMAPS). Effect estimates in these studies range from 0.5 to 3.6% excess risk in mortality per standardized increment (20ppb for 24-hour averaging time, 30ppb for 1-hour averaging time). In general, the NO₂ effect estimates were robust to adjustment for co-pollutants.

Australian multicity studies have found either similar or greater associations between ambient levels of NO₂ and increases in mortality than those reported in European studies (i.e. APHEA1 and 2). Australian studies report increases in mortality from between 0.11% and 0.9% for every 1ppb increase in NO₂ (Simpson et al. 2005a,b; Simpson et al. 1997; Hinwood et al. 2004; Denison et al. 2000). In the US NMMAP study, NO₂ showed statistically significant relative increases in daily mortality from 0.3% to about 0.4% per 10ppb (previous day concentration, lag 1). This effect remained but lost statistical significance after adjusting for PM₁₀ and ozone.

Both cardiovascular and respiratory mortality have been associated with increased NO₂ concentrations in epidemiological studies, however, similar associations are observed for other pollutants, including particles and SO₂. The range of risk estimates for excess mortality is generally smaller for NO₂ than for other pollutants (USEPA, 2008a). In addition, while NO₂ exposure, alone or in conjunction with other pollutants, may contribute to increased mortality, evaluation of the specificity of this effect is difficult. Clinical studies that show haematologic effects and animal toxicological studies that show biochemical, lung host defense, permeability, and inflammation changes provide limited evidence of plausible pathways by which risks of mortality may be increased with short-term exposures to NO₂, but the USEPA concluded that no coherent picture is evident at this time (USEPA, 2008a).

In the REVIHAAP report the WHO (2013) found that, positive and statistically significant short-term associations of NO₂ with all-cause and cause-specific mortality have been reported in the new studies published since the 2005 global update of the WHO air quality guidelines. Robustness of the short-term NO₂ associations to adjustment for particles and other pollutants has been demonstrated in multicity studies from various geographic locations, including Europe. The United States NMMAPS is, however, a notable exception. Overall, WHO concluded that the findings suggest that the short-term associations of NO₂ with mortality are not confounded by the particle metrics used in the studies – that is, mainly PM₁₀, and sometimes PM_{2.5} and black smoke.

(Anderson et al., 2007) reported that increases in NO₂ concentrations (per 10 µg/m³, 24-hour averages) are associated with increases in all-cause mortality: 0.49% (95% CI: 0.38–0.60%) in all ages and 0.86% (95% CI: 0.50–1.22%) for those older than 65 years of age. Results for maximum 1-hour average concentrations of NO₂ were lower: 0.09% (95% CI: -0.01–0.20%) and 0.15% (95% CI: 0.03–0.26%) in all-ages and for those older than 65 years of age, respectively. Increases in daily mortality, for all ages, for cardiorespiratory mortality (0.18% (95% CI: 0.08–0.27%), 24-hour average); cardiovascular mortality (0.34% (95% CI: 0.19–0.48%), maximum 1-hour average, 1.17% (95% CI: 0.82–1.53%), 24-hour average); and respiratory mortality (0.45% (95% CI: 0.21–0.69%), maximum 1-hour average, 1.76% (95% CI: 1.35–2.17%), 24-hour average) were also reported with NO₂. Anderson et al. (2007) also compared multipollutant model estimates for NO₂ from multicity studies and reported that consistent positive estimates for mortality (and hospital admissions) were found before and after adjustment for co-pollutants, with the size and precision of the estimates not being substantially reduced after such adjustment. The authors also concluded that these findings suggested that the short-term associations between NO₂ and health outcomes were unlikely to be confounded by other pollutant measures.

Multicity studies which included adjustment for particles in two-pollutant models, show robust short-term associations of NO₂ with increased all-cause, cardiovascular and respiratory mortality (WHO, 2013).

10.1.1.2 Respiratory effects, asthma and changes in lung function

A number of epidemiological, controlled human exposure, and animal toxicological studies have investigated the effect of NO₂ exposure on respiratory symptoms and lung function. International reviews of these studies concluded that they provide sufficient evidence to infer a relationship between short-term NO₂ exposures and an array of adverse respiratory health effects. The strongest evidence comes from controlled human exposure studies and epidemiological studies that control for the effects of co-occurring pollutants (WHO, 2013; US EPA 2008a; WHO, 2006).

A consistent association has been found in epidemiological studies between air pollution and hospital admissions, emergency department visits and visits to the doctor for respiratory symptoms and asthma in children. Evidence from time-series epidemiological studies indicate increased asthma symptoms and medication use, as well as emergency room visits and hospitalization for asthma, particularly in children, at ambient NO₂ concentrations ranging from 0.018 to 0.036 ppm (24-hour average) (Anderson et al 1997, Atkinson et al. 1999, Galan et al. 2003, Hajat et al. 1999, Lee et al. 2006, Peel et al. 2005, Simpson et al. 2005a, Sunyer et al. 1997).

Australian studies have reported similar associations between hospitalization for respiratory effects, including asthma, and daily NO₂ as overseas studies (Morgan et al. 1998a; Barnett et al. 2005; Erbas et al., 2005; Jalaludin et al. 2004; Rodriguez et al., 2007), although the effect estimates have been mixed, and a few studies reported no associations (e.g. Petroeschovsky et al. 2001). In a meta-analysis of results from 5 Australian and 2 New Zealand cities Barnett et al. (2005) analysed hospital admissions for 3 age groups of children. Statistically significant increases in hospital admissions for respiratory disease (1–4, 5–14 years) and asthma (5–14 years) were associated with interquartile range increases in either 1-hr or 24-hour NO₂. The largest association reported was a 6.0% increase in asthma admissions with a 5.1 ppb increase in 24–30 hour NO₂ and the effect was not reduced by inclusion of PM₁₀ in the analysis. The effect was not reduced by inclusion of PM₁₀ in the analysis.

In the ACHAPS panel study (SCEW, 2012) the most consistent adverse effect was that increased NO₂ exposure was associated with an increased risk of cough and wheezing during the day and night, and increased use of bronchodilators for symptom relief. Relationships between NO₂ and night symptoms and effects were greater for NO₂ 24-hr than for NO₂ 1-hour and were more consistent. For lag 2 NO₂ 1 hour the OR (95% CI) was 1.03 (1.01–1.05) per ppb for the association with night cough. For lag 2 NO₂

24-hour, ORs were 1.06 (1.03-1.09) per ppb for the association with night cough, 1.05 (1.01-1.10) per ppb for the association with night wheeze, and 1.05 (0.99-1.12) per ppb for the association with night shortness of breath. Effects upon symptoms occurring during the day were strongest at lag 0. For lag 0 NO₂ 1-hour, the ORs (95% CI) were 1.02 (1.0-1.03), 1.04 (1.01-1.06), and 1.02 (0.99-1.05) per ppb for associations with day cough, wheeze and shortness of breath respectively. For lag 2 NO₂ 24-hr, ORs were 1.05 (1.02-1.09), 1.11 (1.07-1.16), and 1.06 (1.01-1.11) per ppb for the association with day cough, wheeze and shortness of breath respectively.

Clinical studies indicate that individuals with asthma are more susceptible to the effects of NO₂ compared with healthy individuals. However, the dose-response concentrations have not been adequately studied. In general, young healthy subjects exposed to NO₂ at concentrations below 4ppm for several hours do not experience symptoms, changes in pulmonary function or increased airway resistance. However, exposures to NO₂ in the range of 1.5–2.0ppm can cause small, statistically significant effects on airway responsiveness in healthy individuals. In studies with asthmatics, short term exposure to NO₂ has been associated with increased airway reactivity following exposures to 0.2 to 0.3ppm NO₂ for 30 minutes to 2 hours, and enhanced inflammatory response after exposures to 0.26ppm NO₂ from 15 minute to 30 minutes, followed by an exposure to an airborne allergen (OEHHA, 2007a).

Animal toxicology data support the notion that nitrogen dioxide can induce toxic airway effects, including reduced host defence against microbiological agents and enhanced bronchial hyperresponsiveness in asthmatics to allergen and irritant stimuli. However, these effects have been described in experimental studies following exposure to nitrogen dioxide concentrations far beyond current air quality guidelines and standards. There were no new studies identified that address these issues at concentrations that are considered to be environmentally relevant and that separate the effects of nitrogen dioxide from those of other pollutants (WHO, 2006).

10.1.1.3 Cardiovascular effects

International reviews generally agree that the available evidence on cardiovascular health effects following short-term exposure to NO₂ is inadequate to infer the presence or absence of a causal relationship at this time (WHO, 2013; USEPA, 2008a; WHO 2006). Evidence from epidemiological studies of heart rate variability, repolarization changes, and cardiac rhythm disorders among heart patients with ischemic cardiac disease are inconsistent. In most studies, associations with particles were found to be similar or stronger than associations with NO₂.

A meta-analysis of the associations between pollutants and cardiovascular hospital admissions in the elderly in Brisbane, Canberra, Melbourne, Perth, Sydney, Auckland and Christchurch found statistically significant associations between CO, NO₂, and particles and five categories of cardiovascular disease admissions. The two largest statistically significant increases were for cardiac failure, with a 6.9% increase for a 5.1-ppb unit increase in NO₂ and a 6.0% increase for a 0.9-ppm increase in CO (Barnett et al, 2006).

Studies of hospital admission and emergency department visits for cardiovascular diseases seem to indicate a nitrogen dioxide effect; however, separating the effects of other traffic related pollutants is difficult. Positive associations have been reported in single-pollutant models between ambient NO₂ concentrations and hospital admissions or emergency department visits; however, most of the effect estimates were diminished in multi-pollutant models that also contained CO and particles. Mechanistic evidence of a role for NO₂ in the development of cardiovascular diseases from studies of biomarkers of inflammation, cell adhesion, coagulation, and thrombosis is also lacking. Furthermore, the effects of NO₂ on various haematological parameters in animals are inconsistent and, thus, provide little biological plausibility for effects of NO₂ on the cardiovascular system (USEPA, 2008a).

10.1.2 Long-term exposure

10.1.2.1 Mortality

Results of cohort studies in the United States and Europe examining the relationship between long-term exposure to NO₂ and mortality have been inconsistent. Further, when associations were suggested, they were not specific to NO₂ but also implicated particles and other traffic indicators. Recent European cohort studies provide evidence that the associations between all-cause and cause specific mortality and NO₂ are similar to, if not larger than, those estimated for PM (WHO, 2013).

The recent registry cohort study from Italy (Cesaroni et al., 2013) and the American (Jerrett et al., 2011; Hart et al., 2011) and Canadian (Gan et al., 2011) studies have attempted multi-pollutant models, and they provide support for an effect of NO₂ independent from particle mass metrics. In three of these mortality studies with multi-pollutant models, the major fraction of the populations studied was exposed to NO₂ levels lower than 40 µg/m³; in one of them, nearly all participants were exposed to levels lower than 40 µg/m³ (Jerrett et al., 2011). Four of the six European analyses were centred around 40 µg NO₂/m³. In the French study, areas with (possibly non-representative) monitor averages above 32 µg NO₂/m³ were excluded. A study by Hoek et al (2013) that conducted a review of studies investigating long-term effects of air pollution on mortality outcomes found a 5% increase in all-cause mortality per 10 µg/m³ increase in annual average NO₂.

10.1.2.2 Respiratory morbidity and asthma incidence

International reviews varied slightly in their conclusions about the evidence for an association between long-term exposure to NO₂ and respiratory symptoms, and increases in asthma prevalence and incidence. The US EPA concluded that the epidemiological and experimental evidence is suggestive but not sufficient to infer a causal relationship between long-term NO₂ exposure and respiratory morbidity or asthma incidence (US EPA, 2008a).

The California EPA (OEHHA, 2007b) concluded that the respiratory health effects of long-term exposure to NO₂ have been clearly demonstrated in several large-scale European studies (Ackermann-Lieblich et al. 1997, Schindler et al. 1998; Kramer et al. 2000; Janssen et al. 2003), in a cross-sectional study of children in Alameda, California (Kim et al. 2004) and in the Children's Health Study in Southern California (Gauderman et al. 2004; Gauderman et al. 2005). All agreed that the high correlation among traffic-related pollutants makes it difficult to accurately estimate independent effects in the long-term exposure studies.

The WHO (2000, 2006; 2013) reported qualitative evidence from epidemiological studies of long-term chronic ambient exposures being associated with increased respiratory symptoms and lung function decreases in children at annual average concentrations of 50–75 µg/m³ (0.026– 0.040ppm or higher), which are consistent with findings from indoor studies; although they do not provide clear exposure–response information for NO₂. As with short-term studies, isolating the effects of NO₂ from other pollutants is difficult without the supporting evidence of appropriate clinical and toxicological studies, and the weight of evidence is less for long-term effects. Evidence from animal toxicological studies show that prolonged exposures can cause decreases in lung host defenses and changes in lung structure. WHO (2013) concluded that the association with NO₂ and deficits in lung function growth reported in the 2006 review has been confirmed even in cities with low NO₂ concentrations and there is evidence that this effects is independent of PM₁₀ and PM_{2.5}.

All international agency reviews agreed that studies of lung function, such as the Children's Health Study in California (Gauderman et al. 2004; Gauderman et al. 2005), demonstrate some of the strongest effects of long-term exposure to NO₂. California EPA noted in its review that the findings from the Children's Health Study of reduced lung growth in children exposed to higher levels of NO₂ over

an eight-year period is especially important, since it is a risk factor for chronic diseases and premature mortality later in life (OEHHA, 2007b). These respiratory health effects have been observed in areas with average NO₂ level of 18 to 57ppb, with many in the range of 23 to 37ppb.

The ACHAPS cross-sectional study shows consistent evidence of respiratory adverse effects of NO₂ for both recent and life-time exposure (SCEW, 2012). These adverse effects are manifested as increased risk of asthma-like symptoms (in particular, wheeze), increased airway inflammation and reduced lung volumes. For current asthma and per ppb recent exposure NO₂, the odds ratio (OR) was 1.06 (1.02, 1.10), with OR per interquartile range (IQR) NO₂ 1.26 (1.08, 1.48). For recent wheeze after exercise, the OR was 1.07 (1.03, 1.120) per ppb and 1.32 (1.12, 1.57) per IQR. Airways inflammation as measured by exhaled nitric oxide (NO) increased by 3% (1%-5%) and lung volume as measured by pre-bronchodilator forced expiratory volume (FEV₁) and forced vital capacity (FVC) decreased by 7.1 mL (2.8-11.4) and 6.8 mL (2.7-10.9) per ppb respectively. Effect estimates were slightly smaller for lifetime exposure. Per IQR decreases in lung function measured by FEV₁ and FVC pre- and post-bronchodilator ranged from 27.5 to 29 mL.

There was no evidence that the effects were stronger in atopic subjects. The absence of a greater effect in atopic subjects, the finding that lung volumes, rather than airway calibre (reflected in FEV₁/FVC ratio), and persistence of the effect after bronchodilator, imply that the consequence of NO₂ exposure is not typical asthma; instead, more non-specific lung effects are implicated (SCEW, 2012).

10.1.2.3 Cardiovascular effects

The available epidemiologic and toxicological evidence supporting that long-term exposure to NO₂ and cardiovascular effects is mixed. The Harvard Six City study (Dockery et al. 1993; Krewski et al. 2000) provides some evidence from the US of an association between long-term NO₂ concentrations and both all-cause and cardiopulmonary mortality. The American Cancer Society (ACS) study (Pope, III et al, 2002) failed to find any effect of long-term exposure to NO₂ on cardiopulmonary mortality, while data from Europe (Nafstad et al. 2004), suggested an increased risk of all-cause mortality.

Likewise, European studies provided some evidence of an effect of long-term exposure on lung cancer (Nyberg et al. 2000; Nafstad et al. 2004). Some studies have found associations between chronic NO₂ exposure and cardiovascular disease. Wellenius (2005), Metzger et al. (2004), and Simpson et al. (2005b) all reported an effect of NO₂ on either hospital admissions or emergency room visits for cardiovascular disease after PM was taken into account. Peters et al. (2000) found a strong independent effect of NO₂ on increased risk of defibrillator discharges in patients with implanted defibrillators, while Rich et al. (2005) found that the effect of NO₂ on ventricular arrhythmia was null when PM_{2.5} was included in the model. Pekkanen et al. (2002) found statistically significant associations between risk of ST segment depression and ambient lag 2 day NO₂ in 45 adults with coronary artery disease. NO₂ was moderately correlated with the co-located particle measurements. Two pollutant models for particles and gases were not tested.

10.1.2.4 Cancer

The international reviews concluded that epidemiological studies conducted in Europe have shown an association between long-term NO₂ exposure and increased incidence of cancer, however, the animal toxicological studies have provided no clear evidence that NO₂ acts as a carcinogen (USEPA, 2008a). Both US EPA and WHO suggest that NO₂ may be acting as an indicator of traffic-related carcinogens, and thus the observed increased cancer incidence may be related to exposure of these carcinogens, such as PAHs (USEPA, 2008a; WHO, 2006).

10.1.2.5 Reproductive and development effects

The epidemiologic evidence does not consistently report associations between NO₂ exposure during pregnancy and intrauterine growth retardation; however, some evidence is accumulating for effects on preterm delivery and foetal effects (USEPA, 2008a; WHO, 2006). However, it is unclear whether there is an independent effect for nitrogen dioxide (WHO, 2006). Scant animal evidence supports a weak association between NO₂ exposure and adverse birth outcomes, but it provides little mechanistic information or biological plausibility for an association between long-term NO₂ exposure and reproductive or developmental effects (USEPA, 2008a).

In a review of Australian studies of birth outcomes, few statistically significant associations were demonstrated with NO₂ (Sram et al. 2005). Associations were reported in a Sydney study of approximately 13 400 births of “small for gestational age babies”, where NO₂ was the pollutant associated with the largest reduction in birth weight (34 grams per 0.001 ppm nitrogen dioxide over the third trimester) (Mannes et al. 2005). Similar to the other epidemiological studies, this adverse effect may be due to a mixture of combustion pollutants rather than NO₂ per se. Two other studies in Brisbane reported no association between NO₂ and pre-term birth or sub-optimal foetal growth (Hansen et al. 2006, 2007).

10.1.3 Susceptible groups

Overseas agencies and Australian studies identified infants, children and the elderly (i.e., >65 years of age) as groups that are potentially more susceptible than the general population to the health effects associated with ambient NO₂ concentrations. Individuals with asthma and other chronic lung diseases and cardiovascular diseases are particularly vulnerable (WHO, 2013; NEPC, 2010; USEPA, 2008a; OEHHA, 2007b). The WHO suggest that people with ischemic heart disease and accompanying congestive heart failure and/or arrhythmia constitute a subgroup particularly sensitive to the effects of ambient air pollutants associated with internal combustion engines, including NO₂ (WHO, 2006).

10.1.4 Summary

Overall, there is a large body of epidemiological evidence from overseas and Australian studies showing consistent and statistically-significant associations between adverse health effects and short-term exposure to NO₂ at levels below the current ambient air quality NEPM standards of 0.12 ppm (1-hour average). Ambient NO₂ concentrations from 0.018 to 0.036 ppm (24-hour average) have been associated with increased hospital admissions and emergency department attendance for respiratory symptoms, particularly in asthmatics and children. The effect estimates for NO₂ are robust even after adjusting for the confounding effects of other pollutants. Animal toxicological studies and human clinical trials provide supporting evidence for a mechanism for respiratory effects, with human studies showing cell damage in human lung cells exposed to NO₂ and increased airway reactivity in asthmatics.

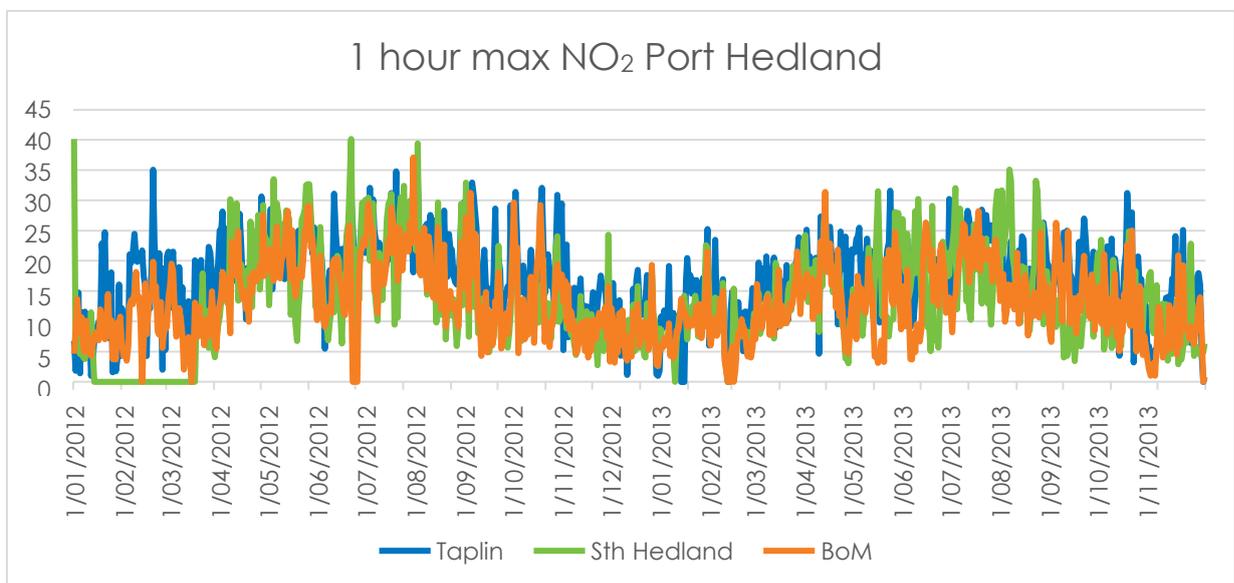
The results from several large U.S. and European multi-city studies and a meta-analysis study observed positive associations between short-term ambient NO₂ concentrations and risk of all-cause (non-accidental) mortality, with effect estimates ranging from 0.5 to 3.6% excess risk in mortality per standardized increment. Australian studies have reported increases in mortality between 0.11% and 0.9% for every 1 ppb increase in NO₂.

Long-term exposure to NO₂ has been associated with decreases in lung function and lung growth and with the prevalence of asthma. No clear association has been found between long-term exposure to NO₂ and cancer.

10.2 Exposure Assessment

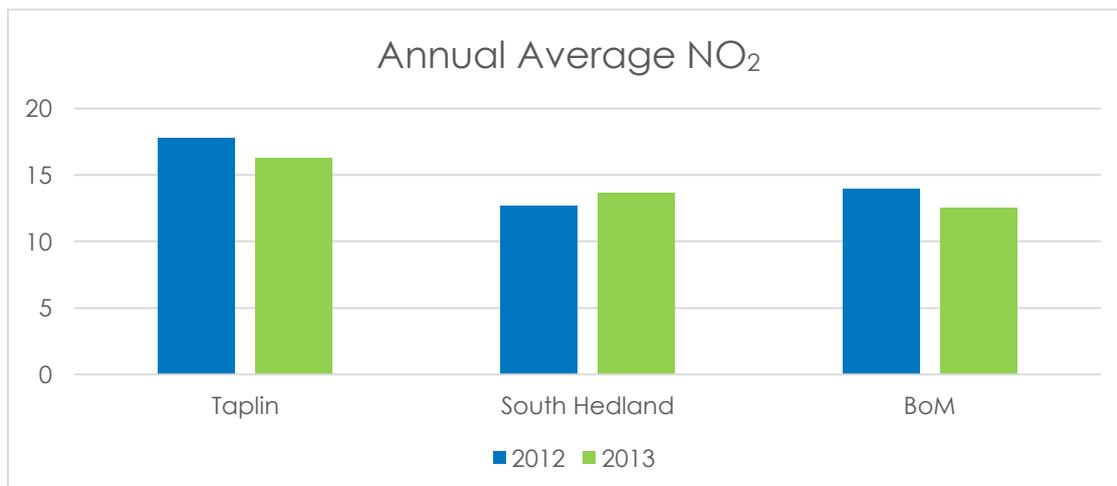
Monitoring for NO₂ was undertaken by PHIC at Taplin St, South Hedland and BoM for the period 2012-2014. **Figure 15** and **Figure 16** show the daily 1-hour maximum and annual average NO₂ levels for 2012-13 which are the most complete datasets. The data shown in **Figure 15** show that for 1-hour maximum values there is very little variation across the monitoring locations. The 2012-13 NPI data (www.npi.gov.au) for Port Hedland identifies that burning (fuel reduction, regeneration, agricultural and wildfires) is the dominant source of oxides of nitrogen (including NO₂) and this is reflected in the regional impact measured at all monitoring sites. Railways and metal ore mining both contribute approximately 6% of total NO_x. The data shown in **Figure 15** is well below the 1-hour maximum NEPM standard for NO₂ of 120ppb.

Figure 15: Daily Maximum 1-hour NO₂ levels



The annual average NO₂ levels are shown in **Figure 16**. The data shown in **Figure 16** show some variation across the monitoring locations with slightly higher levels at Taplin St. All monitored values are below the NEPM annual standard of 30ppb.

Figure 16: Annual Average NO₂ levels



10.3 Risk Characterisation

10.3.1 Health Endpoints

The results of epidemiological studies have shown that a wide range of health effects are associated with exposure to NO₂. Australian studies (NEPC, 2012; EPHC 2006) have found associations between NO₂ levels currently experienced in Australian cities and the following health outcomes:

- Increases in daily mortality
- Hospital Admissions
 - Respiratory disease
 - Cardiovascular disease
- Emergency room attendances asthma

These health outcomes have been assessed in this health risk assessment for the relevant age groups.

Although no studies investigating the long term effects of exposure to NO₂ on health have been conducted in Australia, there have been several international studies that have shown strong associations between long-term exposure to NO₂ and increases in mortality. On the basis of the findings of these studies long-term mortality has also been assessed.

10.3.2 Sensitive Groups

The groups that were identified as being susceptible to the effects of NO₂ are:

- Elderly
- People with existing cardiovascular and respiratory disease
- People with asthma
- Low socioeconomic groups
- Children

10.3.3 Exposure Response Functions

The exposure-response functions in **Table 31** have been taken from Australian studies and in particular two multicity meta-analyses (Simpson et al., 2005; EPHC, 2006). The use of Australian meta-analyses is consistent with the NHMRC (2006) and NEPC (2011) recommendations for selecting exposure response functions for risk assessments for air pollution.

The exposure-response functions for long-term exposure to NO₂ have been taken from the results of a cohort of more than a million adults in Rome (Cesaroni et al., 2013). This study has been reviewed by the WHO as part of the REVIHHAP review. The use of this value is also consistent with the recommendations made by NHMRC (2006) and NEPC (2011).

Table 31: Exposure Response Functions for NO₂ Selected Health Outcomes (EPHC, 2005; Cesaroni et al. 2013)

Outcome	Averaging Period	Exposure Response Function per 1 µg/m ³ increase in NO ₂
Annual all-cause mortality (non-accidental) 30+ years	Annual Average	0.0028
Annual cardiovascular mortality 30+ years	Annual Average	0.0028
Annual respiratory mortality 30+ years	Annual Average	0.0028
Daily all-cause mortality(non-accidental) all ages	1-hour maximum	0.001
Daily mortality respiratory disease - all ages	1-hour maximum	0.0023
Daily mortality cardiovascular disease - all ages	1-hour maximum	0.001
Hospital Admissions respiratory disease 65+ years	1-hour maximum	0.003
Hospital Admissions cardiovascular disease 65+ years	1-hour maximum	0.0014
Hospital Admissions respiratory disease 15-64 years	1-hour maximum	0.001
ED Visits Asthma 1-14 years	1-hour maximum	0.0006

The first part of characterising the risk from NO₂ emissions is to obtain baseline health statistics that are representative of the local community. The baseline health incidence data for Port Hedland and South Hedland from WA Department of Health were used.

The number of cases for each outcome was calculated for the whole population in Port Hedland town and South Hedland using the data monitored at Taplin St and Acacia Way respectively. The number of cases for each day of the year were calculated and then summed to give the annual total. **Table 32** shows the results for NO₂. Sample calculations and baseline health data are shown in Appendix C and Appendix B respectively.

Table 32: Health Outcomes Attributable to NO₂ (number /100,000 population)

Outcome	Port Hedland	South Hedland
Daily all-cause mortality all ages	0.64	0.35
Daily cardiovascular mortality all ages	0.28	0.08
Hospital admissions respiratory disease 65+	546	398
Hospital admissions respiratory disease 15-65 years	2.6	3.4

As can be seen from **Table 32** the number of attributable cases is higher in Port Hedland than South Hedland. In this instance this is due to the higher baseline incidence rates observed for residents of Port Hedland for the outcomes assessed rather than differences in NO₂ concentrations. As can be seen from **Figure 16** there is very little variability in NO₂ concentrations across all monitoring sites.

The highest risk is for hospital admissions for respiratory disease in people 65 years of age and older with 546/100,000 and 398/100,000 additional admissions due to NO₂ for Port Hedland and South Hedland respectively per year. For all other outcomes the risk is lower. For the mortality outcomes assessed there is less than 1 additional death per year per 100,000 population for both Port Hedland and South Hedland.

11 HEALTH RISK ASSESSMENT SULFUR DIOXIDE

11.1 Hazard Assessment

The health effects of sulfur dioxide (SO₂) linked to ambient air exposures have been well studied and reviewed by international agencies such as NEPC (2010), USEPA (2008), WHO (2006) and California EPA (OEHHA, 2000). A review of the SO₂ standard in the AAQ NEPM was also conducted in 2004 by NEPC (NEPC, 2004). As part of this review the health effects of SO₂ were reviewed with a strong focus on studies conducted with short-term exposure (15 mins to 1-hour).

A large number of population-based epidemiological studies have reported a link between short term SO₂ exposure and daily mortality and respiratory and cardiovascular effects. The associations persist when other pollutants, such as particles, are controlled for. The epidemiological evidence is supported by controlled human exposure studies and animal toxicology studies. The strongest evidence comes from controlled human exposure studies examining short term exposure to SO₂ and respiratory effects. These studies have exposed volunteers to SO₂ for periods ranging from 5–10 min up to one hour. Adverse effects, such as sneezing or shortness of breath, occur within the first few minutes after inhalation and are not changed by further exposure. The effects are greater when the person is exercising, and are most pronounced in people with asthma and other respiratory conditions such as COPD, and particularly in exercising asthmatics.

11.1.1 Short term exposure

11.1.1.1 Mortality

A large number of epidemiological studies in cities in various parts of the world, including the United States, Canada and Europe, have reported associations between exposure to ambient levels of sulfur dioxide and increases in all-cause (non-accidental) and respiratory and cardiovascular mortality, often at mean 24-hour average levels of <10ppb (Biggeri et al. 2005; Samet et al., 2000a; Dominici et al., 2003; Burnett et al., 1998a, 2000, 2004; Katsouyanni et al. 1997, 2006; Samoli et al., 2001, 2003; US EPA, 2008; Stieb et al. 2002, 2003). The mortality effect estimates for cardiovascular and respiratory causes are generally larger than for all-cause mortality (Zmirou et al., 1998), and the effect estimates for respiratory mortality are larger than the cardiovascular mortality, suggesting a stronger association of SO₂ with respiratory mortality compared to cardiovascular mortality. The mortality effect estimates from the multipollutant models in the multicity studies suggest some extent of confounding between SO₂ and particles and/or NO₂ (USEPA, 2008).

An association between exposure to ambient levels of sulfur dioxide and increases in mortality is supported by evidence from intervention studies. For example, a sudden change in regulation in Hong Kong, in July 1990, resulted in a restriction that required all power plants and road vehicles to use fuel oil with a sulfur content of not more than 0.5% by weight. Sulfur dioxide levels after the intervention declined by about 50% (from 44 to 21 µg/m³) but PM₁₀ levels did not change. The average annual trend in death rate significantly declined after the intervention for all-cause (2.1%), respiratory (3.9%) and cardiovascular mortality (2.0%) (Hedley et al. 2002). SO₂ was most consistently associated with mortality, whereas the association of PM₁₀ with mortality was only marginal, further supporting the case for SO₂ being more influential than particles, at least in Hong Kong (Wong et al. 2001). Thus, the Hong Kong case study seems to suggest that a reduction in sulfur dioxide (or other pollutants associated with sulfur-rich fuel) leads to an immediate reduction in deaths (WHO, 2006).

11.1.1.2 Respiratory symptoms and diseases

The epidemiological evidence, supported by controlled human exposure studies and a limited number of animal toxicological studies conducted at near ambient concentrations, indicate an association between short-term exposure to SO₂ and several measures of respiratory health, including

respiratory symptoms, inflammation, and airway hyperresponsiveness (Hoek and Brunekreef, 1993; Peters et al., 1996a; Roemer et al., 1993; Segala et al., 1998; Timonen and Pekkanen, 1997; Mortimer et al., 2002; Schildcrout et al., 2006; Schwartz et al., 1994; USEPA 2008).

The epidemiological evidence further indicates that the SO₂-related respiratory effects (\geq 1-hour, generally 24-hour average) are more pronounced in asthmatic children and older adults (65+ years). In the limited number of studies that examined potential confounding by copollutants through multipollutant models, the SO₂ effect was generally found to be robust after adjusting for particles and other co-pollutants (USEPA, 2008).

A number of intervention studies provide further evidence of an association between SO₂ and respiratory morbidity (USEPA, 2008). The Hong Kong "intervention" event described earlier compared the effects of reducing SO₂ (up to 80% in polluted districts) and sulfate (38% in polluted districts) levels on bronchial responsiveness in primary school children living in two districts (polluted and less polluted). The authors found a greater decline in bronchial hyperreactivity and bronchial reactivity in schoolchildren in the polluted than in the less polluted district (Wong et al. 1998). Another study reported a significant decline in symptoms of cough, sore throat, phlegm, and wheezing in children from the polluted compared with the unpolluted district in Hong Kong (Peters et al. 1996b).

The strongest evidence for a causal relationship between respiratory morbidity and short term exposure to SO₂ comes from human clinical studies reporting respiratory symptoms and decreased lung function following peak exposures of 5–10min duration to SO₂. The exact duration is not critical, however, because responses occur very rapidly, within the first few minutes from commencement of inhalation; continuing the exposure further does not increase the effects. These effects have been observed consistently across studies involving mild to moderate asthmatics during exercise. Statistically significant decrements in lung function accompanied by respiratory symptoms including wheeze, chest tightness and shortness of breath have been clearly demonstrated following exposure to 0.4–0.6ppm SO₂.

Although studies have not reported statistically significant respiratory effects following exposure to 0.2–0.3ppm SO₂, some asthmatic subjects (5–30%) have been shown to experience moderate to large decrements in lung function at these exposure concentrations (USEPA, 2008; WHO, 2006). Such effects are enhanced by exercise, which increases the volume of air inspired, thereby allowing sulfur dioxide to penetrate further into the respiratory tract. An acute effect of short-term exposure at rest to 0.2ppm sulfur dioxide is a change in heart rate variability, in which normal young adults responded with small but statistically significant increases in both high and low frequency power, while asthmatic subjects responded with decreases in these parameters of comparable magnitude. A wide range of sensitivity has been demonstrated, both among normal individuals and among those with asthma, who form the most sensitive group for pulmonary function changes. Continuous exposure–response relationships, without any clearly defined threshold, are evident (WHO, 2006).

From the information published to date, the overall conclusion is that the minimum concentration evoking changes in lung function in exercising asthmatics is of the order of 400ppb, although there is the one example of small changes in airway resistance in two sensitive subjects at 100ppb (WHO, 2006). In evaluating this further, judgements are required regarding the clinical significance of such effects, the extent to which particularly sensitive subjects have been represented in the studies, the practical relevance of the enforced exercise required to enhance the effects, and how to relate the short (10- to 15minute) exposures to the more usual hourly average monitoring data (WHO, 2006).

11.1.1.3 Cardiovascular effects

Epidemiological studies have examined the association between air pollution and cardiovascular effects, including increased heart rate (HR), reduced heart rate variability (HRV), incidence of ventricular arrhythmias, changes in blood pressure, incidence of myocardial infarctions (MI), and emergency department visits and hospitalizations due to cardiovascular causes. The epidemiologic evidence from studies of the effect of SO₂ on ICD recorded arrhythmias, blood pressure and blood markers of cardiovascular risk failed to provide consistent evidence to suggest a role for SO₂ in cardiovascular disease development (USEPA, 2008).

Many researchers were unable to distinguish the effect of SO₂ from correlated copollutants while others reported a reduction in the SO₂ effect in two-pollutant models (USEPA, 2008). Tunnicliffe et al. (2001) measured cardiac function associated with acute exposure to SO₂ in a controlled human exposure study involving 12 normal and 12 asthmatic young adults. Exposures were of 1-hour duration, double blind, in random order, >2 weeks apart, and with clean air and 200ppb sulfur dioxide. The sulfur dioxide exposures were associated with statistically significant increases in high frequency (HF) and low frequency (LF) power in the normal subjects, and reductions in HF and LF of comparable magnitude in the asthmatic subjects. No pulmonary function changes or symptom frequency changes were observed in either group. These results suggest that sulfur dioxide exposures at concentrations frequently encountered during air pollution episodes can influence the autonomic nervous system. This may help in elucidating the mechanisms involved in the induction of bronchoconstriction and the cardiovascular effects of ambient air pollution (WHO, 2006).

Although biologically plausible modes of action that could explain short-term SO₂ effects on the cardiovascular system have been identified, consideration of these modes of action in light of findings from additional animal toxicological, human clinical and epidemiological studies led the USEPA to the conclusion that the evidence as a whole is inadequate to infer a causal relationship (USEPA, 2008). Specifically, evidence from human clinical and epidemiological studies of HRV in healthy persons as well as persons with asthma or cardiovascular disease was inconsistent and did not support an effect of SO₂ on the autonomic nervous system, despite some positive findings.

Several studies have observed positive associations between ambient SO₂ concentrations and emergency department visits or hospital admissions for cardiovascular diseases (e.g., all cardiovascular diseases, cardiac diseases, cerebrovascular diseases) particularly among individuals 65+ years of age, but results are not consistent across studies. The strongest evidence comes from a large multicity study conducted in Spain (Ballester et al. 2006) that observed statistically significant positive associations between ambient SO₂ and cardiovascular disease admissions, however, the SO₂ effect was found to diminish by half with PM₁₀ and CO adjustment. In an Australian study, Jalaludin et al. (2006) reported a 3% excess risk in cardiovascular disease hospital admissions per 0.75ppb incremental change in 24-hour average SO₂ in single-pollutant models, which was reduced to null when CO was included.

11.1.1.4 Hospital admissions and emergency department attendances

A large body of epidemiological studies generally report consistent and robust associations between ambient SO₂ concentrations and emergency department visits and hospitalizations for all respiratory causes, particularly among children and older adults (65+ years), and for asthma and chronic obstructive pulmonary disease (COPD) (USEPA, 2008). Mean 24-hour average SO₂ levels in these studies ranged from 1 to 30ppb, with maximum values ranging from 12 to 75ppb (e.g. Barnett et al. 2005; Sunyer et al., 1997, 2003; Anderson et al., 1998; Hajat et al., 1999; Schouten et al., 1996; Spix et al., 1998; Wong et al., 1999a).

Some studies report greater increase in emergency department visits and hospitalizations with season. Schouten et al., 1996; Spix et al., 1998; Wong et al., 1999 and others found the associations, with similar increases in SO₂, to be greater in winter (Castellsague et al., 1995; Tenias et al., 1998; Wong et al., 2002c; Vigotti et al., 1996; Walters et al., 1994). Warmer months were more likely to show evidence of an association with adverse respiratory outcomes in children, while older adults appeared more likely to be affected during the cooler months.

In a case-crossover study of air pollution and child respiratory health undertaken in five Australian and two New Zealand cities, Barnett et al. (2005) found a statistically significant increase in hospital admissions and SO₂ with an interquartile range of 5.4ppb for 1-hour SO₂. The ambient levels recorded during the study included: SO₂ 1 hour mean (3 cities) 7.1ppb, range of means 3.7 to 10.1ppb; 24 hour mean (4 cities) 4.5ppb range of means 0.9 to 4.3ppb. In the 1–4 year age group there was evidence of seasonal impacts on pneumonia and acute bronchitis admissions for SO₂ (May to October 4.9% increase 95% CI, 0.6–10.8%, November to April 10.4% increase 95% CI, 2.1–19.4%) (Barnett et al. 2005).

11.1.2 Long term exposure

11.1.2.1 Mortality

Epidemiological evidence on the effect of long-term exposure to SO₂ on mortality is limited, and according to the US EPA (2008), is inadequate to infer a causal relationship. Overall, reanalysis of results from two major U.S. epidemiological studies (Pope et al. 1995; Dockery et al., 1993) observe an association between long-term exposure to SO₂ or sulfur-containing particle air pollution and mortality (Pope et al. 2002; Krewski et al. 2000; Jerrett et al., 2003a; Elliott et al. 2007). However, several other U.S. and European cohort studies did not observe an association (Abbey et al. 1999; Lipfert et al. 2000b; Nafstad et al. 2004; Filleul et al. 2005; Beelen et al. 2008). The lack of consistency across studies, inability to distinguish potential confounding by copollutants, and uncertainties regarding the geographic scale of analysis, limit the interpretation of a causal relationship (USEPA, 2008).

Evidence from epidemiological studies shows positive and statistically significant associations between a reduction in life expectancy and long-term exposure to particulate pollution (PM_{2.5} and sulfate) and SO₂. This was noted in the Committee on the Medical Effects of Air Pollutants (COMEAP) Report on the Long Term Effects of Particles on Mortality (Committee on the Medical Effects of Air Pollutants, 2001).

11.1.2.2 Morbidity

The results of studies examining the association between long-term exposure to SO₂ and respiratory morbidity are generally inconsistent. Cross-sectional studies conducted in New South Wales in the Hunter and Illawarra regions found no association between annual average levels of sulfur dioxide and prevalence of asthma in children (Henry et al, 1991) and chest colds and respiratory symptoms such as cough and wheeze (Lewis et al, 1998). Studies identified by the USEPA (2008) that examined the effects of long-term exposure to SO₂ on asthma, bronchitis, and respiratory symptoms observed positive associations in children. In the limited number of studies examining the SO₂ associations with lung function, results were generally mixed.

A major consideration in evaluating SO₂-related health effects and long-term exposure is the high correlation, and potential confounding, among the copollutant levels observed, particularly between long-term average particle concentrations and SO₂. The USEPA (2008) concluded in its review that the overall epidemiological evidence on the respiratory effects of long-term exposure to SO₂ is inadequate to infer a causal relationship. The available toxicological and epidemiological evidence on the effect of long-term exposure to SO₂ on cardiovascular health is also too limited to make any conclusions.

11.1.2.3 Birth outcomes

A number of studies have reported associations between exposure to SO₂ and low birth weight and premature birth (Sram et al., 2005; Dugandzic et al., 2006; Jalaludin et al 2007). A study of 123,840 singleton births of over 20 weeks' gestation in Sydney, between 1998 and 2000, found that 4.9% of babies were born at less than 37 weeks gestation. The mean of the one hour maximum SO₂ levels was 3.6ppb. SO₂ level in early pregnancy had a large adverse impact on gestational age in those infants conceived in autumn and winter for a 1ppb increase in SO₂. The authors noted that SO₂ appears to be an important pollutant, despite SO₂ levels in Sydney being well below the national standard, with vehicular traffic being the primary source and it is conceivable that SO₂ is a marker for traffic related air pollutants in the study (Jalaludin et al 2007). A Canadian study found that first trimester exposures in the highest quartile for SO₂ and PM₁₀ suggested an increased risk of delivering a low birth weight infant (Dugandzic et al., 2006). Leem et al. (2006) also found an association between low birth weight and low levels of air pollutants including SO₂ in Korea. In the USA, a time series study undertaken by Sagiv et al. (2005) found evidence of an increase in preterm birth risk with exposure to PM₁₀ and SO₂, which were consistent with prior investigations of spatial contrasts. toxicological studies provide very little biological plausibility for the effects. The limited number of studies, inconsistent results across trimesters of pregnancy, and the lack of evidence regarding confounding by copollutants limit the interpretation of these studies and make it difficult to draw conclusions regarding the effect of SO₂ on birth outcomes.

11.1.3 Threshold for effects and sensitive groups

The reported associations between exposure to SO₂ and adverse health outcomes from overseas studies relate to a range of 24 hour average and daily one hour maximum exposure levels including very low levels, suggesting that there may be no threshold for the health effects associated with exposures to sulfur dioxide in sensitive subgroups of the population.

Asthmatics appear to be the most susceptible group to the effects of sulfur dioxide (WHO, 2006; USEPA, 2008; Streeton, 1997). The elderly are also a susceptible population as they have reduced respiratory reserve as a result of the ageing process. This is also often exacerbated by pre-existing cardio-respiratory disease.

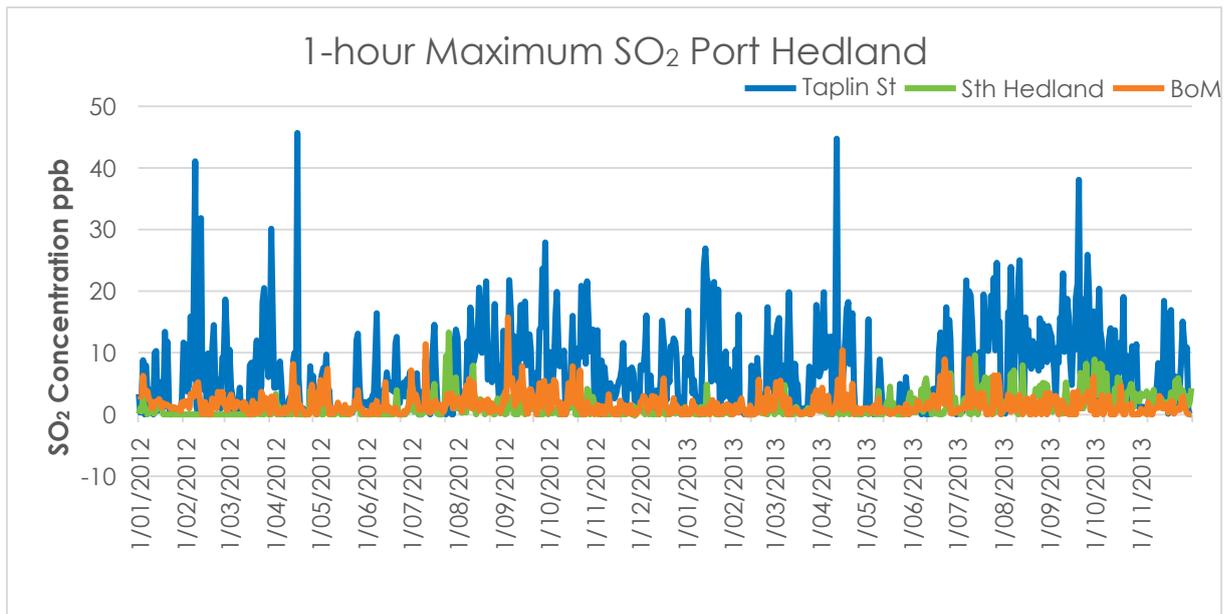
The studies reviewed indicate that short-term exposures of 5-15 minutes to sulfur dioxide are associated with a dose-response effect on lung function of exercising individuals with asthma. The controlled exposure study by Linn et al (1987) in exercising individuals with asthma is indicative of a LOAEL of 0.2ppm for a 15 minute exposure period for this small sample of susceptible individuals. Responses to brief short-term exposures to sulfur dioxide are immediate and do not appear to worsen after longer exposure periods.

Epidemiological studies that have examined longer exposure times (one hour maximum, 24 hour and annual average) indicate that other susceptible populations, in addition to people with asthma, may include those with chronic obstructive pulmonary disease and existing cardiovascular disease, children and the elderly. Compared to healthy adults, children are generally more sensitive to air pollutants as their exposure is generally higher. The reasons for this are that children inhale more air per minute and have a larger contact lung surface area relative to their size compared to adults. Other factors that increase the potential for exposure in children are that they generally spend more time outdoors and exercising.

11.2 Exposure Assessment

The 2012-13 NPI data indicates that the main sources of SO₂ in Port Hedland are water transport support services, metal ore mining, commercial shipping and boating and railways. SO₂ has been monitored by PHIC from 2011-2014 at Taplin St, BoM and South Hedland. The data for 2012-13 (the most complete dataset) is shown in Figure 17 to Figure 19 for the maximum daily 1-hour, 24 hour and annual average concentrations.

Figure 17: Maximum Daily 1-hour Average SO₂ Levels



The data in **Figure 17** shows that the maximum 1-hour SO₂ levels at Taplin St are higher than those observed at both BoM and South Hedland. This is indicative of the influence of the activities at the Port consistent with the NPI data. The recorded SO₂ levels are well below the NEPM standard of 200 ppb 1-hour maximum.

A similar pattern is seen for the 24-hour and annual average SO₂ data shown in **Figure 18** and **Figure 19**.

Figure 18: 24-hour Average SO₂ Levels

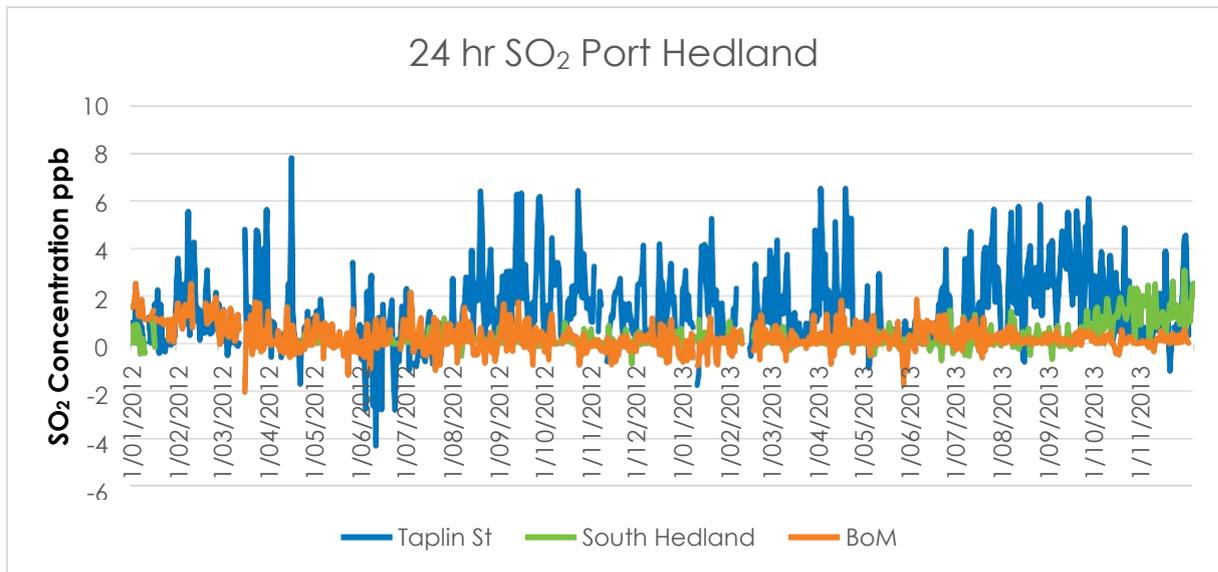
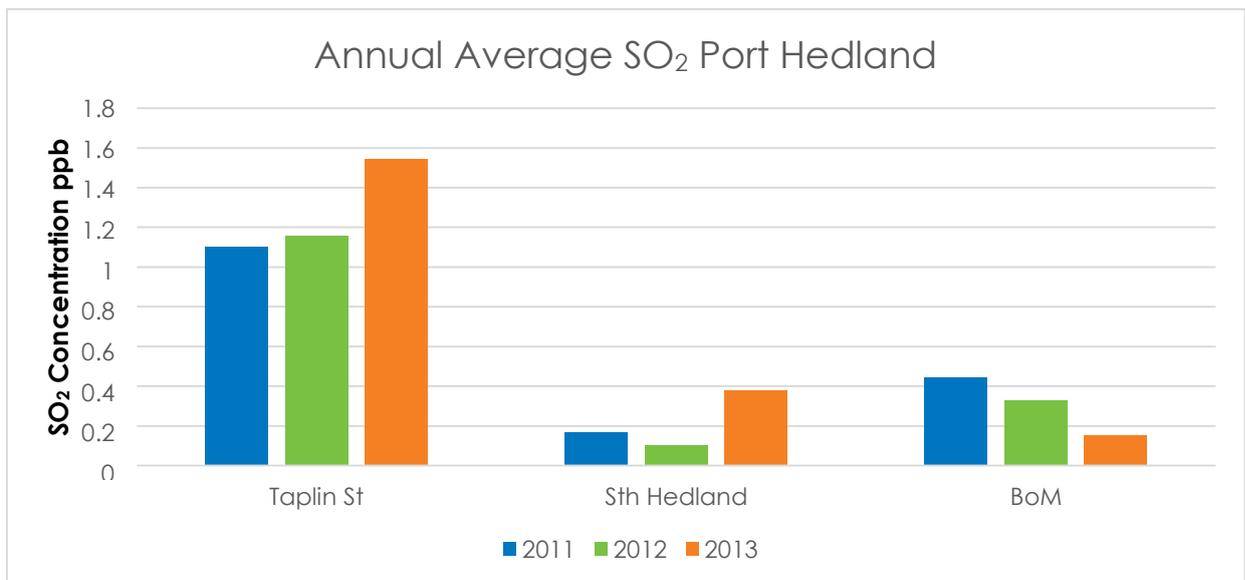


Figure 19: Annual Average SO₂ Levels



The data shown in Figure 18 and Figure 19 are well below the 24 hour and annual average NEPM standards of 80ppb and 20ppb respectively. The influence of the Port activities is still observed with higher levels observed at Taplin St compared to the other sites. The data in **Figure 19** shows that the SO₂ levels are increasing in Port Hedland (as measured at Taplin St) and South Hedland with a substantive increase in annual average SO₂ levels in 2013.

11.3 Risk Characterisation

The risks associated with exposure to SO₂ have been calculated for Port Hedland using the Taplin St data and for South Hedland. The only exposure response relationship that could be identified in the literature for which baseline health statistics were available is for daily all-cause mortality. The exposure response relationship that was used is 0.01% increase in all cause mortality per 1 ppb and 24-hour SO₂. This value has been taken from the APHEA2 study in Europe (Katsouyanni et al., 2006). The risk per 100,000 population for both locations is shown in **Table 33**.

Table 33: Health Outcomes Attributable to SO₂ (number /100,000 population)

Outcome	Port Hedland	South Hedland
Daily all-cause mortality all ages	0.03	0.005

As can be seen from **Table 33** the attributable risk of all-cause mortality associated with SO₂ is very low and below 1 in a million risk which is usually considered as an acceptable level of risk.

12 CONCLUSIONS

The results of the HRA has shown that the pollutant that poses the greatest risk to the health of the Port Hedland community is PM₁₀. For the gaseous pollutants and respirable crystalline silica the risk levels are low and within limits that are generally considered as acceptable risk. For asbestos fibres monitoring data did not find any fibres above the level of detection of the analytical method used.

For the metals studied the risks were below levels of concern although the monitoring data indicate a clear influence of industry in the data collected at Richardson St and Taplin St. The concentrations of the metals are much higher at Richardson St than at the other locations. Although the risk from exposure to manganese has been calculated to be within acceptable levels, it is close to the limit of what is considered acceptable at Richardson St. With increased exports through the Port strict dust management will be required to ensure that the manganese levels do not increase to an unacceptable level of risk.

The risks associated with PM₁₀ exposure are significant. The morbidity and mortality risks per 100,000 population are higher in Port Hedland using either the Richardson St or Taplin St data than those at South Hedland. The risks would be substantially reduced if dust levels were managed so that the interim guideline of 70 µg/m³ or the NEPM standard of 50 µg/m³ could be met. Analysis of the risk of increased mortality at the west end, east end and South Hedland shows that the risk is higher in the west end compared to the other locations. This is consistent with the findings of the 2006 Department of Health study that showed that the rate of hospital admissions for respiratory disease and cardiovascular disease was higher in the west end of Port Hedland compared to the east end.

Analysis of the PM₁₀ monitoring data from all sites including the background site at Yule River indicate that many of the exceedances of the standards in Port Hedland are due to local sources and not regional dust. If regional dust was the main source then elevated levels would be expected at all sites. In the monitoring data provided for the study this situation did occur frequently. Analysis of wind direction on days of high levels of PM₁₀ in Port Hedland shows that the predominant wind direction when levels at Richardson St and Taplin St were elevated were from the S-SE. On many of these days the levels at South Hedland were not elevated. This suggests that there is a source between South Hedland and Port Hedland town that is impacting on PM₁₀ levels at Richardson St and Taplin St. Further analysis is required to fully understand the influence of local sources.

The health risks associated with exposure to PM₁₀ has been assessed using the current monitoring data. As the exports per year increase PM₁₀ and metal levels are likely to increase. Any increase in ambient levels will lead to an increase in risk. When compared against the regulatory standards of PM₁₀ for protecting public health from major developed countries, the level of PM₁₀ reported during the monitoring period in Port Hedland would present an unacceptable level of health risk.

There is a clear influence of industry and associated Port activities on the levels of metals monitored at Richardson St. Although currently below the guideline values adopted for the HRA increased exports, especially of manganese, may change this situation. This needs to be carefully monitored to ensure that air quality guidelines are not exceeded in the future.

13 UNCERTAINTY AND LIMITATIONS

A qualitative assessment of sources of uncertainty was undertaken to provide an indication of the effects of uncertainty on the results of the health risk assessment. Some of the factors that were considered during the assessment of uncertainty were:

- effects due to the selection or rejection of data; such effects were minimised through the data quality assurance process that was agreed to by the WA Department of Environment Regulation so that monitoring data were assessed independently before being used in this assessment. Analytical data was verified by the WA ChemCentre.
- selection of contaminants of concern: some airborne components may not have been identified as important at the time of developing the HRAM. Diesel particulates for example were not considered in this assessment. Given that the levels of PM_{2.5} were subsequently shown not to be significantly elevated the likelihood that diesel emissions are significant contributors to health outcomes is considered to be minimal therefore the effect on this assessment is minimal. It is recommended that future monitoring include PM_{2.5} to ensure to capture combustion related particles and any increases above the NEPM for PM_{2.5} can be detected and addressed.
- adequacy of the sampling strategy and analysis; in principle more monitors would result in greater certainty in describing the distribution pattern of PM₁₀ however the reality is that monitoring is expensive and the number, type and location of monitors were deemed to be sufficient for this assessment. An error was identified in the analysis of Cr VI; incorrect filters were used for chromium VI analysis which was discovered only after a significant period of time had elapsed. Consequently the health impact of Cr VI was not assessed due to lack of data. The potential for this error significantly impacting the outcomes of the metals assessment is high however the likelihood of this occurring is deemed to be very low because Cr VI was considered a minor constituent of airborne emissions in Port Hedland. Nevertheless it is recommended that a short period of campaign monitoring for Cr VI is undertaken to confirm this assumption. The sampling program for Si and asbestiform fibres was shorter than 12 months which is the usual time period needed for assessing pollutants with carcinogenic potential. Given that both pollutants were well below the levels of concern during the three months of monitoring in the locations of greatest concern the impact on the risk calculations is deemed to be minimal. It is however recommended that should the constituents of the ore bodies change to reveal Si and asbestiform material, Si and asbestos monitoring should be considered.
- Background exposure: it was assumed early in the investigation that background exposure to PM₁₀ may contribute substantially to the overall exposure in Hedland. This assessment has cast significant doubt that background exposures from regional dust excursions into Hedland and Port Hedland in particular are significant when compared to local dust sources. This assessment considers the effect of PM₁₀ exposure from any crustal source therefore the local impacts do not readily discriminate between contributory impacts from various industry activities and types except where stated in the hazard assessment sections. To better understand the pattern and extent of local dust sources it is recommended that boundary monitoring data from industry is evaluated to better understand Industry contribution to fugitive local dust.
- Population Exposure: Modelling of PM₁₀ is required to help inform land-use decision making. This assessment has established that 70 µg/m³ should not be exceeded and modelling will

assist with understanding the level of exposure reduction and the level of risk saving that will be required to achieve this level.

- identification of sensitive populations and confounders: TRV's inherently take into account generic susceptible and sensitive individuals. This assessment has gone further by characterising the local population and their health outcomes in terms of morbidity and mortality. This type of epidemiological assessment is highly dependent on quality hospital and health data. While the quality of the epidemiological data was independently verified by the DoH Epidemiological Branch the population numbers are small and subject to overestimation of effects with relatively small changes in population. This is a significant limitation which has been reduced as far as possible through sensitivity analysis in the risk characterisation sections. Impact on the conclusions of the HRA is minimal although it is recognised that there may be a very small number of unusually sensitive individuals that if exposed may contribute disproportionality to the assessment. This assessment makes no attempt to characterise lifestyle and effects of smoking or obesity on the health outcomes other than considering these influences on the HRA outcome in the exposure sections in a qualitative way. This is no different than any other public health assessment using regulatory guidelines and state health statistics.
- Toxicological potency of crustal PM₁₀: the potency of crustal dust has been evaluated extensively in this assessment. The E/R relationships and dose-response relationships rely on established and emerging lines of evidence. The relative potency of airborne PM at Port Hedland (i.e. ore and crustal dust, sea salt, iron oxide) versus locations supplying PM exposure response relationships (i.e. urban PM from large cities, Saharan dusts) has been considered. Where possible the exposure response relationships have been adjusted to compensate for this limitation. It is anticipated that the potency of airborne PM will change as the concentration of gaseous pollutants increases due to population and transport growth. Therefore it is recommended that data gathered for this HRA serve as a baseline assessment against which to assess future PM emissions. It is recommended that a permanent monitoring program is established to enable future assessments for the purpose of protecting public health.
- It is impossible to attempt to characterise interactions between all pollutants, beyond the well-recognised photochemical reactions. In addition, the health effects of some substances are poorly understood. The range of pollutants monitored and analysed for this assessment was restricted to those reasonably anticipated to be of consequence to the population of Hedland and this assessment did not address other pollutants. Regardless of the effect or mode of toxicological action, additivity of either dose or effect was assumed. While this has the potential to overestimate the risks to combined exposures it compensates for those components of unknown toxicity.
- This assessment HRA also did not address nuisance and amenity concerns; while these do not have direct health effects it has been well established that persistent amenity nuisance from intermittent very high particulate concentrations such as those seen in the exceedances of the 70 µg/m³ may lead to indirect health manifestations through stress related to dealing with amenity discomfort. It was not possible to quantify these effects in this assessment however nuisance and amenity should be considered during the land-use planning process. Local greening may assist with reducing both adverse health and negative amenity impacts.

- Indoor exposure: this assessment implicitly assumes that indoor environments are just as polluted in terms of PM as outdoor environments. This is the usual assumption in these types of assessments. It is possible that certain indoor pollutants may be higher than outdoor pollutants and may contribute to the health outcomes. It is not known how much PM infiltration occurs into buildings and the contribution of infiltrated PM on health. Since no personal and building monitoring was undertaken it was reasonable to assess only external ambient air pollution. Current thinking is that an air conditioned/filtered indoors environment reduces exposure however scientifically validated studies are lacking. Future research directions may wish to include a personal and building exposure investigation.
- Synergistic effects of pollutants has not been considered in this report. A review of the current literature did not provide evidence that exposure to the pollutants considered in this HRA have synergistic effects or provide a methodology that could be applied to assess any such potential effects. However, given that the pollutants considered in the HRA are associated with the same health outcomes in many cases, eg., all-cause mortality, and that the epidemiological studies have indicated that the effects of individual pollutants are independent in many cases, the increase in risk could be considered additive for these outcomes. This is consistent with the enHealth guidance on the additive nature of hazard indices (HQ) to give an overall health index for a given health outcome. The HRA has identified that PM₁₀ dominates the health risk to the Port Hedland population due to air pollution. The risk posed by current levels of PM₁₀ exceed the international guidance on acceptable risk. Assessing the additive or total risk for all air pollutants for the same health outcome would not change this conclusion or the actions required to reduce the public health risk in Port Hedland from exposure to air pollution.

14 RECOMMENDATIONS

It is recommended that the following are considered; justification for each is provided in the preceding section.

- On-going ambient air monitoring program for PM₁₀ and PM_{2.5} in Port Hedland and South Hedland be implemented to enable ongoing assessment of risk to the Hedland population attributable to dust. While 50µg/m³ is the acceptable guideline for Australian jurisdictions, it may not be possible to meet this number in all parts of Hedland. The level of risk saving in reducing the exposure from 70 to 50 is currently small and therefore a determination should be made on the level of acceptable for the population. It should be borne in mind that as the population increases so will the number of susceptible individuals therefore the actual number of adverse health outcomes for example increases in mortality or hospitalisation, will increase. Therefore a program to discourage future permanent settlement in Port Hedland may need to be considered as a management strategy to reduce exposure and subsequent public health risk.
- Exposure reduction in the form of greening of exposed land areas should be encouraged.
- A period of monitoring for CrVI and Cr III to enable an assessment of the potential risk posed by these pollutants be undertaken.
- Industry makes their boundary data available for assessment of their contribution to local dust in Port Hedland.

- Modelling of PM₁₀ against current and future scenarios should be undertaken and made available for further assessment through a HRA.
- Regulatory monitoring for Si and asbestos is considered when industry analysis of mineral ores identifies for Si or asbestiform material.
- Nuisance and amenity is considered during land-use planning decisions.
- Future research be conducted to consider personal exposure monitoring.

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Appendix A **RESPONSE TO PEER REVIEW COMMENTS**

PEER REVIEWER 1: Dr Brian Priestley

Issue 1: A number of key references are missing

Response: All missing references have been included in the reference list.

Issue 2: Consideration should be given to including a reference by Hoek et al (2013) which provides support to the findings of the HRA.

Response: Study by Hoek et al has been reviewed and the findings included in the HRA report in Sections 6.1.2 and 10.1.2.

Issue 3: Include discussion on potential synergistic effects in Section 10 of the report.

Response: Discussion included in Section 10 of the report.

Issue 4: Colour shading of Figures 1 and 2 do not match the legends.

Response: These diagrams were taken from the original DoH (2006) report. The error is in the original diagrams and cannot be corrected.

Issue 5: Equations used to calculate the increase in risk should be included in the report to increase transparency. More explanation on the use of the monitoring data needs to be included. Clarification of what year of data was used should be included.

Response: The equation has been included in Section 6.8 of the report with discussion on how it has been applied. Further clarification on the treatment of the monitoring data and what years were used has also been provided Section 6.8.

Peer Reviewer 2: Professor Jack Ng, Entox.

Issue 1: A significant number of references are missing. Report requires careful proof reading.

Response: All missing references have been incorporated and the report has been proof read. All editorial changes recommended throughout the peer review document have been addressed through this process.

Issue 2: Executive summary is too brief and should include discussion on pollutants other than PM₁₀.

Response: Executive summary has been revised and includes reference to all findings of the HRA.

Issue 3: Information on the number of exceedances of the PM₁₀ standards should be included for each year.

Response: A Table showing the number of exceedances for each year for each location has been included Table 6 on page 62.

Issue 4: Further clarification in the last paragraph of the Executive summary is required in relation to the increase in population and risk. Clarification should be provided as to what standard is being targeted in Port Hedland.

Response: The last paragraph has been clarified. Given that the HRA has calculated population risks rather than individual risk, as the population increases if PM₁₀ levels remain the same then the number of people exposed will increase with an associated increase in population risk. This will be reflected in the number of attributable cases associated with exposure to PM₁₀.

With respect to what standard/guideline is to be applied to Port Hedland, this is a Government decision and is not part of the HRA.

Issue 5: Clarification is required in relation to the data quoted from the WA DOH report on hospital admissions (2006) and whether comparisons have been made to the State population. Clarification is also sought as to whether the data has been corrected for asthmatic patients.

Response: The data presented in the 2006 DoH report and reproduced in the HRA is compared to the rates within the Pilbara region more broadly not to the rest of the State. This information is provided in Section 2. The DoH report provides data for all respiratory admissions and does not correct for asthmatic patients. The HRA has not reanalysed any of this data but simply reports the findings of the study. The black areas on the graphs indicate that the results were not statistically significant.

Issue 6: Information on the number of exceedances for PM₁₀ and PM_{2.5} should be included Executive Summary and the risks associated with these exceedances calculated.

Response: The Executive summary has been revised and includes the number of exceedances of the relevant guidelines. The risks posed by PM₁₀ and PM_{2.5} are presented in section 6.3 of the HRA.

Issue 7: Colour shading of Figures 1 and 2 do not match the legends.

Response: These diagrams were taken from the original DoH (2006) report. The error is in the original diagrams and cannot be corrected.

Issue 8: Table 6 (now Table 8): It might be worthwhile to extend the discussion a little noting that the Max and Average of PM10 of all monitoring sites have improved in 2013 with the exception of Richardson Street and why this might be so? Please include standard deviation (or SEM error bars) of the mean in all tables and figures for the monitoring data. This will give the reading an appreciation of the variation. Where appropriate, statistical analysis should be performed for comparison of air quality at different sites.

Response: Summary statistics for all sites have been included in this table. The standard deviation could not be calculated for the dataset as the error in each of the measurements was not made available for the HRA.

Issue 9: Tables 7-11 (now 9-13): Please state which descriptive statistical data was used for the calculation – the average (annual, or over the monitoring period?), median or certain per centile? Whichever was used the authors should justify why. I would have thought that risks based on both average and 75th percentile would be informative.

Response: Further text has been added to describe the data set that has been used. All data has been used, as monitored, in the HRA. For the short-term effects each day of the year was used as monitored. For long-term effects the annual average was used. To meet the alternative standards a roll back procedure was used which adjusted the entire dataset so that the maximum values did not exceed the relevant standards.

Issue 10: Should consider to provide the standard deviation for the average coarse particles. Please also state which methodology was used to determine the coarse particulate fraction.

Response: The information to calculate the standard deviation for the annual average coarse particles was not available. The coarse fraction was calculated by subtracting the same day PM_{2.5} concentration from the PM₁₀ concentration.

Issue 11: Manganese section. Will need to be reorganised. It contains animal studies and human studies, acute and chronic studies all mixed in the same section whereas these are nicely separated in other sections of this report. Some of the studies cited are lacking in dosing regimen/exposure level which makes the discussion less informative. The authors did not provide references in places where they mentioned "some studies", and "few studies".

Response: This section has been redrafted.

Issue 12: Manganese section. This is a quite a large section for chronic effects of Mn. It will benefit from a more organised structure to categorise the types of studies or types of effects. At the moment, whilst the information presented is quite comprehensive, it seems to be a randomised collection of information.

Response: This section has been redrafted.

Issue 14: Pages 87-88: Y-axis label and concentration unit are missing in the Figures. By including error bars of the means, the figures will be more informative.

Response: Graphs have been amended.

Issue 15: Page 97, paragraph 4: "... but the USEPA concluded that no coherent picture is evident at this time". This was the conclusion in 2008. Has the opinion changed to date by USEPA or other agencies?

Response: New studies have been identified by WHO (2013) and these are discussed in Section 7.4.1.1.

Issue 16: Page 99, paragraph 3, NO₂ animal studies: "There were no new studies.... (WHO 2006)". Are there new studies since 2006?

Response: There are new epidemiological studies which are discussed in Section 7.4.1.1. No new animal studies were identified.

Issue 17: NO₂ summary, The Summary only limited to acute exposure, how about extend it to cover chronic exposure effects and possibly cancer

Response: Text has been modified to include these effects.

Issue 18: Note SO₂ in Y-label and chart title is with a subscript 2. Check the correct numbering of Figure 17 to 19. Note Figures 18 and 19 are missing Y-label and units. Bar charts should have standard deviation and significant differences indicated by p value ($p < 0.05$).

Response: Figures have been amended.

Issue 19: Less than one in million risk is of course considered as acceptable risk for SO₂. However, the attributable risk for NO₂ is less than one in 100,000. Is this still an acceptable risk relative to SO₂?

Response: It is generally accepted that between 1 in 100,000 and 1 in 1 million is considered acceptable risk. The NO₂ risk is at the upper bound of this limit.

Issue 20: Page 117: Additivity of risk is assumed here. Then, the authors should summarise and tabulate the "total risk" attributable from air pollutants in Port Hedland.

Response: Additivity is not assumed for all pollutants. The risk is estimated from individual pollutants.

Peer Reviewer 3: Cardno ChemRisk

Issue 1: There is insufficient detail in the exposure assessment in regard to the air monitoring and basis of the air monitoring network in representing population exposure.

Response: Information has been provided in Section 6.2 on the basis of the networks and the sampling methods used. No further information has been provided to the authors if the HRA. Only monitoring data was provided. All available published information has been included in the report.

Issue 2: The calculation of the health risks due to each pollutant is insufficient to reproduce the calculations. The exposure response functions were not available and not all studies could be verified and were not available through PubMed or on the internet. The concentration levels used in the calculations are not provided in any tables.

Response: The equation used to calculate the risk has been included in Section 6.2.1 with further information on the application of the air monitoring data in the calculations. The equation used is consistent with that proposed in the HRAM. In addition all spreadsheets have been included in Appendix C and sample calculations for each pollutant in Appendix B.

All exposure response functions used in the HRA are provided in Section 6.1.7, Tables 4 and 5 for PM₁₀ and PM_{2.5} respectively, Section 10.3.3, Table 19 for NO₂. These values were included in the draft HRA. The exposure response data for SO₂ has been included in Section 11.3 for SO₂.

The studies that are referred to Simpson et al., 2005b and EPHC (2006) and (2011) are all peer reviewed and publicly available documents. The references are included in the reference list. Simpson et al, 2005b was published in Australian & New Zealand Journal of Public Health, 29, 205-12. The EPHC reports are available on the SCEW website www.scew.gov.au/air.

Summary statistics, including annual averages are included in the HRA. All data used in the calculations was incorporated in the spreadsheets that were provided to DoH and the reviewer. The data is labelled and included in column B of all spreadsheets.

A table of baseline health incidence has been included in Appendix B of the report.

Issue 3: There are minor errors in the calculations in the spreadsheets provided. This is due to missing data and 365 days of data not available.

Response: The missing data is due to instrument issues during sampling. In most cases only 1 or 2 days of data was missing which does not change the overall outcomes of the HRA. It is however noted that there will be a minor underestimate of the annual outcomes due to missing data.

Issue 4: The argument that the relative toxicity of PM₁₀ and PM_{2.5} due to crustal particles is compared to other PM₁₀ and PM_{2.5} is moot given the information provided in the report given that further information is required to identify the relative contribution of crustal particles to total PM in Port Hedland.

Response: Although further information is required to quantify the relative contribution of crustal particles to total PM₁₀ in Port Hedland it is generally accepted that the main source of PM₁₀ is from crustal dust including iron ore dust from the Port operations. The discussion on the relative toxicity of crustal material to PM₁₀ from other sources is based on international reviews conducted by WHO (2013) and USEPA (2012) and is provided to provide evidence that the health effects of dust in Port Hedland cannot be ignored given that it is primarily crustal in nature. It is accepted that further analysis,

modelling and source apportionment studies are required to fully answer the question as to the relative importance of sources of PM in Port Hedland to the overall levels. This information was not available for the HRA.

Issue 5: The exclusion of Cr(III) and Cr(IV) sampling data due to analytical problems is not adequately discussed in the report. Additional discussion is required as Cr (III) and Cr (IV) are pollutants of concern and should be assessed in the HRA.

Response: Initially monitoring data was provided for inclusion in the HRA. Subsequently we were advised by DoH that the data could not be used as it was invalid due to the wrong filter media being used for the sampling. No further explanation was provided at that time or subsequently. Therefore no further discussion can be provided at this time. The HRA does recommend that sampling be conducted for Cr (III) and Cr (IV) and that the information be used in a future HRA.

Issue 6: The toxicity reference value for asbestos that is quoted in the TRVs is a soil guideline and is not appropriate for assessing health risks from air. A guideline in fibres/ml should be used.

Response: The DoH guideline (2009) has been used in the assessment. The original TRV in the HRAM was incorrect.

Issue 7: The HRAM discusses the use of air dispersion modelling in the HRA however it has not been included and there is no discussion of the future use of such data.

Response: The results of the air dispersion modelling were not available for the current HRA. The recommendation section of the report recommends that the HRA should be expanded to include such data once it is available. This issue has been discussed in Section 3 relating to the HRAM.

Issue 8: The HRAM discusses five exposed populations however not all have been addressed in the HRA. This needs to be discussed.

Response: This issue is discussed in Section 3 on the HRAM. Wedgefield was not included as it is an industrial area and contains no residential development. Although some industrial sites include worker accommodation no information was available on the population that might live there or the baseline health statistics available for the area. Therefore there was no information on which to calculate potential risks.

With respect to the Tjalkaboorda Aboriginal Community, no information was available on the population within the community or the baseline health status of that community. Although a separate assessment could not be undertaken, the risks calculated using the Taplin St and Neptune Place data will be representative of the risks likely to be experienced by this community.

Issue 9: A discussion of how each monitoring location relates to exposed populations needs to be included.

Response: This information has been included in Section 6.2 of the report.

Issue 10: The information in Table 6 (now Table 8) should be revised and include percentile concentrations in the summary table. A summary table for PM_{2.5} should be included. The number of exceedances of the guidelines should also be included.

Response: Table 8 has been updated to include the 99th, 95th and 90th percentile values for all locations as well as the number of exceedances for each location. The table for PM_{2.5} (Table 9) has been updated to include percentile values.

Issue 10: More context is required in relation to the Spoilbank data and how this data relates to population exposure.

Response: Further discussion of the Spoilbank data has been provided in Section 6.2. This data was provided late in the HRA process and has not been used in the risk characterisation. The data is not representative of the current population in Port Hedland. However, the area has been identified for potential development of a marina and residential development in the future.

Issue 11: More information is required in relation to the monitoring of metals in particular the analytical methods used. Further information is required on the monitoring locations and if they differ from the other monitoring locations.

Response: Information provided to the authors of the HRA has been included in Table 8. No information on the monitoring and analysis methods beyond what is contained in the HRA has been provided to the authors of the HRA. Only the resultant data was provided for the purpose of the HRA. As discussed in Section 7 of the HRA the metals monitoring was conducted at the PHIC monitoring locations. These locations are listed in the HRA in Section 7. The WA Chem Centre data is collected at the same sites.

Issue 12: There is no information on how the PM₁₀ data was adjusted for each of the scenarios assessed. PM₁₀ arises from a range of sources some of which can't be controlled such as sea salt and crustal material. It would be appropriate to truncate the data at the standard being assessed. It appears that all days exceeding the standard has been set to zero.

Response: A discussion on the approach taken to adjust has been included in Section 6.2.1. As discussed the entire dataset has been adjusted so that the peak values do not exceed the relevant standard/guideline. No peak data, or any data, has been adjusted to zero. Although sea salt cannot be controlled there is day-to-day variability in the contribution of sea salt to total PM₁₀ at each site due to meteorological conditions, in particular wind direction. No data is available at present to be able to determine what that contribution might be so it has been assumed that all sources contribute each day and have been included in the calculations.

Issue 13: Table of Contents does not make sense and needs to be revised. Table 15 should be labelled Table 14.

Response: These issues have been resolved. All table numbers have been automated and corrected where required.

Issue 14: Executive summary requires clarification re parameters meeting air quality standards. In particular the issue of Cr being excluded needs to be addressed.

Response: Clarification has been provided in the Executive Summary. All pollutants assessed are discussed and the exclusion of Cr included.

Issue 15: The values for increased daily mortality from cardiovascular causes at Taplin St in table 10 should be 0.7 and 0.6 rather than 1.

Response: The values were rounded to the nearest whole number to enable better understanding of the risks.

Peer Review 4: WA Department of Health

Issue 1: The HRA should explicitly comment on where the assessment has deviated from the HRAM, in particular the need for iterative consideration on the method as the dataset evolves.

Response: A section has been added in Section discussin the HRAM and where the HRA has deviated from it and why. In particular the use of dispersion modelling in the HRA to determine the risk from various sources could not be done as the results of the modelling were not available. It is recommended that further analysis of the health risks using the data be conducted once it is available.

Issue 2: The application of the NHMRC (2006) and NEPC (2011) methodologies for small communities should be discussed in particular any methodological challenges faced.

Response: The NEPC and NHMRC guidance has only been used to guide the systematic review of the literature to inform the hazard assessment. The approach used will not differ on the type of HRA being conducted. It is acknowledged that the approach to exposure assesement and risk characterisation will differ in the derivation of air quality standards compared to the assessment of risk within small communities. However this part of the NEPC methodology has not been applied. The NHMRC guidance only applies to hazard assessment.

Issue 3: The HRAM argues that sea salt may not be as harmful as other sources where the HRA argues that all PM is harmful regardless of source. More detail is required in the HRA and should critically argue the points raised in the HRAM.

Response: Further discussion on this point has been included in Section 3. The information provided in the HRA is based on the recent reviews conducted by WHO (2013) and USEPA (2012) which were not considered in the HRAM and represent the most current reviews on the health effects of PM.

Issue 4: Some context around the population as sensitive receptors is required including location of schools and other sensitive premises.

Response: This has been included in Section 5 of the HRA.

Issue 5: How much does dust from Spoilbank contribute to background PM or exceedances?

Response: The Spoilbank data was provided in mid December 2014. Not contextual data was provided in the report – only the daily PM₁₀ data. No data on wind speed or direction was provided. Without this information, the contribution of dust from Spoilbank at the other monitoring locations cannot be determined.

Issue 6: Background dust – how confident are the authors that background dust is not a major contributor to exceedances.

Response: The authors conducted an analysis of wind speed and direction on days of exceedances in the 2012 and 2013 datasets. An assessment was also conducted on the days where exceedances were observed at each of the monitoring locations. This analysis, which should be considered as a preliminary analysis, showed that on a number of days when exceedances were observed they were not observed at all monitoring sites which would be expected if regional dust was the cause. There were also days when exceedances were observed at the Taplin St and Richardson St sites but not at South Hedland even though the predominant wind direction was from the direction of South Hedland. This indicated that there was a sources or sources of dust between South Hedland and Port Hedland that was impacting on PM₁₀ levels in Por Hedland. It is recommended that a more detiled analysis be

conducted, including the results of the air dispersion modelling, to further explore the key sources of dust in Port Hedland.

Issue 7: The report requires substantial proof reading and editing. In particular the Executive summary is too brief and assumes too much contextual knowledge.

Response: The report has undergone proof reading and editing by a person not involved in the study with Pacific Environment. The Executive Summary has been revised.

Issue 8: Sections including Glossary of Terms, Overview of what was required, Purpose of the report and HRA objectives, Approach to HRA, Environmental Profile of Port Hedland, and the HRAM should be included.

Response: All these sections have been included in Sections 2 and 3 of the HRA. A Glossary has been added at the front of the report.

Issue 9: Appendices should be included showing baseline health statistics for the years used, spreadsheets for each of the calculations conducted, and sample calculations for each new calculation performed or calculation variation.

Response: Appendix B shows the baseline health incidence data and sample calculations. The information in the Appendix has been cross-referenced to the main report. The spreadsheets are provided as a CD to the Department.

Issue 10: The table of Contents should include a list of Tables, Figures and Appendices.

Response: These have been included.

Issue 11: The word 'significant' is used quite often without qualification whether it is statistically, substantive, causal or clinical.

Response: Clarification has been provided in the text in numerous locations in the report.

Issue 12: p45 refers to proposed mine.

Response: This has been corrected.

Issue 13: There are a large number of missing references.

Response: All missing references have been included.

Appendix B: **BASELINE HEALTH DATA**

B.1 BASELINE HEALTH DATA

Baseline health statistics were provided by the WA Department of Health, Epidemiology Branch. A summary of the data that was obtained and the search strategy is summarised below (as provided by DoH).

Data request overview:	Respiratory health statistics for residents of Port Hedland town, WA, 2008 - 2012	
Data sources:	WA Emergency Department Data Collection	
	WA Hospital Morbidity Data System	
	WA Death Registrations	
Inclusion criteria:	Residents of Port Hedland town were identified using a two-step process. Those with postcodes of 6721 and 6722 were first selected, after which those with SA1 that were not in the Port Hedland town (SA1 starting with the digits '50806') were removed. Those with postcodes 6721 and 6722 and missing SA1 were included in the analysis.	
	Death data is not reported for 2012 as the cause of death codes are only available up to 2011.	
Methods:	Major diagnostic category codes were used to identify Emergency Department presentations due to the following conditions:	
	Respiratory	4
	Cardiovascular	5
	ICD-10-AM diagnosis codes were used to identify hospitalisations due to the following conditions:	
	All Respiratory	J00-J99 (excluding J95.4 to J95.9), R09.1, R09.8
	Pneumonia and bronchitis	J12-J17, J18.0, J18.1, J18.8, J18.9, J20, J21
	COPD	J40-J44, J47, J67
	Asthma	J45, J46, J44.8
	All Cardiovascular	I00-I99 (excluding I67.3, I68.0, I88, I97.8, I97.9, I98), G45 (excluding G45.3), G46, M30, M31, R58
	Ischemic Heart Diseases	I20, I21, I22, I24, I25.2
	ICD-10-AM diagnosis codes were used to identify deaths due to the following conditions:	

	All Respiratory	J00-J99 (excluding J95.4 to J95.9), R09.1, R09.8
	Lung Cancer	C34
	All Non-accidental	A00-R99
	All Cardiovascular	I00-I99 (excluding I67.3, I68.0, I88, I97.8, I97.9, I98), G45 (excluding G45.3), G46, M30, M31, R58
	Data was requested to be broken down by selected areas which were defined as follow:	
	Port Hedland	Postcode: 6721
	South Hedland	Postcode: 6722
	Port Hedland - West End	SA1: 50806122215, 50806122209, 50806122212
	Port Hedland - East End	SA1: 50806122210, 50806122204, 50806122211, 50806122202, 50806122203, 50806122213, 50806122208, 50806122205, 50806122201, 50806122206, 50806122207, 50806122214
	It is noted that the total numbers in Port Hedland is more than the sum of numbers in West End and East End as these two latter areas are identified using SA1 which is unknown in a number of the cases.	

The baseline health statistics that were used in the HRA are summarised in the following Table. The statistics for 2011 were used as they were the most recent data available.

Health Outcome	Annual Incidence per 100,000 population (2011)	Annual Incidence per 100,000 population (2011)
	Port Hedland	South Hedland
Annual all cause (non-accidental) mortality all ages	196	127
Annual cardiovascular mortality all ages	88	29
Annual lung cancer mortality all ages	43	31
Annual cardiopulmonary mortality all ages	87	31
Annual Hospital Admissions for respiratory disease 65 + years	16,790	14,600
Annual Hospital Admissions for pneumonia and bronchitis 65 + years	16,790	14,600
Annual Hospital Admissions for respiratory disease 15-64 years	803	1241

Appendix C: SAMPLE CALCULATIONS

Sample calculations are provided for each health outcome assessed with one example for each scenario. The same equation has been used to calculate all attributable risks so not every outcome for every scenario is presented.

The attributable risk due to air pollution has been calculated using the following equation:

The increase in risk per 100,000 population due to PM, NO₂ and SO₂ has been calculated using the following equation:

$$\text{Increase in risk for each health outcome} = \frac{\text{exposure response function/unit increase in pollutant concentration} \times \text{pollutant concentration} \times \text{baseline incidence rate}}{100,000 \text{ population}}$$

To calculate the number of attributable cases the risk per 100,000 was multiplied by the actual population as a fraction of 100,000. The number of cases for each outcome was calculated for the population represented by each monitoring location. For short-term effects associated with daily changes in pollutant concentration the number of cases for each day of the year were calculated and then summed to give the annual total. For the assessment of long-term mortality, the annual average concentrations were used in the calculations.

SAMPLE CALCULATION 1: INCREASE IN MORTALITY DUE TO PM₁₀

For mortality the increase in risk has been calculated for both long-term and short-term exposures. For the long-term exposures annual average PM₁₀ levels were used. For the risk for Port Hedland using Richardson St data the annual average PM₁₀ level was 37.1 µg/m³. The exposure response function identified in Table 4 for all-cause mortality is 0.004 increase per 1 µg/m³ increase in PM₁₀. The baseline health incidence rate for all-cause mortality as shown in Appendix B is 196/100,000. Therefore the resultant increase in risk is:

$$\begin{aligned} \text{Increase in annual all-cause mortality} &= \frac{\text{exposure response function}/1 \text{ } \mu\text{g}/\text{m}^3 \text{ increase in PM}_{10} \times \text{annual average PM}_{10} \times \text{baseline incidence rate}}{100,000 \text{ population}} \\ &= 0.004 \times 37.1 \times 196 \\ &= 29/100,000 \end{aligned}$$

For daily incidence the daily PM₁₀ (24hour levels) were used. It should be noted that the daily values can vary significantly. As daily mortality rates were not available they were estimated by dividing the annual incidence by 365. This introduces some uncertainty in the assessment. As an example of the calculation on 16/1/2013 the 24 hour PM₁₀ value was 25 µg/m³. The exposure response function for daily all-cause mortality calculated from the annual incidence from Table 4 is 0.002 increase per 1 µg/m³ increase in PM₁₀. The baseline health incidence rate for daily all-cause mortality as shown in Appendix B is 0.54/100,000. Therefore the resultant increase in risk is:

$$\begin{aligned} \text{Increase in daily all-cause mortality} &= \frac{\text{exposure response function}/1 \text{ } \mu\text{g}/\text{m}^3 \text{ increase in PM}_{10} \times \text{daily average PM}_{10} \times \text{baseline incidence rate}}{100,000 \text{ population}} \\ &= 0.002 \times 25 \times 0.54 \\ &= 0.027/100,000 \end{aligned}$$

This calculation was repeated for each day of the year and then the increase in risk summed for each day to give an annual total due to short-term exposures. The annual increase is 12.9 per 100,000.

For the scenario where peak levels don't exceed 70 µg/m³ the PM₁₀ data for every day was reduced by the adjustment factor included in Table 10 of 69%. The resulting annual average concentration was 11.5 µg/m³. All other parameters remain the same as for the previous calculation.

Increase in annual all-cause mortality = exposure response function/1 µg/m³ increase in PM₁₀ x annual average PM₁₀ x baseline incidence rate/ 100,000 population

$$= 0.004 \times 11.5 \times 196$$

$$= 9/100,000$$

Using the same example as the previous calculation on 16/1/2013 the adjusted 24 hour PM₁₀ value was 7.8 µg/m³. With all other parameters remaining the same the resultant increase in risk is:

Increase in daily all-cause mortality = exposure response function/1 µg/m³ increase in PM₁₀ x daily average PM₁₀ x baseline incidence rate/ 100,000 population

$$= 0.002 \times 7.8 \times 0.54$$

$$= 0.008/100,000$$

This calculation was repeated for each day of the year and then the increase in risk summed for each day to give an annual total due to short-term exposures. The annual increase is 4 per 100,000.

The same approach was used to calculate the increase in risk for hospital admissions for each outcome assessed.

SAMPLE CALCULATION 2: MORTALITY OUTCOMES FOR PM_{2.5}

For mortality the increase in risk has been calculated for both long-term and short-term exposures. For the long-term exposures annual average PM_{2.5} levels were used. For the risk for Port Hedland using Richardson St data the annual average PM_{2.5} level was 7.1 µg/m³. The exposure response function identified in Table 5 for all-cause mortality is 0.006 increase per 1 µg/m³ increase in PM_{2.5}. The baseline health incidence rate for all-cause mortality as shown in Appendix B is 196/100,000. Therefore the resultant increase in risk is:

Increase in annual all-cause mortality = exposure response function/1 µg/m³ increase in PM_{2.5} x annual average PM_{2.5} x baseline incidence rate/ 100,000 population

$$= 0.006 \times 7.1 \times 196$$

$$= 8.3/100,000$$

For daily incidence the daily PM_{2.5} (24hour levels) were used. It should be noted that the daily values can vary significantly. As daily mortality rates were not available they were estimated by dividing the annual incidence by 365. This introduces some uncertainty in the assessment. As an example of the calculation on 16/1/2013 the 24 hour PM_{2.5} value was 4.8 µg/m³. The exposure response function for daily all-cause mortality calculated from the annual incidence from Table 5 is 0.0023 increase per 1 µg/m³ increase in PM_{2.5}. The baseline health incidence rate for daily all-cause mortality as shown in Appendix B is 0.54/100,000. Therefore the resultant increase in risk is:

Increase in daily all-cause mortality = exposure response function/1 µg/m³ increase in PM_{2.5} x daily average PM_{2.5} x baseline incidence rate/ 100,000 population

$$= 0.0023 \times 4.8 \times 0.54$$

$$= 0.006/100,000$$

This calculation was repeated for each day of the year for which data was available and then the increase in risk summed for each day to give an annual total due to short-term exposures. The annual increase is 2.7 per 100,000.

SAMPLE CALCULATION 3: HOSPITAL ADMISSIONS DUE TO NO₂

For the increase in risk due to NO₂ all exposure response functions used are tabulated in Table 19. For hospital admissions the increase is associated with the maximum daily 1-hour concentrations. It should be noted that the hourly maximum values can vary significantly. As daily hospital admission rates were not available they were estimated by dividing the annual incidence by 365. This introduces some uncertainty in the assessment. As an example of the calculation on 16/1/2013 the 1 hour maximum NO₂ value was 11.8 µg/m³. The exposure response function for daily hospital admissions for respiratory disease 15-64 years of age from Table 19 is 0.0002 increase per 1 µg/m³ increase in NO₂. The baseline health incidence rate for daily hospital admissions respiratory disease calculated from the annual incidence in Appendix B is 2.2/100,000. Therefore the resultant increase in risk is:

Increase in daily hospital admissions for respiratory disease 15-64 years = exposure response function/1 µg/m³ increase in NO₂ x daily 1-hour maximum NO₂ x baseline incidence rate/ 100,000 population

$$= 0.0002 \times 11.8 \times 2.2$$

$$= 0.005/100,000$$

This calculation was repeated for each day of the year and then the increase in risk summed for each day to give an annual total due to short-term exposures. The annual increase is 2.6 per 100,000.

SAMPLE CALCULATION 4: HAZARD INDEX FOR IRON OXIDE

The hazard index is calculated using the monitored concentrations at each location divided by the relevant TRV in Table 2.

For iron oxide the maximum 24-hour average concentration for Richardson St is 9.9 µg/m³. The 24-hour average TRV adopted for the HRA from Table 2 is 120 µg/m³. Therefore the resulting hazard index is:

$$\text{Hazard Index} = \text{concentration iron oxide/TRV}$$

$$= 9.9/120$$

$$= 0.08$$

The hazard indices for the other metal were calculated in the same way. For metals where the TRV adopted is an annual average, the annual average concentration was used.