

PORT HEDLAND WEST END

IMPROVEMENT SCHEME NO. 1

Scheme Report

Western Australian Planning Commission

(Version Updated March 2020)

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1.0 Introduction

1.1 Preamble

This Improvement Scheme Report (this Report) has been prepared on behalf of the Western Australian Planning Commission (WAPC) in support of the Port Hedland West End Improvement Scheme No. 1 (the Scheme). The Scheme is the statutory framework providing for land use planning administration and development control within the Scheme area (Figure 1).

Improvement plans and improvement schemes allow the State Government, through the WAPC to become the planning authority for a particular area of land with the intention to advance its planning and development. These planning instruments are established under Part 8 of the *Planning and Development Act 2005* (PD Act).

Improvement plans are strategic instruments used to facilitate the development of land in areas identified by the WAPC as requiring special planning. The WAPC can recommend to the Minister for Planning that an improvement plan is made “for the purpose of advancing the planning, development and use” of land.

An improvement plan comes into effect when it is published in the *Western Australian Government Gazette* (the *Gazette*).

Improvement schemes are similar instruments to local planning schemes, as they provide development control provisions relating to the area subject to an improvement plan. The WAPC generally prefers that improvement schemes conform with the Model and Deemed provisions for local planning schemes set out in Schedules 1 and 2 of the *Planning and Development (Local Planning Schemes) Regulations 2015* (LPS Regs), although the PD Act does provide for some flexibility in the format of improvement schemes.

The Minister for Planning is responsible for considering improvement schemes and can either approve or refuse a proposal, or request the WAPC to make modifications before resubmitting it for approval.

An improvement scheme comes into effect when it is published in the *Gazette*.

An outline of the improvement plan and improvement scheme preparation process and associated consultation requirements is included in Figure 2 and Figure 3.

1.2 Purpose of the Improvement Scheme Report

This Report sets out the WAPC’s objectives for future planning and development in the West End of Port Hedland (the West End), and includes a broad strategic framework by which to pursue those objectives. This report provides an outline of the strategic intentions for the West End, site opportunities and constraints and an overview of the statutory provisions of the (the Scheme).

Improvement Plan No. 50: Port Hedland West End was prepared to establish the Scheme objectives:

- to provide a strategic planning framework to determine future land uses, considering all land use options, that takes into consideration physical, economic, social, and environment factors;

- to provide a statutory planning instrument through which to implement the strategic planning framework and effectively guide the preparation of statutory plans, statutory referral documentation and policy (as may be required) to facilitate orderly and proper planning of the area; and
- to implement the State Government response to the *Port Hedland Dust Management Taskforce Report to Government* to prohibit sensitive land uses and restrict population growth in the West End of Port Hedland.

The above objectives are given effect through the statutory framework forming the Scheme described as follows:

- *Scheme text*: the range of statutory provisions necessary to effectively achieve the Scheme's objectives.
- *Scheme map*: spatial representation of zones and reserves.
- *Scheme policies and ancillary plans and documents*: articulate specific objectives and criteria for the exercise of discretion provided by the Scheme including development guidelines and control.

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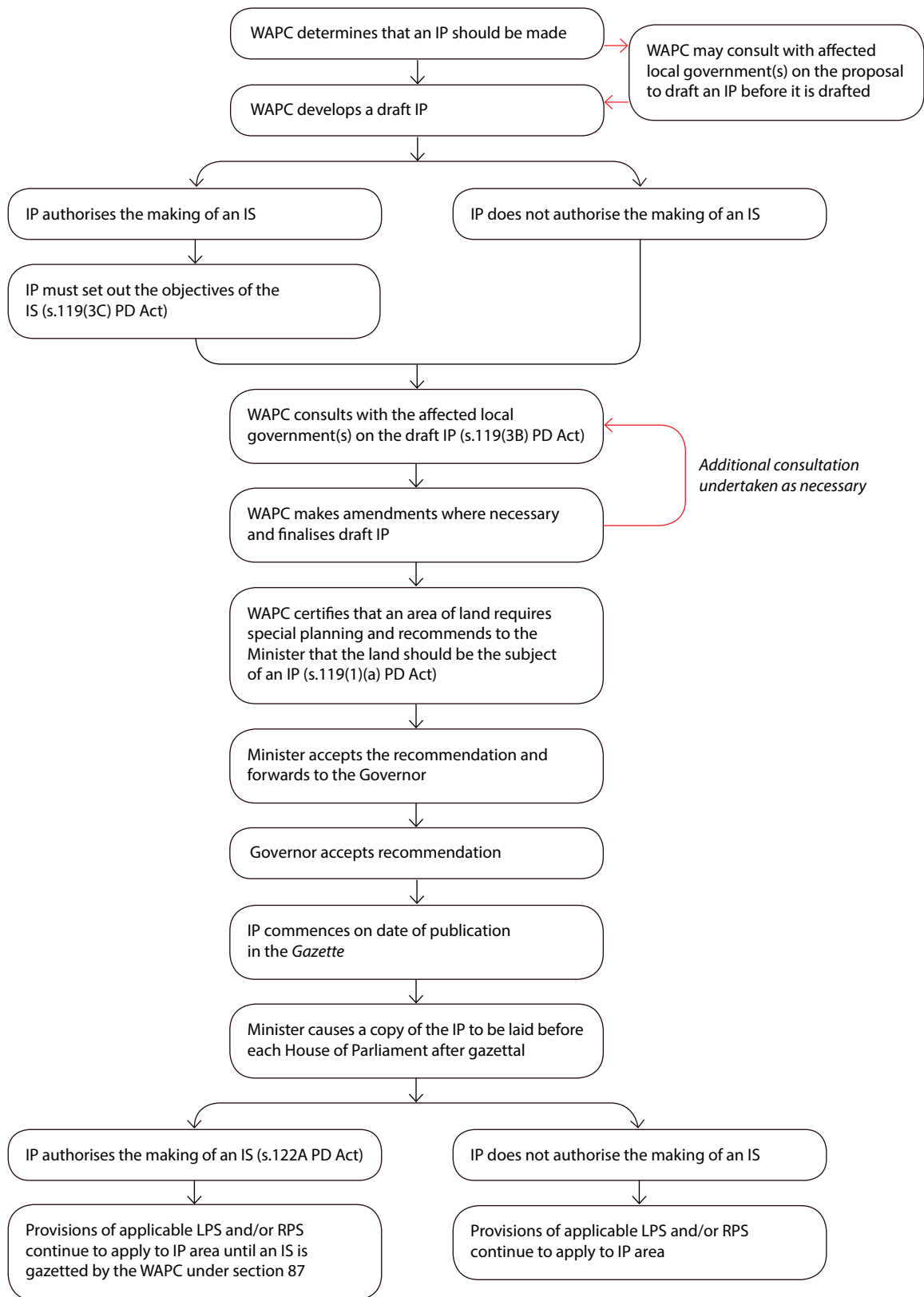


Figure 2: Improvement plan (IP) – statutory preparation

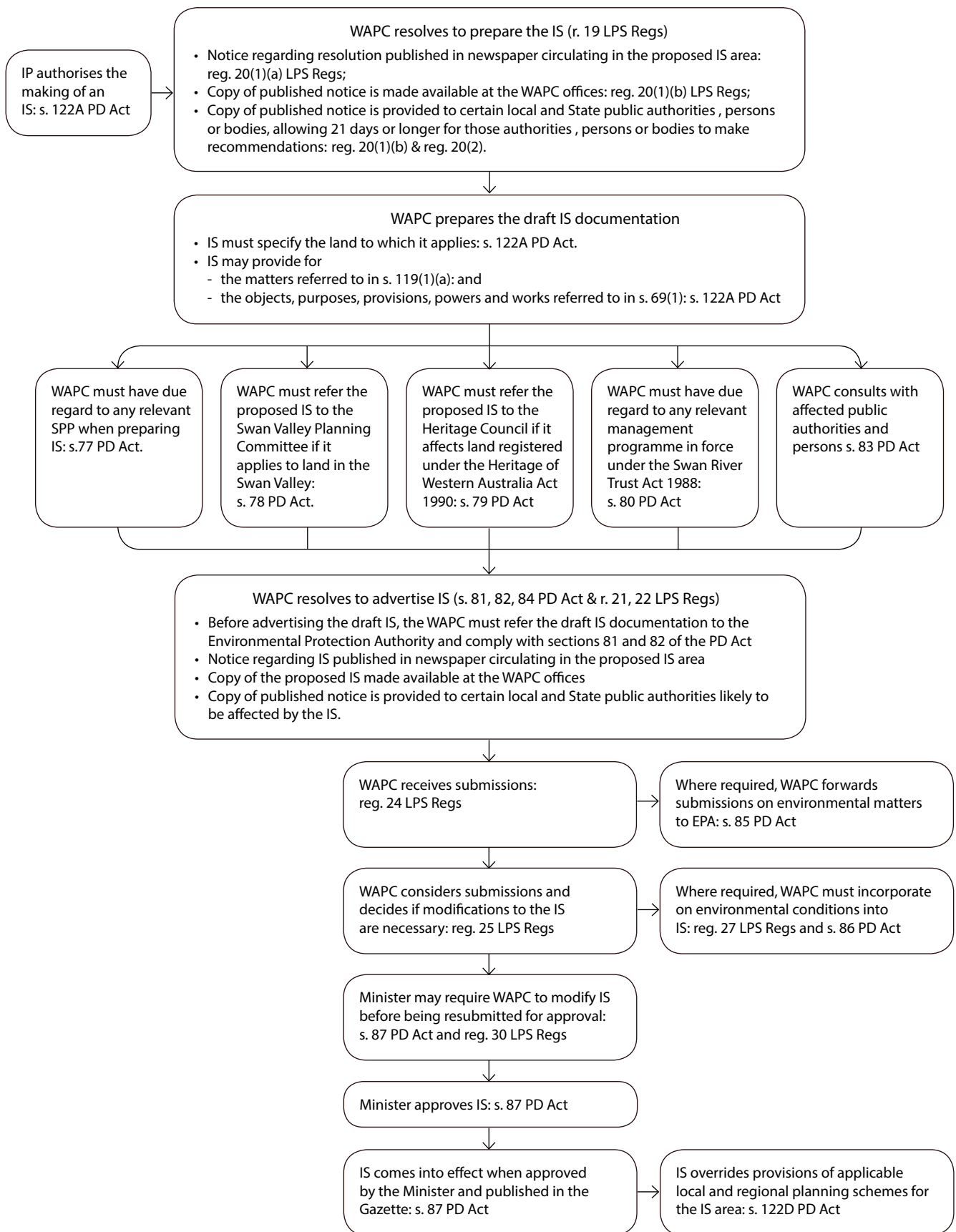


Figure 3: Improvement scheme (IS) – statutory preparation

1.3 Content and structure

This Report outlines the key issues and objectives for planning and development in the Scheme area, and provides the basis for planning provisions in the Scheme. This Report includes spatial plans, and sets out further details of the mechanisms by which the Report's objectives will be followed and the Scheme implemented.

1.4 Relationship to Town of Port Hedland local planning scheme

Section 122D of the *PD Act* provides that, once it comes into effect, an improvement scheme will replace any other planning scheme that applies to land in the improvement scheme area. As such, for the duration of the improvement scheme, the Town of Port Hedland local planning scheme will cease to apply to the land bound by the Scheme.

1.5 Community and stakeholder consultation

The *PD Act* and the LPS Regs outline specific mandatory consultation and notification requirements for the process of preparing improvement plans and improvement schemes, for example, the Minister for Planning must table a copy of the improvement plan before each House of Parliament (s.119 (5A) – *PD Act*) and the WAPC must:

- consult with affected local governments before making a recommendation on whether land should be made subject to an improvement plan (s.119 (3B) – *PD Act*);
- publish a notice of the acceptance of the recommendation of the Governor, and a summary of the improvement plan, in the *Gazette* (s.119 (4) – *PD Act*);
- publish a notice of a resolution to prepare an improvement scheme in the *Gazette* and provide a copy of the notice to bodies and persons affected by the improvement scheme (Reg. 20 – LPS Regs);
- forward a copy of a proposed improvement scheme to the Environmental Protection Authority (EPA) for assessment pursuant to s. 48 of the EP Act (s. 80 and s. 81 – *PD Act*);
- advertise a proposed improvement scheme for public inspection and consider all submissions (Part 4 Division 2 – LPS Regs);
- publish a notice of an improvement scheme in the *Gazette* (s. 87(3) – *PD Act*); and
- publish a notice of an improvement scheme in the *Gazette* and local newspaper, and provide a copy of the notice to each person who made a submission to the improvement scheme (Reg. 33 – LPS Regs).

1.6 Traditional Owners

This report recognises the Traditional Owners of Country and the Traditional Owner groups, including the Kariyarra people.

1.7 Technical documentation

This Report has been prepared having regard for a series of technical reports (appended). These reports and the matters addressed by each are summarised as follows:

- *Port Hedland Air Quality Health Risk Assessment for Particulate Matter* (Department of Health, 2016) – provides an independent health risk assessment of air quality in Port Hedland (Appendix 1)
- *Port Hedland Dust Management Taskforce Report to Government – August 2016* (2016 Taskforce Report): the 2016 Taskforce Report considered the findings of the Health Risk Assessment, and provided further recommendations on dust management in Port Hedland (Appendix 2).

Other reports referred to include:

- the *Port Hedland Air Quality and Noise Management Plan* (Department of State Development, 2010), which included interim measures to limit exposure to dust by groups considered most at risk of health effects
- the *Port Hedland Regional Land Supply Assessment* (WAPC, unpublished draft), which assessed the stock of land available for residential, industrial and commercial purposes, and identified key land use planning and infrastructure provision required to meet demand across the Town of Port Hedland.

2.0 Background and planning framework

2.1 Background

The Pilbara region contributes significantly to the State's economy, with iron ore being the largest export commodity. Although more than 50 mines are presently either operating, committed or under consideration in the Pilbara, private ownership of key infrastructure, combined with logistical and capacity limitations, serve to constrain the realisation of iron ore export potential.

The Pilbara region accounts for approximately 94 per cent of Australia's iron ore production. Iron ore constitutes approximately 99 per cent of Port Hedland's total export volume with other commodities including salt, manganese ore, chromite ore, copper concentrate, spodumene concentrate and cattle also exported from the port¹.

Port Hedland is home to the largest bulk export port in the world, with a total annual throughput of 513Mt in 2018-19². Annual shipping capacity in Port Hedland increased to 617Mt in 2019 due to investment in port dredging, marine technology and other port efficiencies³ cementing the port's supply chain as the core of the Port Hedland economy for the foreseeable future.

Historical urban growth in the West End (defined as west of Taplin Street) and port expansion has resulted in land use conflict. Port Hedland's West End is home to about 600 residents as well as numerous sensitive land uses. Being adjacent to the port, the West End is the most dust affected urban area in Port Hedland, with bulk commodity exports occurring to the south and west. Census figures indicate that 41 per cent of private dwellings in the area are unoccupied and 82 per cent of occupied private dwellings are rented.

The issue of air quality in the West End of Port Hedland, and in particular the potential impacts from dust on human health, has been of concern to Port Hedland residents, business owners, the Environmental Protection Authority (EPA) and State Government agencies for many years.

2.1.1 Port Hedland Dust Management Taskforce (2009)

The Port Hedland Dust Management Taskforce (the Taskforce) was established in 2009 to plan for and provide effective dust management strategies in Port Hedland, particularly for the West End locality. The Taskforce was chaired by the (former) Department of State Development and comprised representatives from relevant State Government departments (including Planning, Lands and Heritage), industry and the Town of Port Hedland.

The Taskforce prepared the *Port Hedland Air Quality and Noise Management Plan* (Department of State Development, 2010; 2010 Taskforce Plan), the recommendations of which were adopted by the former State Government in January 2010. The 2010 Taskforce Plan was a comprehensive management plan for ongoing air quality and noise management in Port Hedland including an implementation strategy and governance framework.

¹ Town of Port Hedland Strategic Community Plan 2018 - 2028

² Pilbara Ports Authority Annual Report 2018-19

³ Western Australia Iron Ore Profile November 2019: Department of Jobs, Tourism, Science and Innovation

In relation to land use planning the 2010 Taskforce Plan recommended a precautionary approach that residential development should occur predominantly in the eastern areas of Port Hedland. Subsequently, Amendment No. 22 (gazetted on 27 April 2012) to the Town of Port Hedland Local Planning Scheme No. 5 introduced provisions applying to existing residential areas in the West End, to promote housing redevelopment that discouraged long-term residency by those more at risk from exposure to dust (aged persons and families, for example).

The 2010 Taskforce Plan also recommended that further work be undertaken to establish the health impacts of the dust and acknowledged this may take three to five years.

During 2011 and 2012, the Town prepared its local planning strategy: the Pilbara's Port City Growth Plan which reflected the recommendations of the 2010 Taskforce Plan.

2.1.2 Health Risk Assessment (2016)

In February 2016, the Western Australian Department of Health published the *Port Hedland Air Quality Health Risk Assessment for Particulate Matter* report (refer Appendix 1), which concluded that there is sufficient evidence of possible negative effects on human health from dust in the West End of Port Hedland to warrant dust management controls and planning measures to reduce community exposure to dust.

2.1.3 Port Hedland Dust Management Taskforce Report to Government (2016)

During 2016, the Port Hedland Dust Management Taskforce considered the findings of the Department of Health's *Port Hedland Air Quality Health Risk Assessment for Particulate Matter* report and provided recommendations through the Port Hedland Dust Management Taskforce Report to Government August 2016 (the 2016 Taskforce Report).

In relation to land-use planning, the 2016 Taskforce Report (Appendix 2) recommended:

- an amendment to the Town of Port Hedland's local planning scheme to:
 - prohibit new permanent residential development and other sensitive land uses, including aged care and child care premises, west of Taplin Street; and
 - apply the land uses described in the Town of Port Hedland Local Planning Strategy's West End Precinct, taking into consideration the findings of the Health Risk Assessment.
- that low-density (R20) residential development be permitted in the predominantly residential area between Taplin Street and McGregor Street, but higher-density residential development and other sensitive land uses be prohibited.

2.1.4 State Government response to the Dust Management Taskforce Report (2018)

After considering the 2016 Taskforce Report, and the submissions received, on 15 October 2018 the State Government adopted the following land use planning position in relation to the management of dust in Port Hedland:

- The Government supports the Taskforce recommendation that appropriate planning controls be implemented to prohibit sensitive land uses and restrict population growth in the West End of Port Hedland. To give effect to this, the Western Australian Planning Commission will be requested to prepare an improvement plan and improvement scheme designed to achieve the land use outcomes described in Recommendation 5 of the Taskforce Report.
- In response to community concerns raised in the consultation process, the Government will act to ensure that any future West End planning controls will not prevent the redevelopment of residential properties – provided that the redevelopment would not result in an intensification or expansion of a non-conforming use should they be more than 75 per cent damaged by a natural disaster such as fire or a cyclone.

2.1.5 Improvement Plan No. 50: Port Hedland West End (2019)

The WAPC considered the State Government's position on the 2016 Taskforce Report and through the Minister for Planning and the Governor, established Improvement Plan No 50: Port Hedland West End (IP 50), gazetted on 30 July 2019.

IP 50 was prepared to advance the planning, development and use of land in the West End precinct of Port Hedland in response to the State Government's consideration of the 2016 Taskforce Report.

The area subject to IP 50 (Figure 1) comprises landholdings west of McGregor Street and Lukis Street and is located adjacent to the port of Port Hedland, where bulk commodity stockpiling and handling, as well as the bulk import and export of other materials such as fuel, occurs to the south and west. The West End precinct is the most dust, noise and hazard-affected urban area in Port Hedland.

The primary objectives of IP 50 are to provide a strategic and statutory land use framework, based on investigation and consideration of all development scenarios for the area under the improvement plan, taking into account the State Government's response to the recommendations of the 2016 Taskforce Report.

IP 50 includes the necessary provisions authorising the preparation of the Scheme with the key objective of reducing public exposure to dust.

2.1.6 Potential Port Hedland Voluntary Buy-Back Scheme

On 13 September 2019, the State Government announced the appointment of an independent consultant tasked with investigating the potential establishment of an industry-funded voluntary buy-back scheme. Following the completion of a three-month consultation process between September to November 2019, a report detailing recommendations on a potential buy-back scheme will be submitted to the State Government in early 2020.

The main objectives of a potential buy-back scheme would be to:

- provide an opportunity for residential property owners in the West End to exit their properties at reasonable values prior to restrictions on land use that will arise from planning permissibility changes;
- reduce regulatory risk to the operations and future expansion of iron ore exports through the port of Port Hedland; and
- stimulate new land uses in the West End which will increase the amenity and viability of the West End.

2.2 Current local planning framework

Preparation of the Scheme over the West End precinct has considered the strategic guidance and statutory development controls currently provided through the Town of Port Hedland's local planning strategy and local planning scheme.

2.2.1 Town of Port Hedland Local Planning Scheme

The Town's current Local Planning Scheme No. 5 (TPS 5) was gazetted on 31 August 2001, and covers a larger portion of the local government area surrounding settlements on the coast, where the majority of the Town's population is located.

Prior to the Department of Health's risk assessment being completed, the 2010 Taskforce Plan recommended that a precautionary approach to residential development in the West End precinct should be taken in the short-term, to encourage redevelopment or retrofitting of existing residential premises in such a way as to mitigate dust and noise, while discouraging occupancy by 'at-risk' individuals (that is, those with clinical respiratory and cardiovascular disease, the elderly, babies and young children).

The Taskforce recommended the progression of an amendment to TPS 5 (Amendment No. 22, gazetted 27 April 2012) to establish the 'West End Residential' zone over existing residential land. The EPA set a formal level of assessment for Amendment 22. As a result of the Environmental Review process and to reduce human exposure to dust, the Minister for Environment required a number of environmental conditions to be incorporated into the amendment to prescribe specific building design and performance standards for development in the zone. The Taskforce considered the 'West End Residential' zone's residential density code range from a minimum of R30 to a maximum of R80 (applied through Amendment 22) was appropriate to provide an incentive for redevelopment or retrofitting of existing housing stock to mitigate dust/noise and discourage occupancy by families and the elderly.

The development control provisions incorporated under Amendment 22 apply to a relatively small proportion of the West End. Currently there are no statutory provisions in TPS 5 that require residential development in the majority of the West End to conform to building design and performance standards to reduce human exposure to dust and noise.

2.2.2 Town of Port Hedland Local Planning Strategy

The Town of Port Hedland's local planning strategy, the 'Pilbara's Port City Growth Plan' was endorsed by the WAPC in 2012 following comprehensive community and stakeholder consultation during its preparation over the period from 2010 to 2012.

Consistent with the recommendations of the 2010 Taskforce Plan, the Town's local planning strategy promotes future residential development in the eastern precinct of Port Hedland (Pretty Pool and Cooke Point), and recommends the progressive transition from permanent residential uses under the current 'West End Residential' zone towards a longer-term land use scenario with no permanent residential uses (short-stay accommodation only) west of Acton Street.

To accommodate future population growth, the local planning strategy also identifies land in Port Hedland and South Hedland suitable for development, with the potential to achieve an estimated 23,000 residential dwellings over the coming decade. The strategy attributes less than 3 per cent of those new dwellings to the West End Residential zone between Acton Street and Taplin Street, while promoting over 5,500 new dwellings in Port Hedland's eastern precinct.

3.0 Key issues and strategic objectives

The Scheme will address a number of planning matters that arise out of the State Government's response to the 2016 Taskforce Report, and takes into consideration findings and recommendations of the Department of Health's *Port Hedland Air Quality Health Risk Assessment for Particulate Matter* (2016).

3.1 Sensitive land uses

Port Hedland is home to the largest bulk export port in the world. Total throughput increased from 247 million tonnes in 2011/12 to 519 million tonnes in 2017/18. The Port Hedland Industries Council forecasts volume to increase to 700 million tonnes by 2027.

With port operations predominantly consisting of bulk iron ore stockpiling and handling, the expansion of the port has seen a commensurate increase in airborne dust emissions, creating a conflict in land use for Port Hedland. As at the 2016 Census, there were approximately 600 residents living in Port Hedland's West End (west of Taplin Street).

A Department of Health assessment of the risks of exposure to dust emissions in Port Hedland found that:

- exposure to coarse dust at levels of up to 70 mgpcm (measured as an average over any 24-hour period) generates an acceptable health risk, equivalent to health risks associated with the National Environment Protection (Ambient Air Quality) Measure (NEPM) standard of 50 mgpcm for coarser airborne particles, noting that some individuals with particular susceptibility to respiratory diseases have higher risks of negative health outcomes at lower levels, as they would in any other part of Australia;
- fine dust particles are assessed to be within the NEPM and unlikely to increase; and
- where dust levels are likely to exceed this level on a regular basis, long-term exposure based on permanent residence presents an unacceptable community health risk generally, and particularly to sensitive receptors such as young children and the elderly.

Existing development in the West End provides for occupation by groups within the general population who are more vulnerable to the effects of air pollution⁴, such as:

- older people (greater than 65 years old)
- children
- people with pre-existing cardiovascular or respiratory disease
- children and adults with pre-existing respiratory conditions (asthma, bronchitis, and chronic obstructive pulmonary disease).

⁴ *Port Hedland Air Quality Health Risk Assessment for Particulate Matter: Department of Health, 2016*

The Scheme will give effect to the State Government response to the 2016 Taskforce Report by prohibiting:

- any form of residential development which provides for permanent occupancy (for example, single, grouped and multiple dwellings, residential aged care facilities)
- other land uses deemed to be sensitive receptors for human health impacts from dust (for example: aged persons centres, child care premises, schools, medical centres).

Daily activities associated with regular employment, short-stay accommodation, and other commercial or leisure activities, including those of tourists and other visitors in dust affected areas, are not assessed to present an unacceptable health risk other than for particularly sensitive individuals.

It is noted that the health risk assessment may change in the future, based on the further development of medical scientific understanding of the health effects of ambient air quality, particularly with respect to dust particles both fine and coarse, but also any other emissions, associated with the transportation, storage and handling of iron ore.

Strategic objectives

- Facilitate implementation of the State Government response to the Port Hedland Dust Management Taskforce Report to Government, through clearly defining sensitive land uses in the context of Port Hedland's West End.
- Prevent further land use conflict within the West End, considering all land use options that take into consideration physical, economic, social, and environmental factors.
- Consider and plan for land use transitions that may cause temporary land use conflict with non-conforming land uses.

3.2 Port operations

As the world's largest bulk minerals export port, the Port of Port Hedland is a major driver that underpins local, regional, state and national economic growth and prosperity.

According to the 2016 Census, iron ore mining employed 1,522 people in the Town of Port Hedland local government area. Employment growth will not only be experienced as a direct result of trade growth, but in creating the infrastructure and support services network to support industrial activities.

The West End presently facilitates general industrial land uses and the supply of essential services associated with Port of Port Hedland operations. Development within the West End provides for a range of industries of regional, state, national and global economic significance, and supply chain infrastructure that supports the operation of the port and industry. For example, uses in this precinct include marine services and logistics, manufacturing and fabrication, warehouses, wholesale trade, transport services, distribution centres and associated storage and waste management facilities.

The West End also provides infrastructure required for the daily operation of the port including security, customs and quarantine requirements, parking facilities, utility installations, and materials transportation infrastructure to support industry.

The Pilbara Ports Authority has identified the construction of additional cargo berths and layby areas in Port Hedland as a priority. These upgrades will allow for direct maritime freight services, which are a key enabler for reducing the cost of doing business in the region. Port upgrades are also necessary to accommodate the imports and exports of emerging industries⁵.

There is a need to provide the community and industry with greater certainty about the future of Port Hedland port and surrounding areas. Property and business owners in the West End remain uncertain about the future of their assets and investments, while the broader community faces uncertainty about the future of the West End as a potential tourism and recreational area.

Dust remains a major development challenge. The *Port Hedland Port Authority Port Development Plan 2012-2016* (Pilbara Ports Authority) identifies air quality and dust as a major environmental issue affecting the future growth of the port. Similarly, port users may find it increasingly difficult to expand iron ore exports through the port in the absence of planning controls to separate conflicting or incompatible land uses.

Strategic objectives

- Facilitate industrial land uses and services within the West End that are associated with port of Port Hedland operations.
- Support the safe and efficient operation and management of the port of Port Hedland, and its associated infrastructure.
- Provide for other development that does not compromise current and future expansion of port operations, port-related industry and supply chain infrastructure.
- Provide for a broad range of industrial, service and storage activities which, by the nature of their operations, should be isolated from residential and other sensitive land uses.
- Accommodate industry that would not otherwise comply with the performance standards of light industry.
- Manage impacts such as noise, dust and odour within the West End zone.
- Create an environment that is conducive to attracting and retaining skilled workers.
- Provide opportunities for the expansion of local workforce training schemes and facilities.
- Increase local workforce participation, in particular for the Aboriginal population.
- Prioritise planning for port-related infrastructure to support the growth and diversification of the regional economy.

⁵ *Pilbara Economic Development Consultation Paper* (Department of Jobs, Tourism, Science and Innovation, 2019)

3.3 Commercial and tourism activation

The existing West End commercial area – generally bound by The Esplanade, Richardson Street, McKay Street and Wilson Street – provides goods and services associated with the port while offering retail, entertainment and cultural experiences for Port Hedland residents and visitors.

Plans are progressing for development of a new marina on the west side of the Spoilbank which will add a significant attraction to the area. In addition, significant development opportunities also exist for ocean-front short-term accommodation development supported by strong connections to the proposed marina, recreation areas of the Spoilbank and the Port Hedland town centre. This may potentially include a hotel, restaurants, various holiday accommodation, and cultural interpretive facilities, all lining a waterfront promenade. In addition, there may be mixed retail and office uses with good visibility from the Anderson Street entry, and supporting commercial development centrally located.

There is demand for a viable commercial centre in the West End which leverages its positioning to service port operations (including maritime, training and education and research activities), as well as the development of tourist, cultural, and recreational attractions in the proposed Spoilbank Marina precinct.

Although the cost of operating a commercial business has, on average, declined in Pilbara towns since 2013, the absolute level of costs remains one of the highest in Australia, with significantly higher wages, rents and other operating expenditures. The high cost structure in the Pilbara, combined with difficulties associated with the availability of appropriate staff and commercial, retail and industrial land and floorspace, will continue to be a challenge for business growth in the Town and the Pilbara region⁶.

A diversified local economy will be an important measure to soften the shocks of resource cycles, and to build a more resilient local business community. Ongoing investment in established and emerging industries provides opportunities for economic development and diversification in the Pilbara.

Continuing to attract skilled workers to the Pilbara is central to attracting investment in the region, and to avoid potential skilled labour shortages. This in turn will help reduce business costs and make local industry more competitive, thereby supporting economic diversification⁷.

The West End also provides opportunities for the temporary accommodation of workforces linked to the establishment of local businesses and infrastructure projects in the region. Land to be used for this purpose should be identified, with the potential for this type of development to be later converted for tourism purposes or short-term accommodation for employees.

A related issue is the need to provide short-term accommodation for people visiting Port Hedland from remote areas to access government support services.

⁶ *Pilbara Regional Investment Blueprint (Pilbara Development Commission, 2015)*

⁷ *Pilbara Economic Development Consultation Paper (Department of Jobs, Tourism, Science and Innovation, 2019)*

Strategic objectives

- Facilitate commercial development that provides services and activities to support the ongoing operation and growth of the Port of Port Hedland.
- Provide for a 'Tourist Esplanade': a vibrant short-term accommodation node, with supporting mixed-use facilities (where it does not limit the ability of the port's key industries or associated infrastructure to achieve their ultimate capacity and operating efficiency).
- Facilitate short-term tourist accommodation located to the north of Kingsmill Street and Moore Street, to maximize on coastal views, and northerly site orientation.
- Provide for the development of a mix of varied but compatible land uses such as short-term accommodation, offices, showrooms, hospitality establishments and recreational and cultural facilities that enhance the amenity of the West End.
- Support commercial development central to this precinct and close to the Spoilbank Marina.
- Promote and provide for tourism opportunities.
- Provide for a variety of holiday accommodation styles, including retail and service facilities, where those facilities are provided in support of the tourist accommodation and are of an appropriate scale where they will not impact detrimentally on the surrounding or wider area.
- Encourage the location of tourist facilities so that they may benefit from existing road services, physical service infrastructure, other tourist attractions, natural and cultural features, and urban facilities.

3.4 Environmental management

Coastal hazards

Low lying and coastal areas of the Port Hedland townsite are subject to erosion and storm surge inundation hazards, due to its urban form and location on a narrow headland.

The Town of Port Hedland has prepared a Coastal Hazard Risk Management and Adaptation Plan (CHRMAP) (2019) which identifies and considers coastal hazards and risks for the Port Hedland townsite. The CHRMAP identifies areas that are particularly vulnerable to coastal erosion and flooding over the next 100 years.

Residential properties and public infrastructure (beaches, roads and playgrounds) within the foreshore bounding the Scheme area to the north are vulnerable to erosion. The West End is also shown to be at high risk of coastal inundation for so-called 'one-in-100-year events'. The CHRMAP report states a detailed storm-water drainage assessment is required as "*protection against inundation is not considered viable*". Therefore, the key item for management of the issue appears to be improvement of direct drainage connection to the ocean.

The current foreshore reserve in Port Hedland is insufficient in width to maintain the social and environmental functions of the reserve once erosion risk has been taken into account,⁸ and potential adaptation measures will impact on land use outside the current foreshore reserve, including portions of the Scheme area.

Flora and fauna

The Town of Port Hedland is known to contain many significant flora, vegetation, and fauna values that are identified under both State and Federal legislation and policy. The Port Hedland coastline provides vital habitat for the flatback sea turtle, which is a threatened species. Cemetery Beach (located north of the Scheme area) and Pretty Pool Beach are key nesting sites. The turtle-nesting season is a significant environmental and tourism event.

Lighting from coastal development can disrupt the behaviour of nesting adult turtles and hatchlings, as well as other animals. Avoidance and management of light impacts on coastal and marine fauna can be applied using a risk-based approach and by applying best practice methods to development.

Bushfire hazard

The majority of the Town of Port Hedland is declared a 'bushfire prone area' under the *Map of Bush Fire Prone Areas* prepared by the Office of Bushfire Risk Management (OBRM 2019), including around areas of existing urban development within the localities of Port Hedland and South Hedland. In accordance with State Planning Policy 3.7 Planning in Bushfire Prone Areas and the Guidelines for Planning in Bushfire Prone Areas Version 1.3, the identification of a site within an area declared as bushfire prone necessitates that a further assessment of the determined bushfire risk affecting the site is carried out as part of detailed planning and design.

None of the identified key environmental factors alone present a significant environmental impact which would preclude development within the Scheme area. Notwithstanding, a responsible environmental management approach is required to satisfactorily address these environmental factors.

Strategic objectives

- Ensure that development and the location of coastal facilities takes into account coastal processes, landform stability, coastal hazards, climate change and biophysical criteria.
- Ensure the identification of appropriate areas for the sustainable use of the coast, including for housing, tourism, recreation, ocean access, maritime industry, and commercial activities.
- Provide for public coastal foreshore reserves and access to them on the coast.
- Protect, conserve and enhance coastal zone values, particularly in areas of landscape, biodiversity and ecosystem integrity, and cultural significance.
- Protection of known turtle nesting areas within the Port Hedland townsite area.

⁸ Port Hedland Coastal Hazard Risk Management and Adaptation Plan (GHD, 2019)

3.5 Cultural and historic heritage

The Scheme recognises areas where Traditional Owners may seek access to undertake customary practices and traditional uses. Other land uses may be considered including those that would assist in the economic development of the region and may provide for structures and/or activities associated with traditional Aboriginal law and culture, and resource development.

The *Aboriginal Heritage Act 1972* provides for the identification and protection of Aboriginal heritage sites throughout Western Australia. There are three registered Aboriginal heritage sites in the West End, particularly in areas that have been disturbed through urban activity.

European settlement in the Port Hedland region has a history of only about 150 years, dating from the first pastoral leases established on the De Grey River in the 1860s. Most of the remnants of this early European settlement history are concentrated in the West End, which was virtually the extent of the town until the 1960s.

The West End contains four places on the Heritage Council of Western Australia's Register of Heritage Places:

- Dalgety House
- St Mathews Church site
- (Former) Medical Officer's Quarters (now Dome Coffee)
- (Former) Lock Hospital site.

Additionally, there are various landholdings within the West End included in the Town of Port Hedland Municipal Inventory of Heritage Places, that are of considerable cultural heritage significance to the community and worthy of recognition and protection through provisions of the Scheme.

Strategic objectives

- Development that recognises that Traditional Owners maintain a cultural and strategic interest in Port Hedland and their economic, cultural, and land use aspirations are acknowledged.
- Development appropriately recognises the importance of, and opportunities associated with, sites registered on the WA Heritage Council database, in addition to sites identified in the Town of Port Hedland's Municipal Inventory of Heritage Places.

4.0 Port Hedland West End Improvement Scheme No. 1

4.1 Improvement Scheme arrangements

The Scheme depicts the intended land use and development scenario arising out of the State Government's response to the 2016 Taskforce Report, and extensive engagement with key stakeholders from State and local government, community and industry.

Improvement schemes are not bound to reflect the Model and Deemed provisions set out in the LPS Regulations, as sections 256, 257A and 257B of the PD Act do not apply. This Scheme has been prepared, however, to reflect the Model and Deemed provisions as relevant to the requirements of the West End.

4.2 Land use

4.2.1 Sensitive land uses

Key to implementing the State Government's response to the 2016 Taskforce Report, the Scheme provides a definition of 'sensitive land use' in accordance with the findings of the Department of Health's *Port Hedland Air Quality Health Risk Assessment for Particulate Matter*, which noted that certain groups within the general population have been determined to be more vulnerable to the effects of air pollution. These include:

- older people (over 65 years)
- people with pre-existing cardiovascular or respiratory disease
- people with pre-existing respiratory conditions (asthma, bronchitis, chronic obstructive pulmonary disease)
- children.⁹

The Scheme provides aims, and West End zone objectives, to give effect to the State Government response to the 2016 Taskforce Report that sensitive land uses be prohibited, and population growth restricted, in the West End.

4.2.2 'West End' zone

The Scheme sets out a singular West End zone across all land west of Taplin Street, excluding the Spoilbank and foreshore areas. The West End zone will apply the State Government response to the 2016 Taskforce Report within the Scheme by prohibiting:

- any form of residential development which provides for permanent occupancy (for example: single, grouped and multiple dwellings, residential aged care facilities)
- other land uses deemed to be sensitive receptors for human health impacts from dust (for example: aged persons centres, child care premises, schools, medical centres).

⁹ *Health Risk Assessment Port Hedland* (Toxicos, 2015)

The provision of predominantly discretionary land use permissibility within the zoning table is intended to provide the WAPC with the ability to properly consider proposals for non-sensitive land uses on their merits against the objectives of the zone and of the aims and purposes of the Scheme (among other matters). Additionally, it is considered that there is general merit and consistency with the principles of orderly and proper planning in establishing appropriate uses as discretionary in order to provide for planning and investment flexibility into the future, without the need for future amendments to the Scheme.

The approach to populating the permissible uses within the Scheme zoning table is specific to the West End context, having regard to the findings of the Department of Health's *Port Hedland Air Quality Health Risk Assessment for Particulate Matter* and the State Government's response to the 2016 Taskforce Report.

The Scheme includes a 'use not listed' clause in respect of uses that do not fall within the use classes outlined within the zoning table. Given the evolving nature of the resources sector and technological advancements it is considered appropriate to incorporate such a clause into this Scheme in order to 'future-proof' it and allow flexibility within the West End. The clause requires that the WAPC determine that the use is consistent with the objectives of the zone prior to approval, providing a framework for the exercise of discretion.

4.2.3 Non-conforming uses

The Scheme applies the model non-conforming use provisions as detailed in Schedule 1 of the LPS Regs.

Provisions have been incorporated into the Scheme to reflect the State Government's commitment to ensuring that any future West End planning controls will not prevent the redevelopment of existing residential properties – provided that the redevelopment would not result in an intensification or expansion of a non-conforming use should they be more than 75 per cent damaged by a natural disaster such as fire or cyclone.

4.2.4 'Landscape Interface' reserve

A 'Landscape Interface' reserve is provided within the Scheme for the public open space corridor along Anderson Street to interface with adjacent industrial land uses, south of the Scheme boundary. The reserve will also continue to serve townsite drainage functions.

4.2.5 Roads

The Scheme identifies primary distributor, local distributor and local road reserves to distinguish the main heavy vehicle route along Wilson Street accessing the port from local traffic routes in the West End.

Further to the road classifications detailed in the Scheme, there is potential for future upgrades to the intersection, and realignment of Anderson Street and Short Street. Aimed at creating a unique sense of arrival to the West End, while focusing heavy vehicle traffic along Wilson Street; and with only limited access along Anderson Street, it will minimise conflicts between visitor traffic and industrial traffic.

4.3 Implementation of Improvement Scheme controls

The Scheme represents a staged, iterative approach to applying the State Government response to the 2016 Taskforce Report.

4.3.1 Land use planning changes to the area between Taplin Street and McGregor Street

Contemporary dust monitoring data indicate that Taplin Street, the eastern boundary of the Scheme area, remains the most appropriate boundary for limiting residential and sensitive land uses in proximity of the port.

However, the 2016 Taskforce Report supported the extension of the development controls to cap residential densities and limit population growth eastwards to McGregor Street, as a precautionary measure, allowing for possible increases in dust levels associated with future port expansion. Within the Taplin Street to McGregor Street precinct, the Taskforce supported low-density (R20) residential development, but higher-density residential development and other sensitive land uses (for example, aged persons centres, child care premises, schools, medical centres) will be prohibited.

The Department of Water and Environmental Regulation (DWER) has proposed a nominal five-year timeframe for the implementation of the State Government's industry regulation response to coordinate dust management in the West End (the Port Hedland Dust Management Program <https://www.der.wa.gov.au/our-work/community-updates/435-port-hedland>). At the completion of the five years, DWER will report on the final outcomes, along with any recommendations for required future investigations, and additional actions such as the extension of development controls east of Taplin Street should the regulatory approach not fully meet its aims of reducing the residential population's exposure to dust.

To accommodate this potential uncertainty over the outcome of the State Government's industry regulation response to dust in the West End, the Scheme is initially applied to cover only those landholdings west of Taplin Street.

4.3.2 Environmental assessment of the proposed Spoilbank Marina development

The State Government's proposed Spoilbank Marina development project intends to provide public access to the waterfront and the harbour (including boat ramps, marina boat pens, public open space and community facilities) where it does not compromise public safety or the security of port operations, or result in adverse impacts on environmental values.

Development opportunities adjacent to the proposed marine within the Scheme area includes marine support industries, coastal rescue services, tourism, commercial, light industry, educational facilities, public open space and public access to the waterfront and marina.

The Spoilbank Marina project is progressing to detailed design stage, with the necessary information to support an application to the Environmental Protection Authority (EPA) for consideration to the proposed development works being prepared.

The timeframe for the formal development proposal to be submitted to the EPA could be impacted by changes to the marina design and dredged basin layout. This may in turn impact on the EPA's consideration of an improvement scheme which proposes to apply development controls that would facilitate the proposed Spoilbank Marina development.

To accommodate this potential uncertainty over the outcome of the EPA's consideration of the Spoilbank Marina development, the Scheme will initially be prepared to exclude the proposed Spoilbank Marina development and landside activation areas. Once the marina has been constructed, if appropriate, the Scheme may be amended to extend over this area.

4.3.3 Subdivision and development proposals

Development, including construction work and carrying out of activities, will require the approval of the WAPC, the responsible authority administering the Scheme. The WAPC, supported by the Department of Planning, Lands and Heritage, will receive, assess and determine applications for development.

4.3.4 Improvement Scheme policies and ancillary matters

It is intended that development controls within the Scheme will be guided by structure planning, or Scheme planning policies that supplement the Scheme through generally accommodating the present spatial arrangement of uses and precincts within the West End, including:

- retention of the existing town (commercial – retail) centre and general industry areas;
- expansion of tourism uses adjacent to the coast to take advantage of coastal views and the proposed Spoilbank Marina;
- expansion of the central commercial area to provide additional land supply for service commercial uses associated with the port; and
- rationalisation and reservation of various landholdings to form a vegetation corridor separating industrial uses south of Anderson Street from expanded tourism uses to the north.

Ancillary planning documents ensure planning decisions consider the locational constraints of land uses, the significant investments represented, and the current and future benefits and costs to communities. The broad aims of these documents are to:

- implement the State Government response to the 2016 Taskforce Report;
- interpret the permissibility of land uses, including incidental or ancillary uses, in accordance with the aims of objects of the Scheme; and
- place no constraints on the operation of the port of Port Hedland.

Development proponents within the West End will be expected to undertake due diligence in consultation with the Department of Planning, Lands and Heritage to ascertain the specific requirements that will apply to their development proposal, which are likely to vary depending on the nature of the development and its spatial response to site constraints.

The realisation of land use synergies within the West End is dependent on the type and location of development, as determined by proponents' own independent feasibility studies, which may or may not be in line with structure planning or improvement scheme planning policies. Such documents are therefore ancillary to the provisions of the Scheme, intended to provide a broad framework and be flexible in nature, enabling applications to be considered on merits by the WAPC, having regard to the advice of relevant authorities.

5.0 Administration and review

The facilitation of development within the Scheme area is governed through the statutory administration and approvals processes.

5.1 Administration

The WAPC is the authority responsible for implementing the Scheme and any improvement scheme policies that are prepared. The WAPC will ensure that the statutory planning framework aligns with both State and local government broader strategic objectives for Port Hedland's West End.

Decisions made within the Scheme area will have regard for inter-relationships with the administration of the port by Pilbara Ports Authority under the *Port Authorities Act 1999*, as well as development control for the balance of the Port Hedland townsite under the Town of Port Hedland's local planning scheme.

5.2 Review

Periodic review of the operation of the Scheme will provide the opportunity to assess the effectiveness of the Scheme in implementing the State Government's response to the 2016 Taskforce Report. It also allows assessment of the efficiency of the provisions from an operational point of view.

The PD Act requires improvement schemes to be reviewed after each five-year period of operation. This ensures they remain current, and are appropriately serving strategic objectives. The outcomes of a Scheme review may lead to three possible scenarios:

- (1) The Scheme is adequately serving the strategic objectives of the project and no changes are required;
- (2) With appropriate amendments to reflect emerging industry needs, the Scheme will continue to meet the strategic objectives; or
- (3) The circumstances associated with the Scheme area have changed significantly, requiring amendments to the Scheme.

Each of the potential review outcomes requires referral to the Minister for Planning for determination.

List of abbreviations

AEP	Annual Exceedance Probability (flooding)
AHD	Australian Height Datum (sea level)
ARI	Average recurrence interval (flooding)
ASS	Acid Sulfate Soils
DOH	Department of Health
DMP	Department of Mines, Industry Regulation and Safety
DPLH	Department of Planning, Lands and Heritage
DWMS	District Water Management Scheme
EAG	Environmental Assessment Guidelines
EAR	Environmental Assessment Report
EPA	Environmental Protection Authority
EP Act	<i>Environmental Protection Act 1986</i>
EPBC Act	<i>Environmental Protection and Biodiversity Conservation Act 1999</i>
GIA	General Industry Area
HIA	Heavy Industry Area
ILUA	Indigenous Land Use Agreement
IP 50	Improvement Plan No. 50: Port Hedland West End
JTSI	Department of Jobs, Technology, Science and Innovation
LA Act	<i>Land Administration Act 1997</i>
MCC	Metallurgical Company of China Australia Sanjin Mining Pty Ltd
MDP	Model and Deemed Provisions
Mgpcm	Micrograms per cubic metre
Mtpa	Million tonnes per annum
OEPA	Office of the Environmental Protection Authority
PD Act	<i>Planning and Development Act 2005</i>
LPS Regs	<i>Planning and Development (Local Planning Schemes) Regulations 2015</i>
PAA	<i>Port Authorities Act 1999</i>
PEC	Priority Ecological Community
PPA	Pilbara Ports Authority
SIA	Strategic Industrial Area
TEC	Threatened Ecological Community
ToPH	Town of Port Hedland
WAPC	Western Australian Planning Commission

APPENDIX 1

Port Hedland Air Quality Health Risk Assessment for Particulate Matter

(Department of Health, 2016)



Government of **Western Australia**
Department of **Health**

Port Hedland Air Quality Health Risk Assessment for Particulate Matter

Environmental Health Directorate

January 2016

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EXECUTIVE SUMMARY

- There is sufficient evidence of potential impacts on human health from dust, specifically PM₁₀ in the Toxikos HRA to warrant dust management controls and strategic and land-use planning to reduce community exposure to dust.
- The majority of the public health burden of dust in Port Hedland is associated with PM₁₀ concentrations over 70 µg/m³. The burden is based on certain health outcomes that show an increase in all-cause mortality and an increase in hospitalisation for respiratory conditions. The areas affected are closest to the Port.
- The number of affected individuals is very low but only because the population is small. If the population was larger or is allowed to become larger, the health outcomes would be more readily discernible and demanding of more immediate regulatory control.
- A legacy of the rapid growth of Port Hedland is the close proximity of residential areas to commercial operations at Nelson Point and the port. This means that fugitive dust from port and commercial operations at Nelson Point and Finucane Island disperses over residential areas under certain meteorological conditions, despite good dust management control.
- PM₁₀ concentration in ambient air decreases with distance from the harbour therefore the level of risk could be well managed in residential areas further east toward Pretty Pool by reporting against the interim guideline (24-hr average of 70 µg/m³ PM₁₀, + 10 exceedances). The guideline could also be applied to South Hedland and Wedgefield.
- There is sufficient evidence based on the pattern of exceedances that areas nearest the harbour at Nelson Point are unlikely to meet the interim guideline. Targeting industry alone however, would ignore broader dust management and exposure reduction opportunities. Exposure risk in these areas could be managed through land-use planning to limit exposure and population growth in Port Hedland.
- Further recommendations to manage and understand the risks posed by dust in Port Hedland are presented. These include continued air monitoring, air-quality modelling to better understand the dust impact area of the port, Nelson Point and Finucane Island operations and further research on the health effects of crustal dusts.

BACKGROUND

Port Hedland is the world's largest volume port for bulk materials export. Iron ore, salt, manganese, chrome and copper concentrates and other commodities, including cattle, fuel and chemicals pass through Port Hedland. Stockpiles containing iron ore, salt, manganese and copper are located relatively close to residential areas at Nelson Point (see Figure 1). Heavy vehicles and ships, material stockpiling and handling and a predominantly dry, windy climate contribute to dust (particulate matter or PM¹) dispersal over the local residential areas.



Figure 1. Aerial view of Port Hedland showing proximity of residential areas to Nelson Point and Finucane Island commercial operations (courtesy Port Hedland International Airport).

In early 2009, the Environmental Protection Authority expressed concern that 24 hour PM₁₀ dust concentrations regularly exceeded the air National Environmental Protection Measure (air NEPM) of 50 µg/m³ (+ 5 exceedances for natural events) and that existing planning arrangements allowed for residential development in the West End, defined as the area between McKay Street and Taplin Street, without any precaution for dust and noise levels. They 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”.

In response, a Taskforce (The Port Hedland Dust Management Taskforce) reporting to the Premier was convened by the Department of State Development (DSD) in May 2009. The Port Hedland Dust and Noise Management Plan (DNMP) was prepared and released in March 2010.

¹ Particulate matter is categorised as PM₁₀ or PM_{2.5}. PM₁₀ refers to particles of 10 micrometres in diameter and smaller while PM_{2.5} refers to particles of 2.5 micrometres in diameter and smaller.

The Taskforce recognised five broad categories where the DNMP must give clear direction for action. These were:

- health risk assessment and analysis (in terms of air-quality);
- environmental management controls;
- land use planning;
- industry initiatives; and
- governance.

A legacy of the rapid growth of Port Hedland is residential areas in close proximity to Port operations. This means that fugitive dust from Nelson Point and Finucane Island operations may disperse over residential areas under certain meteorological conditions, despite good dust management control. The Taskforce was therefore concerned that the PM₁₀ NEPM could not be achieved in Port Hedland. Independent toxicologists from the University of Newcastle in Sydney and researchers from the Lung Institute of Western Australia (LIWA) confirmed the NEPM for PM₁₀ was intended for an urban setting rich in combustion PM and that it was reasonable to expect that it might not apply to Port Hedland because the PM in Port Hedland was crustal (of natural origin). They recommended a precautionary approach that involved reducing dust concentrations over time and implementing strategies to monitor and assess air quality in Port Hedland. As part of a literature review, LIWA recommended a 24-hour coarse dust (PM_{10-2.5}) guideline of 70µg/m³ on the basis of the unique physiochemical profile of PM in Port Hedland.

The Taskforce subsequently set an interim 24-hour PM₁₀ guideline of 70µg/m³, with 10 days of allowable exceedances for a period of five years. Importantly the interim guideline was expected to be met east of Taplin Street and it was expected to be exceeded west of Taplin Street where earlier modelling for the DNMP had predicted the number of exceedances would remain well above the interim guideline. Under the DNMP the interim guideline was therefore also the target guideline for the West End of Port Hedland for industry to work towards achieving as part of an agreed continuous improvement framework to reduce emissions over time. The target guideline is a 'cumulative' reduction target and applies to all industries, not a particular company. The Port Hedland Industries Council (PHIC) was established in parallel to the Taskforce to facilitate whole-of-industry cooperation with the target guideline specifically and the DNMP generally and to develop an integrated approach to air-quality (and noise management).

Part of the rationale for setting the guideline for PM₁₀ and not PM_{10-2.5} as recommended by LIWA was to make use of existing monitoring infrastructure; to allow for exceedances from regional bushfire smoke and dust storms and to allow improvements in emissions to be judged against existing data. A five year period was to allow time for PHIC to implement continuous emission control initiatives and for government to undertake a human health risk assessment of the potential health impacts of dust (particulate matter) in Port Hedland.

A. COMMENTARY ON THE TOXIKOS HEALTH RISK ASSESSMENT

Toxikos Pty Ltd was engaged to undertake an independent health risk assessment of air quality in Port Hedland. The Taskforce convened a health subcommittee chaired by the Department of Health (DOH) to engage Toxikos and facilitate their information requirements. Toxikos was requested to focus on risks associated with exposure to coarse and fine particulate matter and its constituents of certain metals, silica and mineral fibres.

Toxikos completed the Draft HRA report (Toxikos Report) in December 2014 (unpublished). It concluded the pollutant having the greatest impact on public health in both Port Hedland and South Hedland was particulate matter (PM), specifically PM₁₀. The health impact (based on certain health endpoints) of exposure to the current concentrations of PM in Port Hedland was reported to be higher than that observed in Perth and other Australian capital cities on a 100,000 population basis. It further found that the magnitude of the exposure in Port Hedland was influenced largely by the exceedances of the interim guideline and therefore Toxikos concluded that to manage potential risks from exposure to dust the interim guideline should not be exceeded. The analysis in the draft report was difficult to follow which cast considerable doubt over the veracity of the conclusions. Notwithstanding this, three peer reviews supported the conclusions of the Toxikos HRA with certain caveats to be addressed in the final report.

Toxikos completed the final HRA report in July 2015 (unpublished) responding to the peer review comments. The final report addressed the comments in varying degrees of thoroughness and transparency which unfortunately did not improve the clarity or readability of the report. Indeed, the content added to their Executive Summary helped to further divide opinions as to the interpretation of the risk estimates. To the casual reader their Executive Summary appears to portray the risks of living in Port Hedland as immediate and urgent though on thorough reading of the entire Toxikos report, this is not the case.

DOH evaluated the rationale for the claims made in Toxikos' Executive Summary. In those evaluations DOH found that the peer review comments could have been more thoroughly considered. DOH concluded that, in building the case for the risk estimates, there are instances in the report where the choices that were made regarding the method of assessment, the parameters and the data treatment employed, were not reported with sufficient detail or transparency even for a technical report. This together with the numerous editorial and grammatical errors detracts from the readability of the report and casts doubt on the conclusions reached. However the fundamental scientific integrity of the report should not be sacrificed because it falls short of the expected level of reporting. The fundamental science is sound as the peer reviews have attested.

DOH also considered whether Toxikos fulfilled the scope of the work required. The scope was fulfilled with respect to determining the risk estimates and the appropriateness of the interim guideline for PM₁₀ for Port Hedland. However environmental factors contributing to the exposure risk were not assessed with the detail expected and therefore did not meet the scope in this regard. This work was subsequently undertaken by the DOH to supplement the Toxikos HRA and is presented in the sections that follow. The reader may be referred to the

Toxikos Report from time to time so that elements that have been well considered in the Toxikos Report are not reproduced unnecessarily.

The assessment that follows also considers certain elements and concepts presented in the Toxikos Report that were raised by reviewers as requiring additional information.

1. HUMAN HEALTH RISK ASSESSMENT

This assessment is the final health risk assessment and must be read in conjunction with the Toxikos Report.

1.1.WHAT IS A HUMAN HEALTH RISK ASSESSMENT?

Human health risk assessment (HHRA) is a process intended to estimate the risk to a population following exposure to a substance of concern. The process considers uncertainties in determining the risk, the characteristics of the substance of concern and the characteristics of the population of interest.

Many social and economic factors impinge upon risks to human health and wellbeing but these are not included in a human health risk assessment. A human health risk assessment is strictly an analysis that uses information about the substance of concern to estimate a theoretical level of risk for people who have been exposed or might be exposed to the substance.

A HHRA does not measure individual risk. It measures population risk and applies conservative margins into the risk analysis to ensure that susceptible and vulnerable people (e.g. children and the elderly) are included.

1.2.WHAT IS INCLUDED IN A TYPICAL HHRA

A HHRA assembles and summarises scientific information to determine whether the substance of concern is a potential hazard and the extent of possible health risks.

A HHRA identifies the exposure pathways, in other words it identifies the ways people are exposed to a substance, and it includes an assessment of acute (immediate or short term) and chronic (delayed or long-term) health risks that might arise from the exposure. In the context of Port Hedland, people are exposed to dust in many ways however only by breathing dust is there potential for harm.

The Toxikos HRA evaluates the potential for a range of identified pollutants in ambient air to cause direct harmful effects to people who are exposed. It considers the potential for health effects to occur from exposure to dust from all sources rather than a single source.

1.3.HOW ACCURATE ARE RISK ESTIMATES

In general, risk assessments are limited by a lack of complete information simply because it is not possible to know everything about the substance of concern or the population exposed. To counteract this, risk assessments are performed using safety-margins that are likely to overestimate the risk.

During an assessment, it may not be possible to fully identify and uncover all the information required; when the information gaps are small and not critical to the central issues this should not compromise the integrity or quality of the scientific and technical aspects of the

assessment. Additional analysis can be undertaken to fulfil the requirements at a later stage in the assessment process or even at a later date.

For example, potential risks of chromium III and VI were not assessed in the Toxikos HRA due to a critical sampling error during the data collection period. This became obvious only after the sampling period had passed. While this weakens the risk assessment for chromium, it does not weaken the assessment of the other substances of concern because these were assessed separately. A period of monitoring will be required in the future to enable an assessment from any potential risk posed by chromium.

1.4. HOW ARE HUMAN HEALTH RISK ASSESSMENTS USED

A HHRA helps regulatory agencies, industry managers and the public determine strategies that will ensure that human health (or public health) is protected from substances of concern. For example, if the substance of concern is emitted from an industrial facility, a HHRA helps to define the boundaries or conditions under which the facility can operate.

A HHRA should not include decisions about how a risk is managed, instead it should characterise the estimated risk. Instead, the HHRA should be incorporated into risk management and policy decisions by regulatory agencies and facility managers.

This assessment (incorporating the Toxikos Report) is intended for use by planners and policy makers to guide risk management options for dust in Port Hedland. It should not be the only source of information guiding the decisions; instead it must be combined with findings of other studies independently commissioned including the noise model, the air-quality model and the source apportionment model.

2. HEALTH RISK ASSESSMENT METHODOLOGY

2.1. GENERAL OVERVIEW

The enHealth 2012 document 'Guidelines for assessing human health risks from environmental hazards' sets out a framework that describes the steps that should be followed when undertaking a human health risk assessment. The framework is shown in Figure 2.

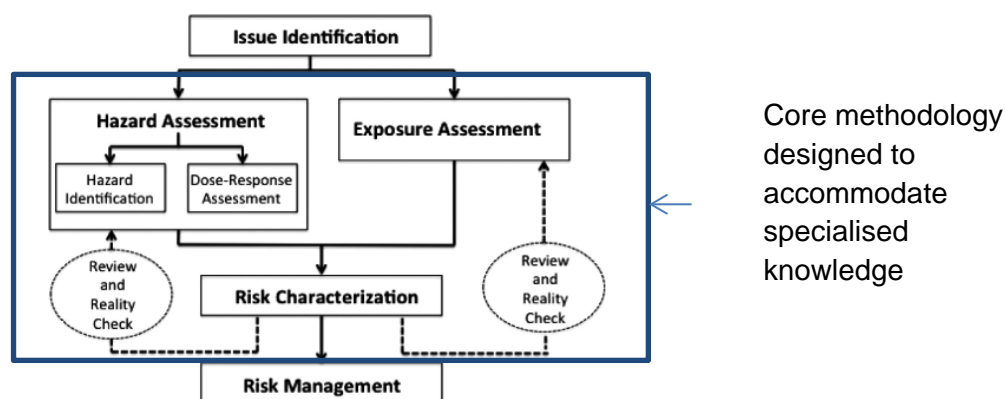


Figure 2: HHRA Framework

- I. **Issue Identification** – describes the key issue(s) amenable to risk assessment. It also establishes a context for the risk assessment by specifying the problems the risk assessment will address. It describes the link between the substances of interest and the population exposed.
- II. **Hazard Assessment** – looks at the capacity of agents to produce adverse health effects and where possible compares known concentrations to safe guideline values to identify which chemicals represent the greatest concern. This includes the collection and analysis of relevant data, where they exist.
- III. **Exposure Assessment** – Investigates the character of the exposure; the population exposed and estimates the exposure concentration for all exposure pathways to the relevant population.
- IV. **Risk Characterisation** – combines the information from the above stages. It characterises the potential for adverse effects to occur.
- V. **Risk Management** – The overall goal of the risk assessment process is to provide the best possible information to support effective risk management decision-making.

Steps I and V accommodate those with the best knowledge of the issue and responsible for managing the risks such as government agencies and facility managers. This is the focus of the Taskforce.

Steps II to IV form the core of the HHRA framework designed to accommodate specialist knowledge; this is the focus of this assessment.

3. PORT HEDLAND

Port Hedland is a relatively small town in the north west of Western Australia approximately 1300 km from Perth. The local government area covers approximately 10,000 square kilometres and is the largest town council in the Pilbara region. The local government area includes the suburbs of Port Hedland, South Hedland, Wedgefield and satellite Aboriginal communities and camps and is known as the 'Town of Port Hedland'. For the purposes of this assessment, the term 'Port Hedland' refers specifically to the geographic peninsula west of Pretty Pool and the term West End refers to areas between McKay Street and Taplin Street.

The Town Council of Port Hedland reported a permanent population of 4590 in 2012/2013. A relatively large population of FIFO (fly-in-fly-out) workforce is also accommodated in South Hedland and mining camps close to the airport (3000 strong workforce according to the Town Council). This population is not considered in this assessment because of the transient nature of the workforce and changing profile of individuals within the workforce. Furthermore the majority of the workforce was not accommodated within the risk assessment area for the duration of the monitoring period. A population age profile for Port Hedland is provided in section 5 of the Toxikos Report.

Port Hedland is located in a semi-arid environment. The rich mineral content of the region is reflected in the red soil and dust. The winds are predominantly from the east-south-east in the morning and north-north-west in the afternoon. Seasonal variation is seen in the change of wind direction January through to March and again in October through to December.

The port of Port Hedland is the largest bulk port in the world accommodating major mineral exporting companies operating within or close to the port at Nelson Point and Finucane Island. Residential areas lie in close proximity to the Nelson Point operations as a legacy of the rapid growth of Port Hedland (see Figure 1). Emissions to the environment from the major operators are regulated by the Environmental Protection Authority (EPA) and the Department of Environment Regulation (DER), which includes the functions of the former Department of Environment and Conservation. Due to the close proximity of the residential areas fugitive dust emissions and their associated impacts on the health of the community are an ongoing concern for the community and the Taskforce.

4. ISSUE IDENTIFICATION

The sustainable development of Port Hedland is a key policy of the State Government which is seen as vital to the State's future. Although economic development is considered important in developing Port Hedland the health of the community is a critical issue.

During the government and community consultation period leading to the release of the DNMP for Port Hedland the issues raised included concerns about fugitive dust emissions from transporting, stockpiling and handling of mineral ores at the extensive Nelson Point and Finucane Island facilities and about the impact of further growth in mineral exports on dust levels impacting the West End.

The main issue concerned the impact of dust (PM_{10}) on the health of the nearby community. Previous dust management reports had reported frequent PM_{10} concentrations in excess of the NEPM 24-hour guideline of $50 \mu g/m^3$ (+5 exceedances).

Other concerns identified by the Taskforce included the potential harm from heavy metal particulates and possible silica and asbestos contaminants present in the metal ore concentrates.

The issues were presented to Toxikos in a series of questions in a technical briefing document as follows:

- What health effects are associated with PM and other identified pollutants in the air in Port Hedland?
- What is the contribution of port operations and the adjacent Nelson Point and Finucane Island operations to dust exceedances (this presumes it is possible to determine these contributions from dispersion modelling)
- Is the interim guideline of $70 \mu g/m^3$ for non-specific PM_{10} appropriate for Port Hedland?

- What is the potential impact on health of increasing (or decreasing) PM pollution in Port Hedland?

5. HAZARD ASSESSMENT

The primary export at the port and commercial operations at Nelson Point and Finucane Island is iron ore, but other important mineral exports are salt, manganese, chromite and copper concentrate. The mineral concentrates may also contain silica and asbestos contaminants from geo-located substrates at the mine site. A list of substances was created for the risk assessments and is based on the prevalence of the substance in the air or the potential to cause harm. The substances of interest are the Contaminants of Concern (CoC) listed below:

- Particulate matter: PM₁₀ & PM_{2.5}
- Chromium: chromium III & VI
- Copper
- Manganese
- Iron oxide
- Silica
- Asbestos

The gases sulphur dioxide and nitrogen dioxide were not considered CoC but were included in the assessment because of the potential for additive or synergistic effects with co-pollutants in the atmosphere and to provide a baseline for future reference.

The list focuses the risk assessments on these substances and their potential for harm through inhalation exposure as this is the exposure pathway of concern. In finalising the list it was recognised that the analytical methods employed had the capability to identify a more extensive number of metals and these would be discussed should any show significant results.

The most important criteria for assessing the CoC and determining harm are the following criteria:

- i. The availability of good quality toxicological and scientific evidence. This is the basis of hazard assessment and underpins the HHRA process.
- ii. The availability of ambient air standards or guidelines against which results of monitoring are compared.

Other criteria that were considered included:

- The potential for significant community exposure in Port Hedland since without exposure there is no harm. This was based on previous monitoring and analysis of

dust samples and proximity of residential areas to the port and commercial operations at Nelson Point and Finucane Island.

- The potential for significant variations in exposure that might reveal source control issues. Any variations of this nature would be raised as a separate parallel issue.
- The availability of analytical techniques with the appropriate sensitivity to measure pollutants in ambient air
- The level of community concern in regard to dust and industry emissions.

Table 1 in the Toxikos Report lists the CoC and the standards or guidelines that are used in this assessment. These are based on extensive reviews of available toxicological and exposure data by international and national regulatory agencies and effectively provide an independent measure of health risk. An existing guideline for iron oxide was not available and was therefore derived by ToxConsult an independent consultant and was reviewed by Toxikos. The derivation is presented in section 4.1 of the Toxikos Report.

The health effects of the CoC are briefly discussed below however a detailed literature review is presented in sections 6 and 7 of the Toxikos Report.

5.1. Particulate Matter:

The body of literature for the adverse health effects from exposure to particulate matter is large and well established. The health effects associated with exposure to ambient air particle pollution ranges from small transient changes in the respiratory tract and impaired lung function to mortality.

Overall the evidence is strong for effects on both the respiratory and cardiovascular systems for both long term and short term exposure. It appears that long term repeated exposure is much more detrimental than short term sporadic exposure. The most severe effects being reduced life expectancy due to long-term exposures.

PM varies in composition and size from region to region even location to location reflecting different sources. For this reason there is still relative inconsistency in the scientific literature of the effect of size over composition. It is generally agreed however that physicochemical characteristics of particles may be an equally important predictor of toxicity and reactivity as size.

Recently the US EPA classified the $PM_{2.5}$ size fraction as 'causing' cardiovascular effects and increasing mortality and, as likely to cause respiratory effects. $PM_{2.5}$ was also linked to a lesser extent to adverse reproductive and developmental effects.

A recent 2013 World Health Organisation review of scientific literature since 2005 found that coarse particles, that is particles between $PM_{2.5}$ and PM_{10} have effects independent of the $PM_{2.5}$ fraction. Coarse ($PM_{2.5-10}$) and fine ($PM_{\leq 2.5}$) particles deposit at different locations in the respiratory tract, have different sources and composition, act through partly different biological mechanisms, and result in different health outcomes. The WHO review concluded that the weight of evidence supports the notion that short-term exposure to coarse particles

(including crustal dust) is associated with adverse respiratory and cardiovascular effects on health including premature mortality.

Very few studies have looked at the health effects of crustal dust (particulates derived from the earth from natural sources e.g. mineral ore dusts) specifically. A commonly held belief among scholars has been that crustal dust was likely to be less potent than urban dust. Indeed, this was the reasoning behind the Taskforce's interim PM₁₀ guideline for Port Hedland. Recently however studies including the WHO review have emerged that challenge that belief. Epidemiological studies have identified associations between crustal derived dusts and increased morbidity and mortality in Europe and Asia.

5.2. Metals, silica and asbestos

Iron-oxide

Since the major component of airborne dust arising from port and commercial operations is iron oxide it is reasonable to assume it may be a major contributor to any health effects that may occur in relation to PM₁₀ exposure. Iron-ore dust exposure has been suspected of causing, through association, a variety of observed health effects (siderosis, Labrador lung, cancer of the lung, cancer of the stomach and cardiovascular disease), an exacerbating agent (asthma, COPD, allergy mediated respiratory diseases) and an adjuvant to other inhaled particulate pathogens and toxins (PAHs, silicas, bacteria, spores). However, despite epidemiological studies investigating observed health effects associated with iron-ore dust inhalation, and *in vitro* studies investigating the effect of iron oxides on reactive oxygen species and cellular reactivity, there is no clear causal link between iron-oxides and disease. Overall, there is no clear evidence of a causal link between exposure to airborne iron oxide particles and disease. There is evidence that exposure to dust from iron ore mining may be associated with an increased risk of mortality and morbidity. However, it is more likely that the mechanism of injury involves iron oxide in combination with other agents, in a synergistic or additive relationship.

Manganese

Manganese is an essential nutrient however it exhibits toxic effects if exposure is excessive or prolonged. Manganese neurotoxicity is the most common health effect associated with prolonged inhalation exposure. Effects are characterised by damage to regions of the brain that control muscle movement and behaviour and manifest as changes in gait, dexterity and cognitive function.

Copper

Copper is an essential nutrient but can exhibit toxic effects when inhaled in high concentrations. Most public health effects associated with over exposure is irritation; coughing and sneezing. More severe effects like pulmonary fibrosis tend to occur in workers exposed to copper dust on a long term basis.

Chromium

Chromium was not assessed due to a sampling error. It was listed as a CoC firstly, because it is found in the bulk ore exports and secondly for its toxic potential. Analysis done earlier to inform the LIWA literature review did not reveal any exceedances of the guideline for chromium therefore there is no immediate concern that it was not able to be assessed. Chromium is an essential nutrient. It is found in crustal material therefore it is ubiquitous in the environment. In the environment it can exist predominantly in two valence states – trivalent chromium (Cr III) and hexavalent chromium (Cr VI). Cr III is the most stable and abundant form of chromium. Most health effects associated with over exposure to inhaled Cr III are upper respiratory tract irritation with some lower respiratory inflammatory affects. Chromium is a skin irritant and can cause dermatitis. Renal effects have been reported following exposures at extremely high concentrations in occupational settings.

Hexavalent chromium is a genotoxic carcinogen. Chronic inhalation of hexavalent chromium compounds increases the risk of lung cancer. The potential for human exposure to hexavalent chromium in the population is generally small because it is relatively unstable in the environment converting quickly to Cr III in the presence of oxidising organic matter in the air and soil. Lung cancer from Cr VI exposure is almost exclusively confined to exposures in occupational settings and communities living near unregulated toxic waste landfills in developing countries.

Silica

Crystalline silica is carcinogenic, causing lung cancer when inhaled. Lung cancer is mostly associated with occupational exposure to respirable crystalline silica. Additionally, it can cause silicosis, which in severe cases can be disabling, or fatal. The respirable silica dust enters the lungs and triggers scar tissue to form thus reducing the lungs' ability to take in oxygen. Since silicosis affects lung function it increases susceptibility to lung infections. Although crystalline silica is a crustal component it is not associated with health effects until it is reduced in size and shape to respirable particles through mechanical processing like grinding or crushing. Lung cancer and silicosis from silica exposure are not known to have been reported outside occupational settings.

Asbestos

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 over a longer period of time. Asbestos-like material has been identified to occur alongside deposits of mineral ores.

Nitrogen dioxide and sulfur dioxide

Nitrogen dioxide and sulfur dioxide occur naturally in the atmosphere and are also produced by combustion. Both can serve as good markers for traffic-related pollution and tend to be

highly positively correlated with other combustion sources and particulates in urban environments. Adverse health effects are mostly linked to short term exposures and tend to be transient and short lived. These include airway inflammation and increased respiratory symptoms particularly in people with asthma. They differ in their capacity to cause adverse health effects in important ways. Sulfur dioxide is a more potent trigger of short-term respiratory effects compared to nitrogen dioxide while nitrogen dioxide combines readily with other compounds in the air including particles in a synergistic or additive relationship which have the potential to cause longer term respiratory health effects.

5.3.SUMMARY

- The CoC were chosen for their prevalence in the air in Port Hedland or their toxic potential.
- PM₁₀ is the contaminant of most concern in Port Hedland. Although the evidence is strongest for adverse health effects associated with combustion particulates and the small fraction size (PM_{2.5}), the WHO asserts that there is sufficient evidence for respiratory and cardiovascular harm from inhaling coarse particles that includes crustal dusts.
- Silica, asbestos, manganese and hexavalent chromium are contaminants with the most toxic potential in Port Hedland. It is important to acknowledge that human toxicity is dependent on the concentration taken into the body. Therefore if the CoC are present in the air in Port Hedland at a concentration below the recommended guideline or standard, it is unlikely that exposure will lead to any health impact.
- The gases nitrogen dioxide and sulphur dioxide are good markers for combustion related pollution. They associated with short-term acute respiratory effects but nitrogen dioxide can combine in synergistic or additive relationships with other pollutants to cause longer term respiratory health effects.

6. EXPOSURE ASSESSMENT

Exposure assessment describes how the residents of Port Hedland come into contact with the CoC. It determines the magnitude, frequency, extent, character and duration of exposures of a population in the past, currently and into the future. It assumes because susceptible or vulnerable people are included in the exposure analysis most people will be protected. It is important to recognise that not every individual will be protected; those who are highly sensitive such as those with asthma or allergies may not be fully protected.

Exposure to CoC in Port Hedland may occur in a number of ways (Figure 3) however direct exposure via inhalation is the most important exposure route and the focus of this assessment.

6.1.DUST SOURCES

Dust sources from operations of interest in the region were obtained from the Port Authority administration and Town of Port Hedland administration and listed in Tables 1 and 2. The list

is not exhaustive but represents major contributors at the time of monitoring. Emissions typically include those from vehicles, material handling, wind erosion and storage. Natural background dust is presumed to contribute to the dust burden at each location.

An emissions inventory in terms of amount emitted from each industry was not available for 'commercial in confidence' reasons. The dust dispersion model for PM undertaken by Pacific Environment Limited was not available to Toxikos at the time of writing this assessment and therefore it should be recognised that this assessment is based upon the monitoring results from the monitors in the monitoring program implemented for the HHRA.

Table 1: Dust sources from operations of interest in Port Hedland during monitoring period*

Company	Operations
BHP Billiton Iron Ore (BHPBIO)	Iron ore transport, transfer, processing, stockpiling and shipping.
Fortescue Metals Group Limited (FMG)	Iron ore transport, transfer, processing and shipping.
Dampier Salt Pty Ltd	Salt "manufacture", transport, transfer, storage and shipping.
Molly Metals	Iron ore transport, transfer, processing, stockpiling and shipping.
Process Minerals International	Iron ore and manganese transport, transfer, storage and shipping
Consolidated Minerals	Manganese and chromite transport, transfer, storage and shipping.
BP	Oil and fuel storage.
Caltex	
Coogee Chemicals	Chemical import and storage.
Roy Hill Iron Ore	Iron ore transport, transfer, processing, stockpiling and shipping.
Atlas Boodaire power station	210MW gas-fired power station
Toxfree Waste Incinerator Wedgefield	Hazardous waste incinerator

* The list of mining companies operating in Port Hedland could not be confirmed for *commercial in confidence* reasons (PHIC).

Table 2: Dust sources from key local sources

Location	Activity
Spoil bank	Wind erosion, recreational vehicles.
South Hedland	Construction - Housing
Wedgefield	Construction - light industrial
Upgrade road and railway corridors b/w Port & South Hedland.	Construction - transport
Pretty Pool	Upgrade sewerage treatment plant Port Hedland
Airport	Construction - commercial

6.2.EXPOSURE PATHWAYS

Figure 3 summarises the exposure pathways considered for this assessment.

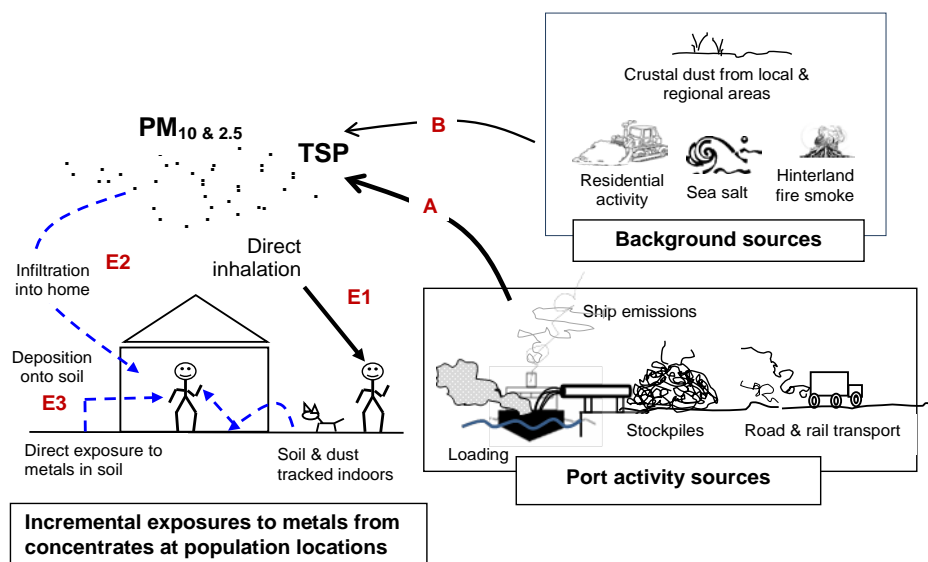


Figure 3: Schematic representation of dust sources and exposure pathways. Adapted from PHHRAM, 2010

Dust sources:

- A. Port activities & operations associated with handling mineral commodities
- B. Background, all sources other than those associated with those in A.

E1: Direct inhalation: This is the most important of the exposure pathways and the focus of this assessment.

E2: Infiltration into buildings: This is a well-recognised exposure route, and since adults and children spend an average 20 hours indoors between home, work and school it is potentially significant. However there is no good quality indoor air-quality data available for assessing this exposure therefore it is assumed that the indoor:outdoor air pollutant ratio equals unity. Hence, this assessment considers that indoor PM exposure is included in the outdoor PM exposure.

E3: Deposition onto soil: Particles in TSP² and PM₁₀ have a tendency to fall out of the air. Larger particles settle more closely to the emission source than smaller ones. Exposure to substances in PM occurs from ingestion of soil and unwashed vegetables, particles can also be tracked indoors. Based on existing evidence, it is not expected the CoC will be taken up and assimilated by plants. Casual inspection of gardens also suggests vegetable growing is not widespread in Port Hedland. Most of the CoC are relatively non-toxic by the oral route and the effects are generally different from those which occur from inhalation exposure. In addition the area experiences one or two cyclones every second year and occasional winter

² TSP – total suspended particulate –dust particles up to 100 micrometres in diameter

rains; this would expect to substantially remove loose surface dust that has been deposited from dust generated by Port activity. Consequently this exposure pathway was not assessed.

Drinking water: This is not an exposure pathway of concern. The East Pilbara Water Supply Scheme supplies customers in Port Hedland, South Hedland, Wedgefield Industrial Area and the local port operation. The Water Corporation *Annual Water Quality Reports 2011-2014* reports this scheme water to meet the Australian Drinking Water Guidelines (ADW) for metals, hydrocarbons, pesticides, radiological and chemicals. The relevant CoC meet the ADG. There are no known residents, according to the Town Council, who rely on rainwater tanks as a source of potable water. Consequently this exposure pathway was not assessed.

6.3.EXPOSURE SCENARIOS

The following sections deal with exposure to dust from all sources. The exposure assessment is based on the measurement of PM₁₀ at each monitor. The monitors are located to best represent community exposure; however it is neither practical nor feasible to cover all residential locations. The addition of air quality modelling would enable a more comprehensive assessment by filling in the small number of data gaps between monitors. This would allow for a more thorough assessment of the contribution of the port and commercial operations and general background. The air quality model undertaken for Port Hedland had not been fully completed at the time of writing this assessment.

The model can run simulations of the following or similar scenarios:

Scenario 1 (S1) – Background exposures defined as PM pollution arising from other commercial activity, domestic activities, sea spray and crustal sources excluding stockpiles (near and far).

Scenario 2 (S2) – Exposures associated with PM pollution from port and commercial operations including all processes associated with exporting ore concentrates including the infrastructure required for port and commercial activity, rail, and truck and ship movement (including Finucane Island).

6.4.HOW MUCH ARE PEOPLE EXPOSED TO THE CoC?

The extent to which people are exposed to the CoC is determined by the concentration of the CoC in the air and personal activity. Individual monitoring is neither feasible nor practical therefore exposure is usually determined on a population basis. This approach considers what the risk might be for a 'hypothetical most at risk' individual(s) and assumes that if the risk is low for that individual(s) then the risk will be low for the majority of the population. The enHealth document *Australian Exposure Factor Guidance: guidelines for assessing human health risks from environmental hazards 2012* provides a range of behavioural and physiological based exposure parameters associated with exposure pathways for calculating exposure.

Information critical to assessing exposure in Port Hedland comes from the monitoring data. The exposure assessment assumes the data captured at each monitoring location

represents the exposure of the population in the area of the monitor. This includes sensitive populations represented by the two primary schools within Port Hedland (one school east of Taplin and another east of Neptune Streets). Both schools are outside the impact zone³ of the port activities including operations at Nelson Point and Finucane Island. Table 3 lists the monitor locations and the substances monitored (Figure 4 shows a map of the monitor locations).

Table 3: Monitor locations and substances monitored

No	Monitoring Station Location	CoC											
		Particles		Metal						Other			
		PM ₁	PM _{2.5}	Fe	Mn	Cu	Cr	M	Na	Si	Fibre	SO	NO _x
1	Richardson Street – Commercial/ Residential	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
2	Kingsmill Street – Port Hedland – Residential (Hospital Site)	✓	✓	x	x	x	x	x	x	x	x	x	x
3	Taplin Street – Port Hedland -Residential	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
4	Neptune Place – Port Hedland -Residential	✓	x	x	x	x	x	x	x	x	✓	✓	✓
5	Wedgefield – light industry	✓	x	✓	✓	✓	✓	✓	✓	✓	x	x	x
6	Bureau of Meteorology (BoM) - airport	✓	✓	✓	✓	✓	✓	✓	✓	✓	x	✓	✓
7	Acacia Way (urban background) – South Hedland - Residential	✓	✓	✓	✓	✓	✓	✓	✓	✓	x	✓	✓
8	Yule River (rural background)	✓	✓	✓	✓	✓	✓	✓	✓	✓	x	x	x

The monitoring network was established primarily to collect data for the HHRA but included an existing monitor at the port (monitor 1) used for compliance. This monitor was considered appropriate for capturing emissions from the Nelson and Finucane Island operations nearest the town centre and its assortment of residential and commercial premises. Data from other compliance monitors were not available.

Monitor 5 was located in the Wedgefield light industrial area which has very few permanent residents but provides site worker accommodation. It is not the focus of this assessment but is included for comparison with the other locations.

The monitoring program and sample collection was managed by the Port Hedland Industries Council (PHIC). This assessment uses data provided from the monitoring program from January 2012 to June 2014 inclusive. Some substances were not monitored from the

³ Impact zone - areas west of Taplin Street.

beginning of the program at some locations and where data differs substantially the differences will be highlighted. The entire dataset had been provided by PHIC and validated by the DER.

Table 7 in the Toxikos Report lists the analytical methods used for the samples collected during the monitoring program; these were within the scope of Australian best practice.

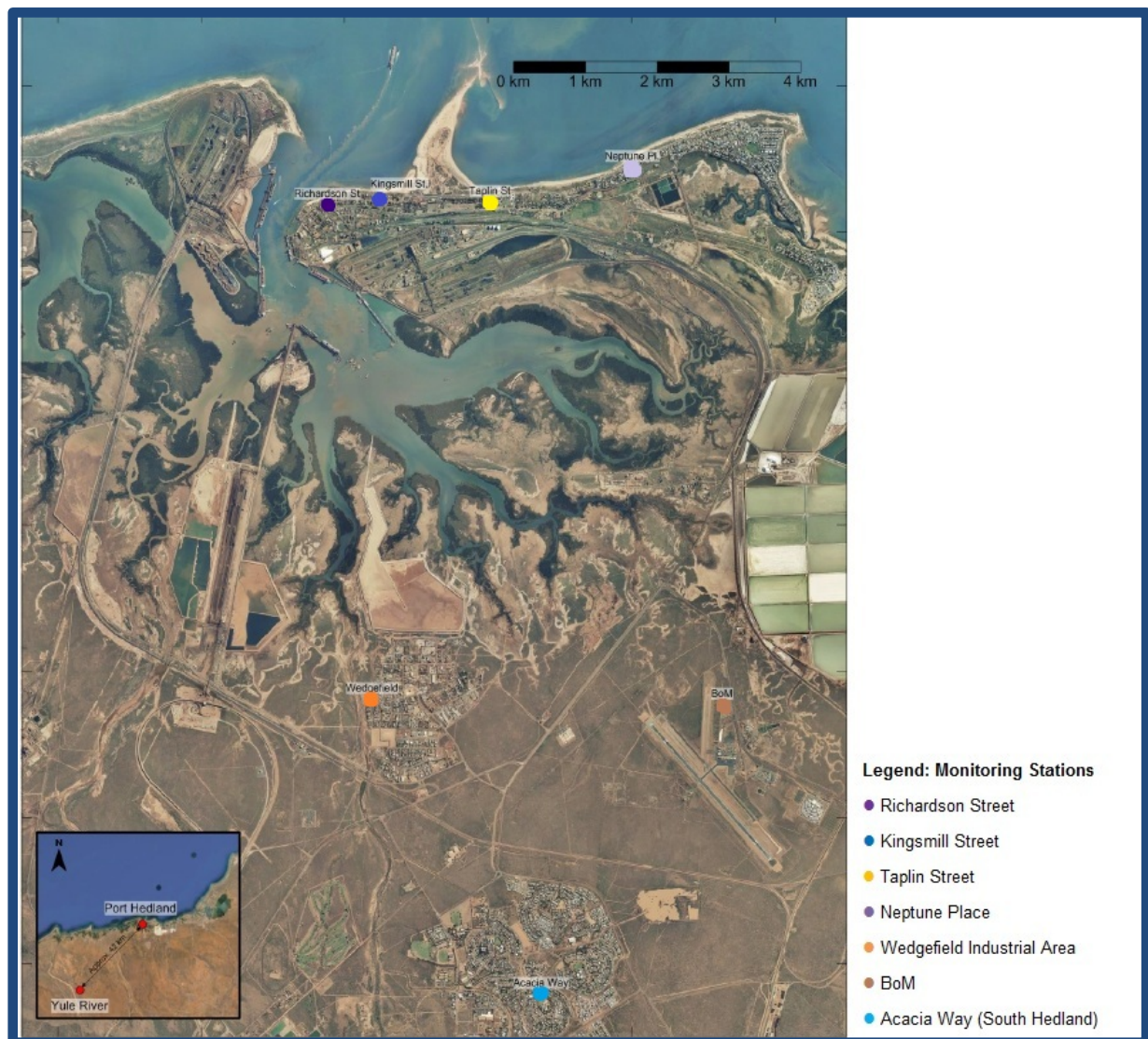


Figure 4: Monitor locations

6.5. PARTICULATE MATTER (PM₁₀) EXPOSURE ASSESSMENT

A simple way to assess the population exposure is to use these data and calculate an average over a specific time period and compare the result with a guideline or standard risk which represents the acceptable risk.

This assessment presents results for two datasets: data provided by PHIC as collected from each monitor and data corrected for extreme values because of bushfires or dust storms. The bushfires and dust storms are typical for the region during spring and summer but the number and magnitude of events is not predictable. Removing the extreme values is necessary to better assess typical exposure. It is important to recognise that values were only adjusted when all monitors were affected. In doing so the effect of local sources was preserved. The dataset was adjusted simplistically by substituting the extreme values with the 5th percentile value assessed for each location. The peak events undoubtedly impact population health however a method to quantify their significance is not yet available. This is discussed further in the next section.

The influence of the bushfire days on exceedances is clearly demonstrated. However when the dataset was adjusted, local sources, rather than natural background sources appeared to dominate exceedances under certain meteorological conditions, particularly in winter and spring. This is supported by the observation that exceedances in Port Hedland were not always seen at Acacia Street (South Hedland) and Yule River.

Figures 5 to 7 show the 24-hr average PM₁₀ concentrations, or the day to day pattern of dust fluctuations at each location for the given year for the entire dataset. All monitors exceeded both the NEPM standard and the interim guideline for PM₁₀. The most notable impact on PM concentration in the region was bushfires and smoke between October and December 2012. Extreme PM₁₀ exceedances were recorded at all monitors during these events. Exceedances of PM_{2.5} coincided with bush fire events (Figure 8) during the same period.

Exceedances also occurred at individual monitoring stations independent of increased PM₁₀ concentrations at other sites; indicating impacts are likely to be from local dust sources. Notable among these are the exceedances at the Wedgefield monitor (monitoring began in October 2012). Most of the Wedgefield exceedances are probably due to local sources in Wedgefield and reflect a light industrial area with unsealed dirt roads, sandy verges and regular heavy vehicle movements.

Exclusive exceedances also occurred at the Richardson, Kingsmill and Taplin Street monitors, implying that sources close to or in the West End contribute to background levels of PM. A limited monitoring program on the spoil bank undertaken by Landcorp coincided with six months of monitoring for the HRA in 2014. These data show exceedances at the spoil bank coinciding with exceedances at the Taplin and Kingsmill Street monitors during onshore winds (Figure 9).

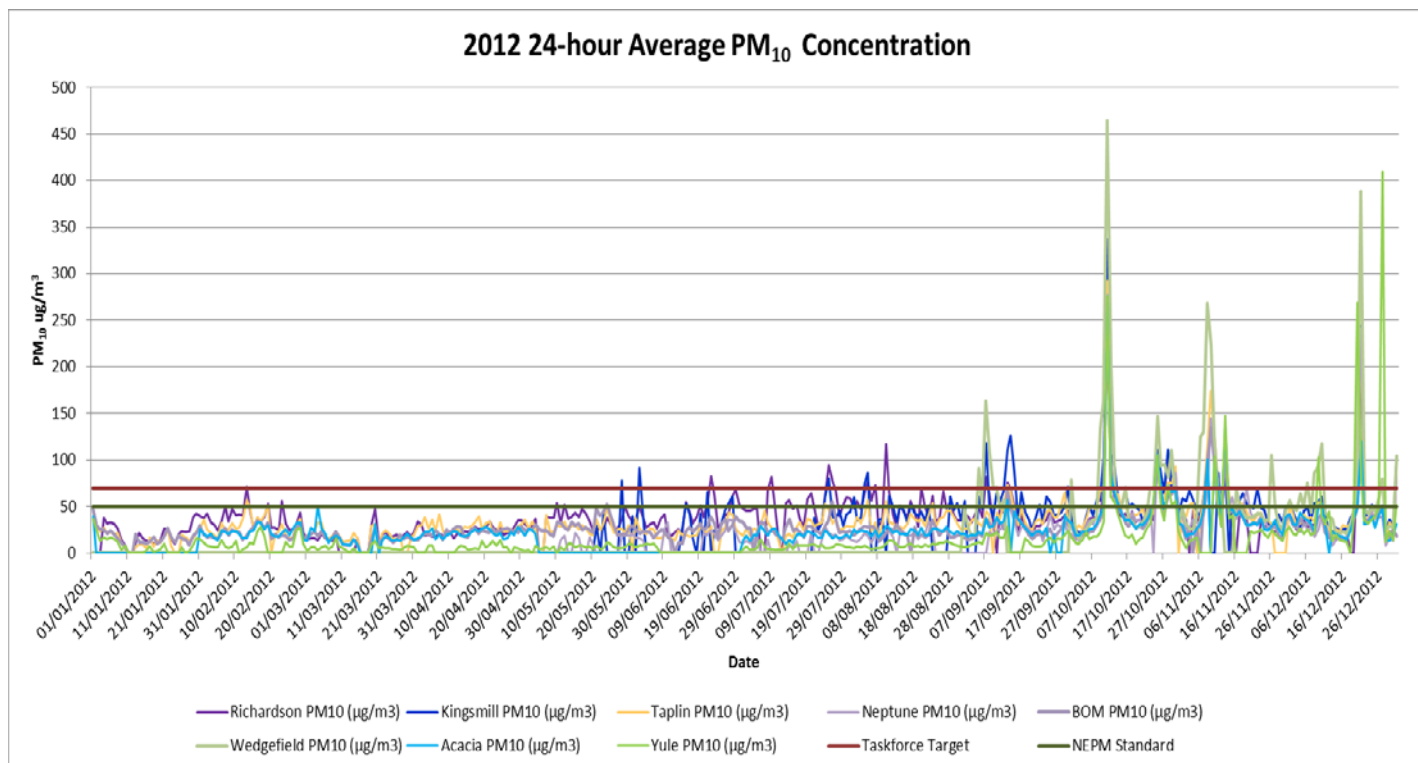


Figure 5: 2012 24-hour PM₁₀ concentrations at all locations (Wedgefield monitoring begins in October)

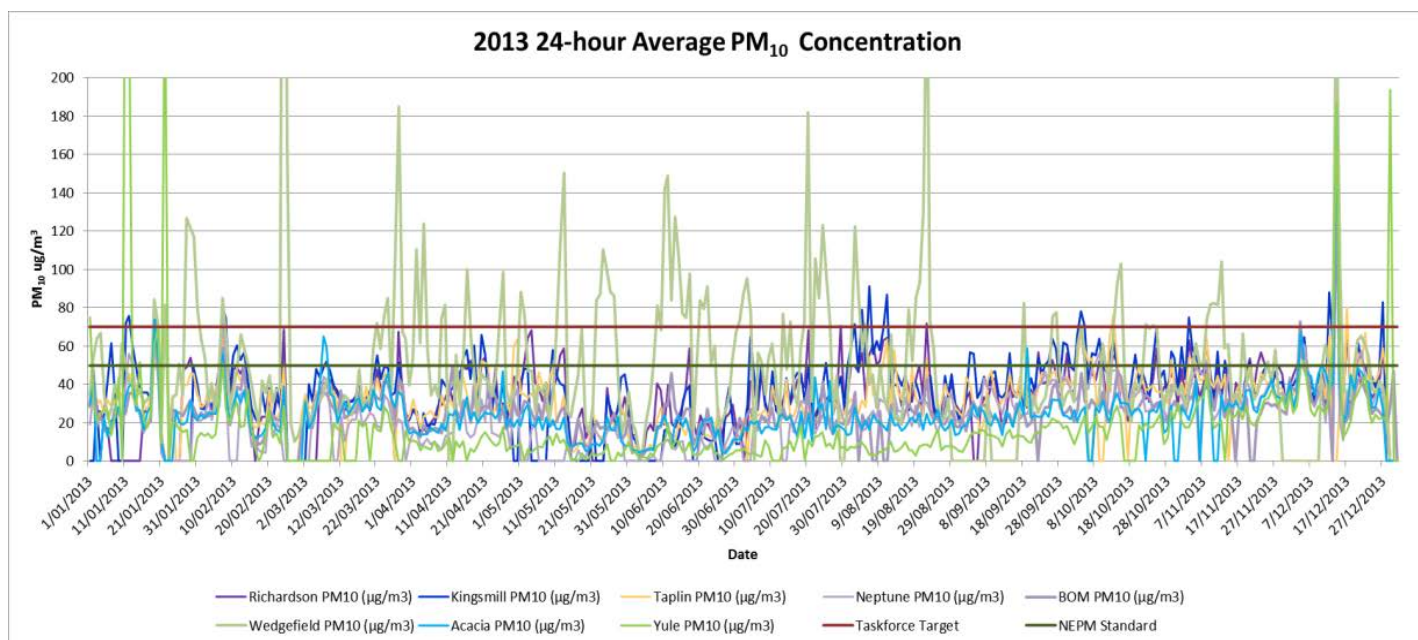


Figure 6: 2013 24-hour PM₁₀ concentrations at all locations

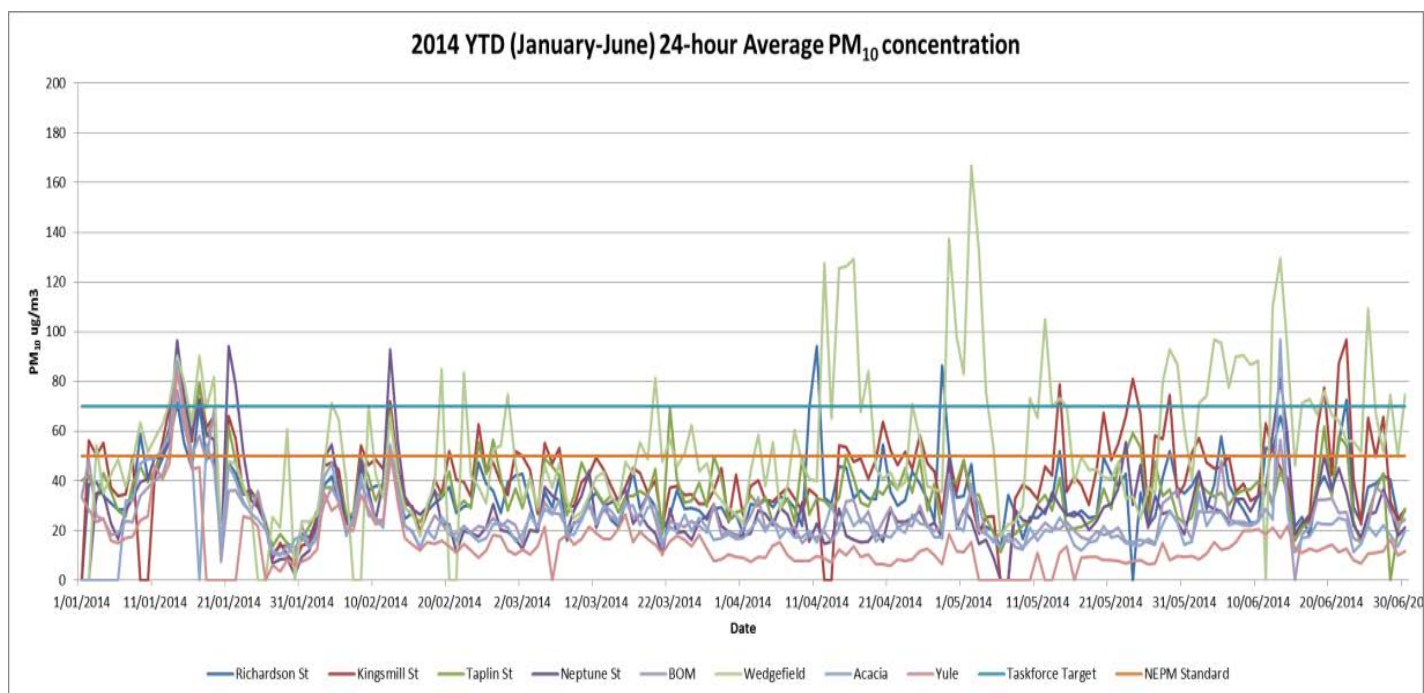


Figure 7: 2014 24-hour PM_{10} concentrations at all locations – January – June inclusive

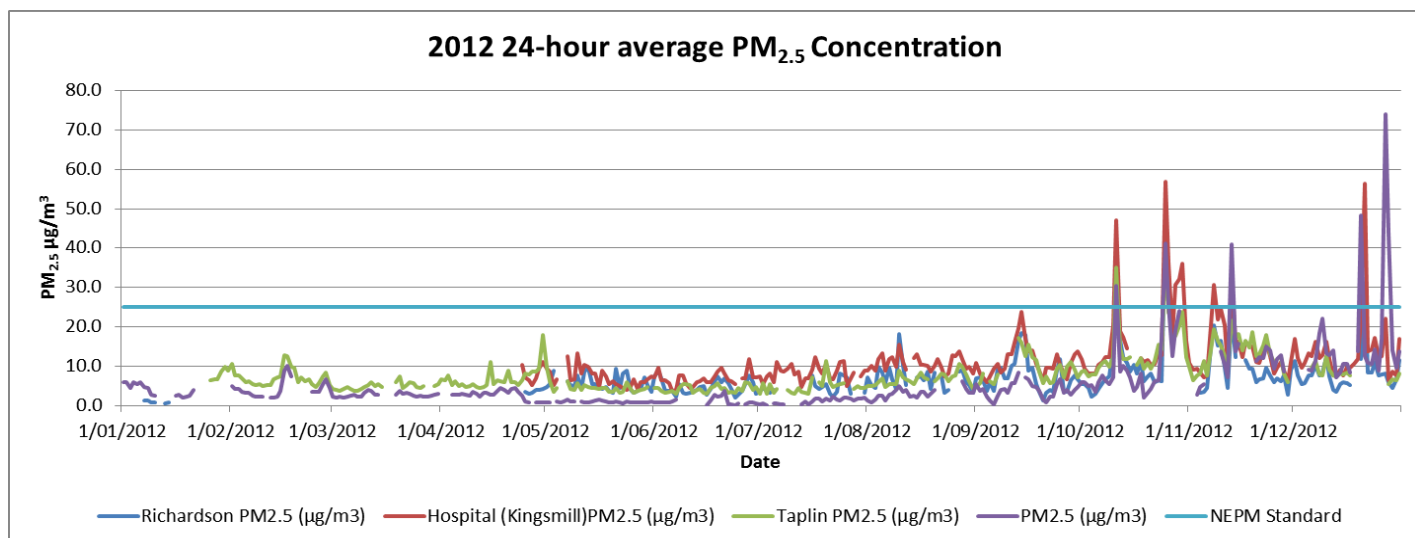


Figure 8: 2012 24-hour $PM_{2.5}$ concentrations at all locations – shows exceedances of the NEPM standard for $PM_{2.5}$ exclusively associated with bushfire days between October and December.

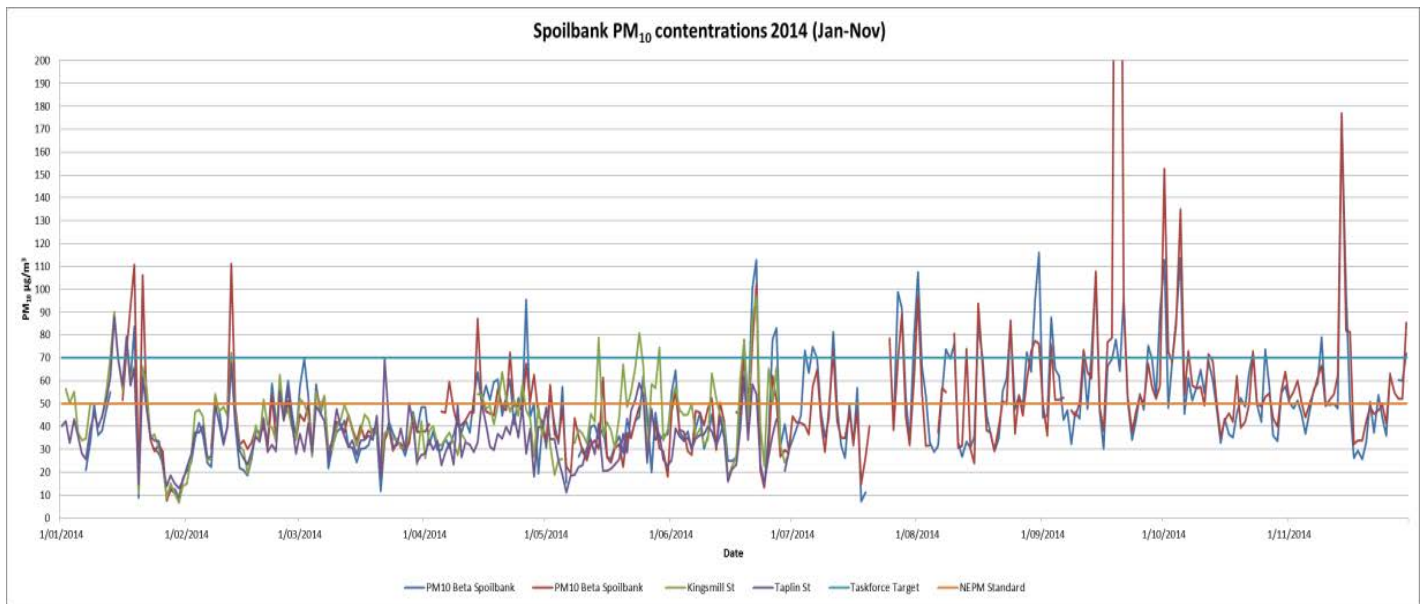


Figure 9: PM₁₀ concentrations from the spoil bank Jan-November 2014. Jan-June exceedance pattern compared with Taplin and Kingsmill. Extreme value truncated; truncated value reflects activity close to monitor site not apparent at the other nearby spoil bank monitor.

Figure 10 shows the percentage of days above the 24-hour PM₁₀ concentration level at each location for 2012 and 2013 combined. The extreme days have been removed. Nevertheless, the higher percentage of days at increased PM₁₀ concentration at Richardson and Kingsmill Streets (representing the West End) compared with the background locations of South Hedland (Acacia St) (representing non-commercial/port activity sources) and Yule River (representing regional sources) is still evident. For example, 16% of sampled days were above the NEPM guideline at Richardson Street compared with 3% and 2% at South Hedland (Acacia St) and Yule River, respectively.

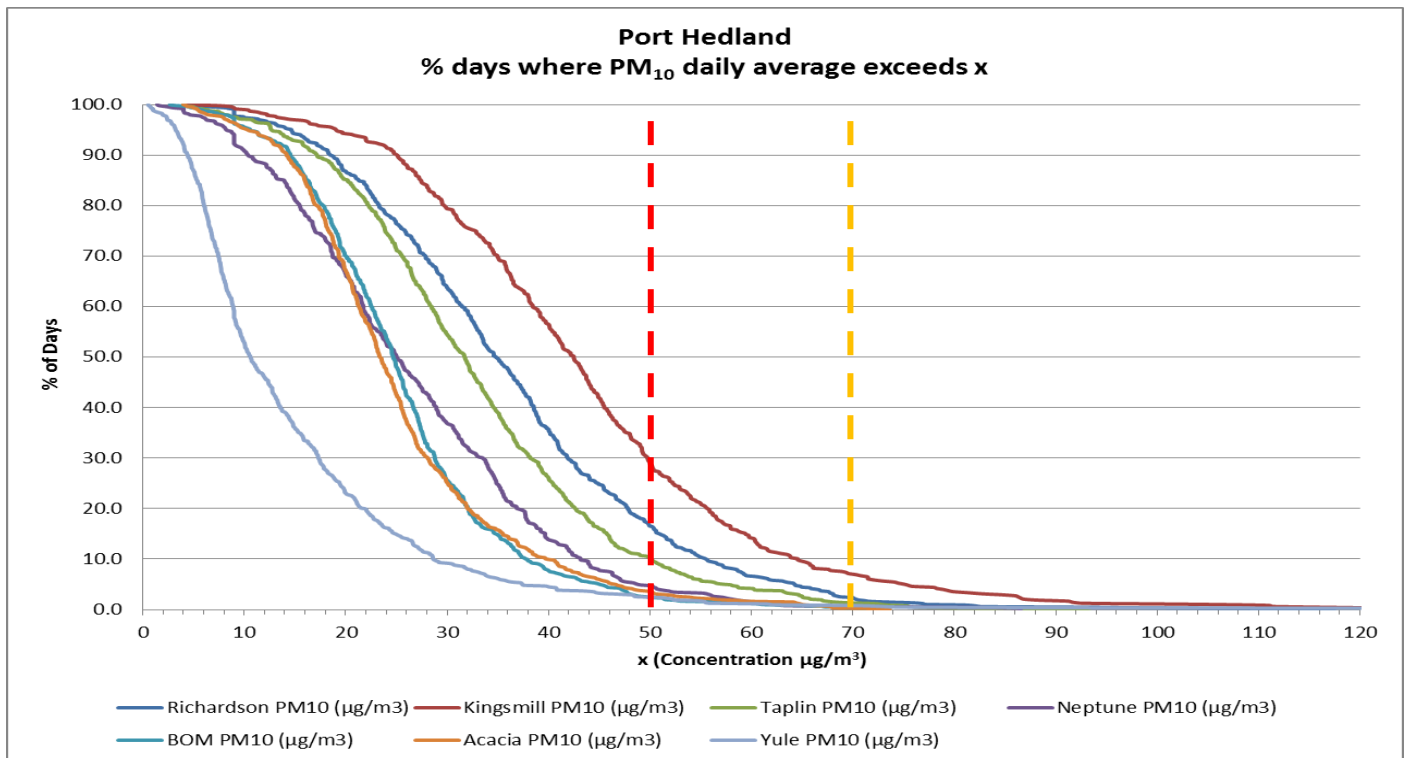


Figure 10: Inverse relative frequency distribution (percentage of days above each concentration level) of daily 24-hour average concentration of PM₁₀ for 2012-2013 inclusive: minus regional background data. Wedgefield not displayed.

The monitoring data in Figure 11 indicates that concentrations of PM₁₀ decrease further away from the West End; the area of greatest impact.

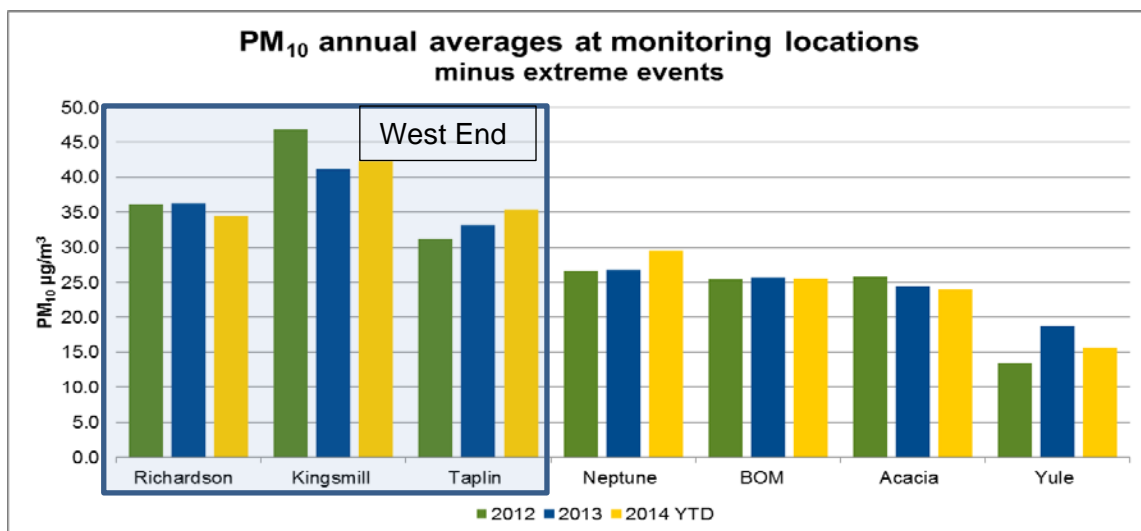


Figure 11: PM₁₀ annual concentrations at each monitoring location

Brief exceedance analysis - The raw data suggest that the majority of exceedances at the Neptune, Taplin, Kingsmill and Richardson Street monitors were independent of PM₁₀ concentrations at the background monitors at South Hedland (Acacia St) and Yule River (Figures 5 – 7).

The number of exceedances of the interim guideline (and the NEPM) increased with proximity to the West End as Figure 12 shows; of interest is the seasonal influence on exceedances as seen in Figure 13. Exceedances were dominant in winter and spring at the Richardson and Kingsmill Street monitors. By contrast, there were no exceedances in autumn and a few exceedances at Kingsmill Street in the summer which were attributed to local sources. A close study of wind direction and speed may reveal the level of influence background dust levels have on exceedances however a casual inspection of the data suggests that exceedances at Richardson Street may be largely independent of background dust levels for much of the year. Figure 12 also shows a small number of exceedances of the NEPM at the Yule River monitoring station; these appear to be associated with local dust events of short duration and not typically associated with exceedances at the other monitoring locations.

This is consistent with preliminary modelling data undertaken for the DNMP prior to 2010 which indicated Nelson Point and Finucane Island operations dominated the background levels and exceedances of PM in the West End at Richardson through to Taplin Street.

An analysis of the metals data in the Toxikos Report further shows that PM₁₀ captured during periods of exceedance contained particulates associated with the port and industrial operations.

An assessment of pollution roses and dispersion modelling (air-quality modelling) would provide a more thorough understanding of how wind direction and speed influence the exceedances and better help identify sources and impact areas so that mitigation strategies can be better directed.

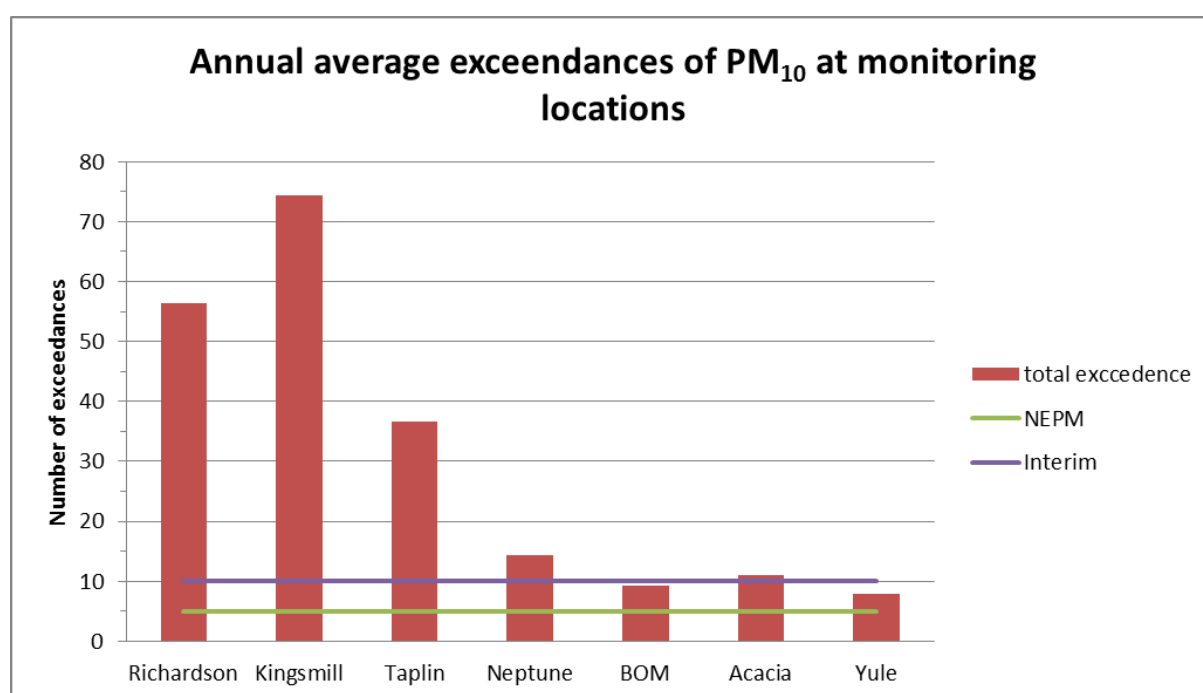


Figure 12: Number of exceedances by location

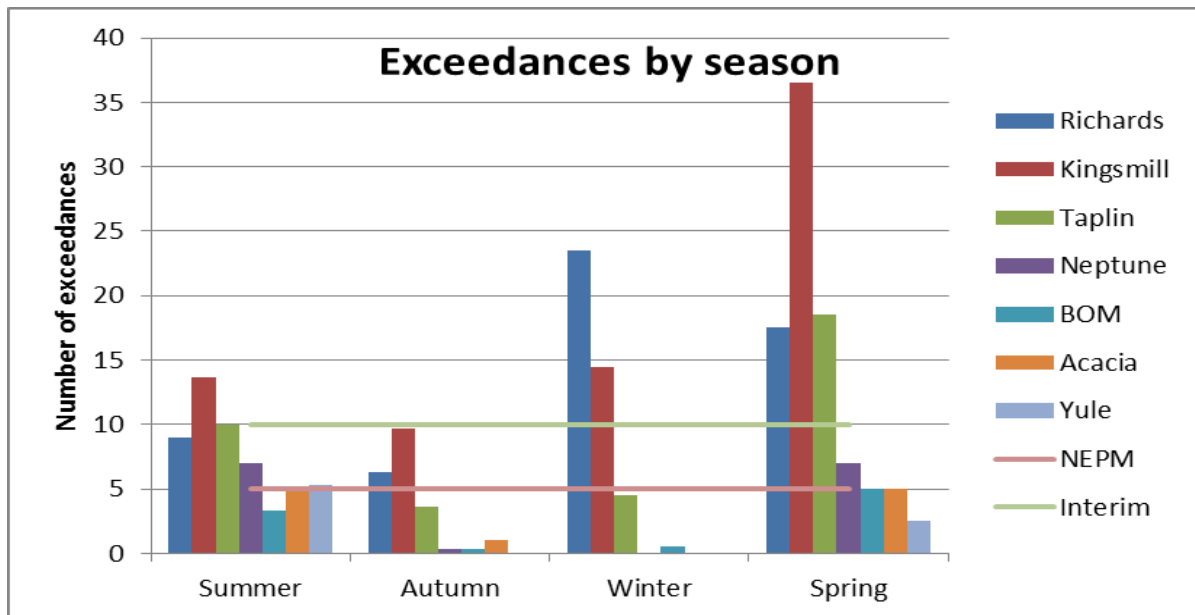


Figure 13: Pattern of average exceedances by season 2011-2014

6.6.SUMMARY

- The reported PM₁₀ levels were consistently above the relevant guidelines under certain meteorological conditions.
- Short-term extreme excursions above the relevant standards were associated with extreme events such as bushfires.
- Extreme events such as bushfires have immediate and obvious impacts in the region driving up PM₁₀ concentrations across all of Port and South Hedland.
- Other exceedances were associated with activities and sources close to the monitors and independent of regional background dust.
- There is a distinct increase in PM₁₀ concentrations with proximity to the port monitor location.
- There is a distinct seasonal influence on exceedances over the interim guideline with a greater proportion occurring in the winter and spring in the West End.
- Under certain meteorological conditions occurring in summer and spring background dust contributes to the PM₁₀ load at all monitors and may influence the exceedances at the port monitor during spring.
- PM₁₀ dispersion modelling would assist to better understand the influence of meteorological conditions and background dust on the pattern of exceedances.
- The sandy spoil bank most likely contributed to exceedances at both the Taplin and Kingsmill Street monitors.

7. RISK ASSESSMENT – PM₁₀

The Toxikos HRA provides a comprehensive and detailed hazard assessment in section 6. A brief summary of the main findings is provided as follows:

- The health outcomes of importance are:
 - Increase in daily mortality (premature death),
 - Increase in hospital admissions associated with:
 - Respiratory disease
 - Cardiovascular disease
 - Pneumonia and bronchitis.
 - Increase in emergency room attendance for pre-existing respiratory conditions, i.e. asthma.
- Persons susceptible to elevated PM₁₀ levels include:
 - Older people (>65 years old).
 - People with pre-existing cardiovascular or respiratory disease.
 - Children and adults with pre-existing respiratory conditions (asthma, bronchitis, chronic obstructive pulmonary disease (COPD)).
 - Children.

As discussed thoroughly in the Toxikos Report the evidence is strong that short-term exposure to PM₁₀ is associated with health effects, and that these effects are independent of the effects of PM_{2.5}. Furthermore the literature is clear that increasing concentrations of PM₁₀ is associated with increased levels of acute and chronic respiratory and cardiovascular health effects. The local hospital health data that was used to inform the Toxikos assessment indicates that it is likely that the short term exceedances experienced in Port Hedland have exacerbated asthma symptoms in some sensitive individuals (reported as morbidity in the Toxikos Report). However, it is not obvious from the small numbers which exceedances (from local sources or from bushfires) contributed more to the increase in symptoms. The long term effects appear to be associated with increased mortality from all-causes. Again, the actual numbers are too small to discern the impact of dust. This means that dust alone does not cause the differences but that dust contributes to the number of increased health outcomes.

As stated earlier a simple method for assessing risk is to compare the monitoring data with the recommended health based guideline or standard. This is relatively straightforward for pollutants that exhibit a threshold effect, but for those like PM where there is no evidence for a threshold concentration below which adverse health effects of PM are not observed the emphasis is to minimise exposure as much as possible and practicable. The NEPM guideline for PM₁₀ represents a metric and a means with which to regulate particulate matter pollution. The NEPM is therefore set so that jurisdictions can achieve the lowest possible concentration of PM in the context of local constraints and capabilities. In setting the NEPM the concentration of background dust is considered and it is therefore possible that the NEPM which was developed for urban environments may not be able to be met in environments with naturally higher background PM levels such as Port Hedland.

Since PM₁₀ concentrations in Port Hedland clearly exceed the NEPM standard as demonstrated in this assessment then health risks attributable to dust are also expected to increase. The question then is not whether attributable risks increase but by how much attributable risks increase. To determine this Toxikos modelled mortality and morbidity rates for various concentrations of PM₁₀, these are presented in tables 11 & 12 of the Toxikos Report. The numbers in table 11 may appear excessive at first glance however it should be recognised that these are extrapolated to a population of 100,000 at levels of dust concentrations currently only found in the far west of the town, closest to the port monitor.

To determine the risks, modelling scenarios are based on concentrations of PM₁₀ from worst case exposure to least case exposure or Richardson Street to South Hedland (Acacia St) respectively. The modelling assumes the entire population of Port Hedland is exposed to the PM level being modelled. This is done so that small changes in risk can be detected. It does not mean that there was or will be an extra death in the West End. It means that the level of PM₁₀ in the West End is conducive to an extra death occurring, and therefore to guard against that possibility – the PM₁₀ level should be reduced or exposure reduced in some other way. Modelling scenarios (using South Hedland and Taplin Street data) indicated that a PM₁₀ concentration of 70 µg/m³ should provide adequate protection of health and wellbeing.

The actual number of people potentially affected in Port Hedland is very small because the population is small and this is reflected in table 12. For instance, based on the modelling, the current levels of PM₁₀ at Richardson Street may account for one extra death per year from all causes and two to three extra hospital admissions per year for respiratory disease.

Given the expected expansion of the port, the interim guideline may be difficult to attain nearest the harbour as is currently evident. Therefore a clear and obvious way to manage exposure is to restrict population growth and manage land-use in areas nearest the West End. The majority of the population live in areas furthest from the harbour where the data (Figure 10) indicates the interim guideline could be met if local dust sources could be better managed; e.g. dust from the spoil bank and unsealed roads and eroded ground as seen in Wedgefield. To determine if the risks across Port Hedland differ according to distance from the port monitor, Toxikos modelled monitoring data collected from Richardson Street, Taplin Street and Acacia Street respectively. The health outcome estimates are reported in in Tables 11 and 12 in the Toxikos Report and show that the risk of being adversely affected by PM₁₀ levels reduces with distance from the port monitor. The risk in Port Hedland may be up to twice as high than for those living in South Hedland. This is consistent with the findings related to hospital admissions for respiratory and cardiovascular disorders from an earlier DOH investigation in 2006 which showed that hospital admissions were higher in the west of Port Hedland than the eastern side. This investigation is discussed further in the Toxikos Report.

The data suggest it may currently be possible to meet the interim guideline at Kingsmill Street if dust from the spoil bank is managed. Air-quality modelling would help determine if this is the case and identify additional buffer distances that may be required to meet future needs of the port.

How well the risk estimates reflect the real risk is subject to a range of uncertainties not the least of which is the mathematical function used in calculating the risk. The relationship between the concentration of PM₁₀ in ambient air and the related adverse health effect can be described mathematically by a linear concentration-response function (CRF: also referred to as dose or exposure response in the scientific literature). The CRF is a value that is usually derived from epidemiological studies of an exposed population and relies on the population being large enough for the health effect to be discernible.

The CRF is also dependent on the potency of the PM which is related to the source of the PM and patterns of exposure. This means that multiple CRFs have been determined in the scientific literature, each for a health effect in question and location of interest. This can lead to disagreements among risk assessors on which CRF best describes the real relationship between exposure and effect. To overcome this risk, assessors attempt to match the CRFs to the studied population and locations. Nevertheless, the variability in the CRF models generally means that risk estimates may be over or indeed under estimated. Therefore, the reported risks should be interpreted with caution.

To provide some context for Port Hedland, specific CRFs for the Port Hedland population cannot be determined because the population is too small and the PM₁₀ potency is not yet understood; that is, it is not yet clear if crustal PM has different potencies to urban PM. There remains insufficient evidence in the literature to differentiate between the toxicity of the PM₁₀ in Port Hedland and the PM₁₀ from large urban centres. Under such circumstances the only option is to apply CRFs derived from a large population centre to estimate the health risks. The most meaningful CRFs are usually selected for the purpose. The CRFs used for Port Hedland were deemed to be appropriate by the peer reviewers. These are presented in Section 6 of the Toxikos Report with sample calculations in Appendix C.

Toxikos used the entire dataset for modelling the risk estimates and did not exclude extreme values due to bushfires in the region. This is consistent with current practice for assessments of this nature and recognises that peak events undoubtedly impact on population health. Although the practice is not to truncate data, some adjustment is necessary to exclude extreme values so that the data can be scaled down to assess different exposure scenarios; i.e. 70 µg/m³, 50 µg/m³ and 40 µg/m³. The adjustment technique should consider background dust levels otherwise the dataset and health outcomes may become unrealistic for the location. The 2013 PM₁₀ dataset was used for the Toxikos assessment which meant the background contribution was less critical than data from 2012 which contained the highest outliers. Nevertheless by not considering extreme background values any predicated health benefits from reducing PM₁₀ concentrations can be overstated or unrealistic and can create the impression that dust exceedances are amenable to control. Detail is lacking in the Toxikos Report on the influence of adjustment factors on health outcomes and on the limits placed on the accuracy of the predictions by using a single year of data.

Central to the issue within government is whether the interim guideline represents an 'acceptable' level of risk for PM exposure in Port Hedland. To answer this question, Toxikos modelled scenarios that considered the health outcomes of applying the lower 24-hour PM₁₀ guideline levels of 50 µg/m³ (NEPM) and 40 µg/m³ (the proposed new NEPM guideline). Toxikos concluded the level of risk between the interim guideline of 70 µg/m³ and the NEPM

was not discernible for the current population level in Port Hedland. According to the scenario that modelled future population growth, health impacts may become discernible after the population more than doubles, assuming that air quality does not change (Table 13 in the Toxikos Report).

A key issue for some community members is how the risks in Port Hedland compare to those in Perth. Risks are usually reported per 100,000 rather than for the current population. This is a standardised population size commonly used to compare health outcomes between areas that have different population numbers. To the casual reader the numbers of people affected when risk estimates are presented in this way may be alarming and give the wrong impression that a risk is immediate because of the large numbers quoted. It is important to recognise that the actual number of individuals involved for the population under study may indeed be very small as is the case in Port Hedland. Various limitations are associated with deriving risk estimates, some of these are discussed in Section 13 and Appendix A of the Toxikos Report. Important limitations relevant to Port Hedland include the small population studied and the small health dataset available for the region. Therefore using the derived health outcomes for Port Hedland to compare with Perth or other large urban cities may not be useful. Nevertheless, it is important to acknowledge that the risks are not excessive or urgent, precisely because the population is small and health adverse effects are not readily discernible among a small population.

The Toxikos exposure assessment was unable to determine relative risk attributable to crustal coarse particles separately from those attributable to PM₁₀ more generally. This is not surprising as the relative toxicity of crustal particles compared to non-crustal particles is a matter that is still debated. Neither was it possible to determine whether salt has an independent health effect. Both of these issues are well considered in the Toxikos Report.

7.1. SUMMARY

- The risk estimates reported come with a range of uncertainties. Nevertheless, overall there is sufficient evidence to demonstrate that concentrations of PM₁₀ over 70 µg/m³ are associated with increased morbidity and mortality. This is clearly supported by the literature that increasing concentrations of PM₁₀ at any level is associated with increased levels of acute and chronic respiratory and cardiovascular health effects.
- There remains insufficient evidence in the literature to differentiate between the toxicity of the PM₁₀ in Port Hedland and the PM₁₀ from large urban centres. Therefore, CRFs from large urban centres was the only option available to assess population exposure.
- The PM in the West End is richer in iron-oxide and manganese but both of these are below their respective health based guidelines (see next section), therefore there is justification for treating all PM across Port Hedland as equally potent based on mass.
- The risk of being adversely affected by the airborne PM₁₀ levels increases with proximity to the harbour. In the West End of Port Hedland the risk may be up to twice as high than for those living in South Hedland but the actual numbers affected are very small because the population is very small.

- Modelling scenarios of the lower 24-hour PM₁₀ concentrations of 50 µg/m³ and 40 µg/m³ indicated the level of risk between the interim guideline of 70 µg/m³ and the NEPM was not discernible for the current population level in Port Hedland. Therefore the interim guideline of 70 µg/m³ should provide adequate protection of health and wellbeing.
- Air-quality modelling would assist to define areas in Port Hedland that could meet the interim guideline. It appears that areas as far West as Kingsmill Street could meet the interim guideline.
- Further dust reduction may not be possible in the West End so exposure control could be achieved by controlling population growth and land-use.

8. PM_{2.5} AND OTHER CONTAMINANTS OF CONCERN

PM_{2.5} concentrations fluctuated daily at each monitoring location. Figure 8 provides an example of fluctuations associated with impacts of bushfire smoke in the region. Twenty two exceedances recorded during the study period were associated with days that reported bushfires in the region between October and December 2012 and coincided with the extreme fluctuations in PM₁₀ for the same period. The maximum average exceedance above the NEPM for PM_{2.5} was 54 µg/m³ compared to the average on other days of 8 µg/m³. The otherwise low concentration levels of PM_{2.5} reflect the low combustion sources in Port Hedland and the dominance of coarse particles (PM₁₀) in the dust. The annual NEPM of 8 µg/m³ was exceeded at Kingsmill Street which possibly reflects the higher rate of data recovery at this location and 5 exceedances recorded during bushfire days but independent of exceedances at other locations. Since the NEPM (24-hour average of 25 µg/m³) for PM_{2.5} was met at all other times, it is doubtful that the impacts of these extreme events would be discernible against background rates of relevant health indicators. Toxikos reports that this is indeed the case. Given that exceedances of PM_{2.5} can be largely explained by bushfire impact in the area and that these are not amenable to local controls PM_{2.5} exposure is not discussed further in this assessment.

Some specific concerns were expressed by government about exposure to metals in ambient air, particularly iron oxide. The air monitoring results for all monitored metals indicate that exposure to these metals in ambient air in the Port Hedland is unlikely to pose any risk to human health. The risk characterisation method employed by Toxikos is typical and consistent with current practice. The assumptions are conservative and therefore the risks are considered negligible. The same can be concluded for both silica and asbestos.

The most notable finding was the clear decrease in ambient concentrations of iron, copper and manganese with increasing distance from the port monitor (Figures 12 – 14 in the Toxikos Report). For example ambient air levels of manganese were 60% of the relevant standard at Richardson Street compared to 20% at Taplin St, 10% at South Hedland and 7% at Yule. The maximum ambient air levels of copper were below 1% of the relevant standard and iron below 8% of the relevant standard at Richardson Street and decreased with distance from the port monitor. The impact of the Nelson Point and Finucane Island operations is clearly demonstrated in this analysis.

The implication of the manganese concentration at Richardson Street is that while the standard has not been breached the concentration is close to the standard and manganese concentrations in ambient air should be monitored in the community so that any exceedances in the future are quickly detected and adequately managed.

9. CONCLUSIONS AND RISK MANAGEMENT

There is sufficient evidence provided in this assessment and the Toxikos Report of potential impacts on human health from dust to warrant the development of a strategic plan to reduce community exposure to dust.

The Toxikos HRA together with this assessment describes the magnitude of the public health burden of dust pollution in Port Hedland. The burden is associated with PM₁₀ concentrations over 70 µg/m³. The actual numbers of individuals affected (Table 12 in the Toxikos Report) are low but only because the population is small. If the population was larger or was permitted to become larger, the health outcomes would be more readily discernible and demanding of more urgent regulatory control on dust sources.

In terms of 'risk', the literature is clear that any increase in PM above a baseline level contributes to greater risk (because near linear exposure-response is assumed) in developing or worsening of respiratory and cardiovascular symptoms. Therefore theoretically it could be said that the risk is greater in areas with higher PM, such as Port Hedland relative to areas with lower PM such as South Hedland. The interim guideline provides a useful measure to differentiate between areas of higher and lesser risk.

A significant aspect of the Toxikos HRA was to determine whether the interim guideline should continue to apply East of Taplin Street which is the boundary of the West End, or whether the West End area should be redefined and a new guideline developed for the West End. Since the areas closest to Richardson Street and Kingsmill Street are affected by PM of different characteristics to PM further east, it was conceivable that a new guideline might be developed for this area either on the basis of composition or exceedance patterns (excluding natural events). This does not appear to be feasible on the basis of composition because the toxicity of the different PM captured in the West End while rich in metals, did not contribute to health effects above those attributed to PM mass.

On the other hand a new guideline based on exceedance patterns may be feasible. This assessment indicates that dust exceedances follow a seasonal pattern and that during certain seasons the meteorological conditions are such that fugitive dust escaping the Nelson Point and Finucane operations may be inevitable. A new guideline for the West End would be higher than the interim guideline and would only be recommended for commercial areas that excluded permanent residences. The new guideline would provide an air-quality buffer between the port, Nelson Point operations and the more permanent residential areas where it would be expected that the interim guideline would be met. The air quality model would help to define the extent of this area; the pattern of exceedances indicates that the areas West of Kingsmill Street / Darlot Street may be appropriate, assuming the dust is managed at the spoil bank.

The monitoring data confirms that the interim guideline identified by the Taskforce has been exceeded in the West End and that industry contributes to the higher dust concentrations before bushfires. Industry has a responsibility to implement best practice dust emission controls to minimise the distribution and impact of dust in Port Hedland however targeting industry alone would ignore broader dust management and exposure reduction opportunities. These include managing dust from the spoil bank and other small eroded open areas (supported by the Wedgefield data) and the exclusion of permanent residences from the West End.

This assessment and the Toxikos HRA identified that air-quality modelling is important to identify areas of lower and higher PM₁₀ distribution so that the West End can be redefined if necessary.

10. RECOMMENDATIONS

As stated earlier in this assessment, risk management is a separate process to that of the analytical assessment of risk however the recommendations provided below may assist in scoping dust mitigation criteria.

While the risks are not urgent, ongoing expansion of the port and associated Nelson Point and Finucane Island operations is expected any additional emission sources in the future will require careful management. The close proximity to residential areas means that increased activity and further expansion is of concern to both the community and industry. Further expansion will place additional pressure on dust management initiatives by industry and the government.

Recommendations:

a) Guideline and Exposure Reduction –

- Introduce exposure reduction measures that include capping the number of permanent residents to current numbers in areas most impacted by dust currently to the west of Taplin Street. Because acceptable risk is based on population size, a strategy must be introduced now to manage and restrict future population growth in Port Hedland. The closer to the port and Nelson Point operations the tighter the restrictions. A long-term land-use planning strategy may offer a tool for gradually moving the residential areas away from the operations area. Since the aim of government is not to disadvantage anyone currently living in the area planning tools such as amendment 22 may offer a means to manage exposure while also managing population growth.
- Current regulatory controls for managing dust from operations at Nelson Point and Finucane Island may be aided by declaring a buffer between the port and residential areas further to the east. Air-quality modelling can help define this area but should not be the sole decision making tool used for determining the buffer boundary.
- Apply the current interim guideline of 24-hour PM₁₀ of 70 µg/m³ (+ 10 exceedances to accommodate natural events) in residential areas of Port Hedland within a reasonable time frame that allows for local dust sources to

be identified and managed (i.e. the spoil bank). A period of 5 years is suggested.

- The interim guideline can be applied to South Hedland and Wedgefield but it may also be possible to achieve the NEPM in South Hedland if the source of local exceedances can be identified and managed.
- A coordinated approach to reduce dust from all sources not just industry is required. Various government sectors (planning, transport, energy) may be needed to develop and effectively develop and implement long-term policies and strategies that reduce exposure.

b) Air quality monitoring –

- An ongoing air quality monitoring program is vital to monitor exposure risk. This program should include the criteria NEPM pollutants (minus lead) and manganese. The program should have the capacity to include additional pollutants as indicated by the development of new industries or changes to existing industries.
- Exceedances of the interim guideline should be investigated and reported to DER.

c) Impact assessments for new developments and future expansion of existing industry should include baseline air quality data and consider additional impacts on air quality on the Port Hedland air-shed.

d) Promote and encourage existing efforts at continuous improvement among stakeholders. Even small reductions in overall PM can have incremental benefits which, at least theoretically, contribute to improved amenity, reduced potential health risks and improved health status on a population basis.

e) Assist local government to promote a community awareness of the benefits of reducing exposure to PM overall. This may include ways to reduce personal exposure during extreme events.

f) Promote an all of government support for further research on the health effects of crustal dust and the importance of exposure reduction.

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Report

REPORT

HEALTH RISK ASSESSMENT PORT HEDLAND

WA Department of Health

Job No: 08083

20 July 2015

PROJECT TITLE: Health Risk Assessment Port Hedland

JOB NUMBER: 08083

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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
OEHA	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).

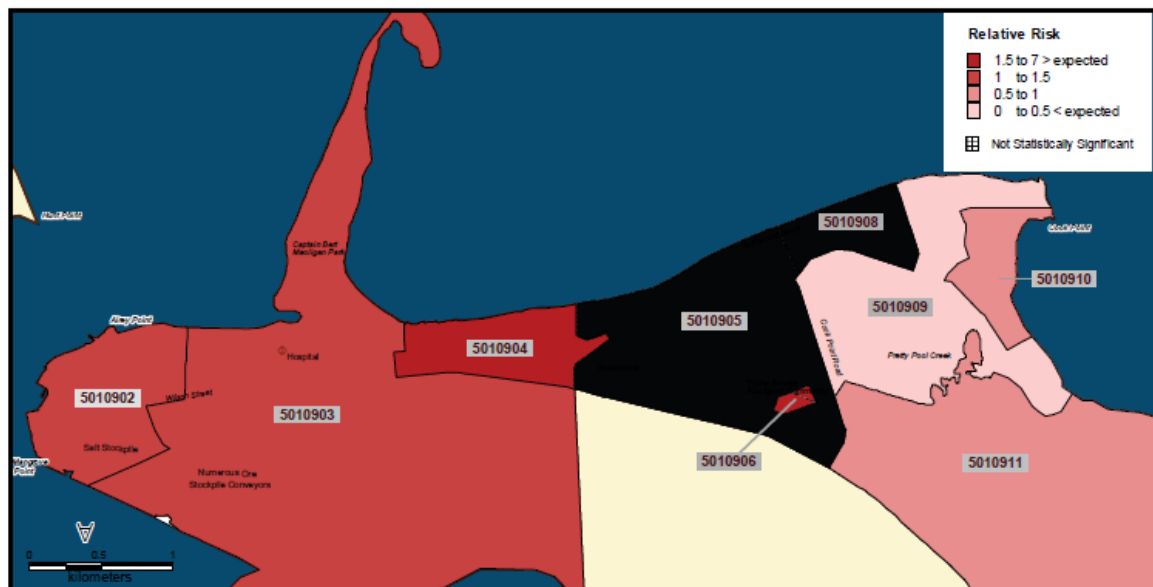
Previous research had shown that the hospital admission rate for the population in remote Western Australia was higher than that of the population of the state (Somerford, 1995). The results also showed that the rate of respiratory admissions for the Shire of Port Hedland as a whole is significantly 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).

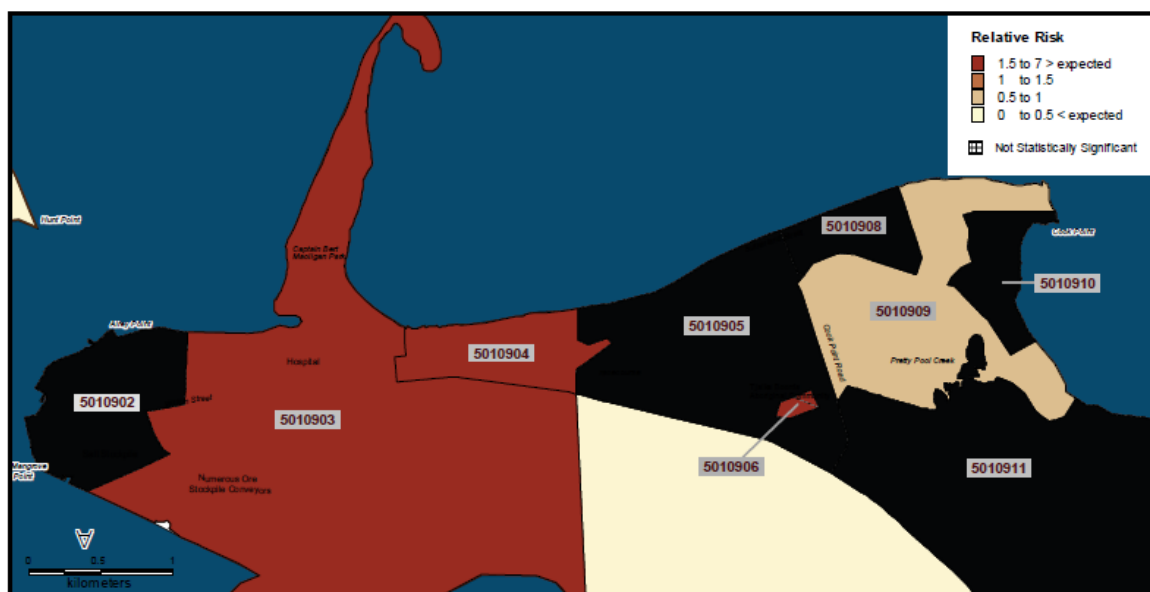
Figure 1 and **Figure 2** (reproduced from the WA DoH 2006 report) show the variation in relative risks for hospital admissions for respiratory diseases (**Figure 1**) and cardiovascular diseases (**Figure 2**).

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

^a In 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).

^b This 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	

^a HRAM – 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 pneumoconiosis 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), OEHA 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 OEHA

Based on the combined studies from Gleason, 1968 and Whitman, 1957; 1962, OEHA 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 OEHA. 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 OEHA 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).

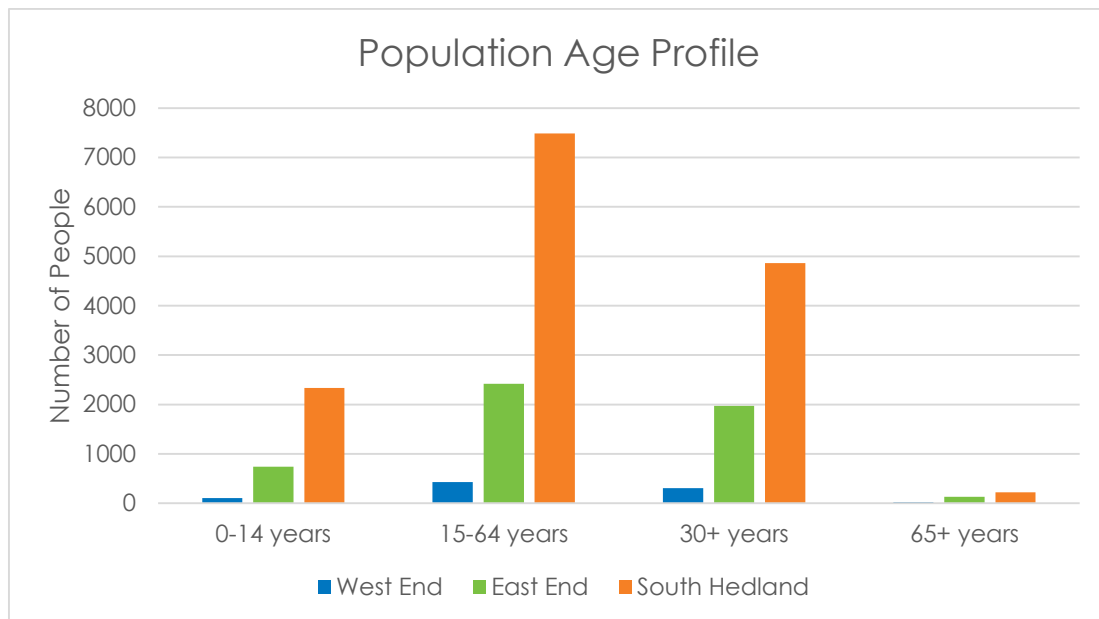
For the permanent population data was available for Port Hedland as a whole, the West End (west of Taplin St), the East End (east of Taplin St) and South Hedland. **Table 3** summarises the population data as a whole compared with the Western Australia average.

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 10µg/m³ 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 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; 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 10µg/m³ 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 48µg/m³.

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 46.1µg/m³. 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 64.5 µg/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 60.8 µg/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 10 µg/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 PM_{10-2.5} and PM₁₀ among females (RR = 1.38 [95% CI: 0.97–1.95] per 10 µg/m³ PM_{10-2.5}; RR = 1.22 [95% CI: 1.01–1.47] per 10 µg/m³ PM₁₀), but not for males (RR = 0.92 [95% CI: 0.66–1.29] per 10 µg/m³ PM_{10-2.5}; RR = 0.94 [95% CI: 0.82–1.08] per 10 µg/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. PM_{10} was associated with decline in FEV_1 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 FEV_1/FVC ratio pre-bronchodilator for recent $PM_{2.5}$, and recent PM_{10} 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 PM_{10}), 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, PM_{10} and NO_2 (Zemp *et al.*, 1999). These associations were strongest for PM_{10} . The investigators estimated that an increase of $10\mu g/m^3$ PM_{10} (within the observed range across cities of $10.1 - 33.4\mu g/m^3$), 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 PM_{10} versus NO_2 in the observed associations could not be ascertained, as NO_2 concentrations were strongly correlated with PM_{10} levels.

The SAPALDIA investigators also examined lung function (FEV_1 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 PM_{10} , sulfur dioxide, and nitrogen dioxide, with the strongest effects being related to PM_{10} (-3.4% for FVC and -1.6% for FEV_1 in healthy never-smokers, per $10\mu g/m^3$ annual average PM_{10}). The mean PM_{10} concentration in this study was $21.2\mu g/m^3$, with a range of $10.1 - 33.4\mu g/m^3$.

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 studies 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 $10\mu g/m^3$ 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 $10\mu g/m^3$). 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 $PM_{2.5}$ ($29.9\mu g/m^3$) and 1.1 times higher for $PM_{2.5-10}$ ($16.4\mu g/m^3$) 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 $10\mu g/m^3$ 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 $10\mu g/m^3$ increase at lag 1 day. When both NO_2 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 $10\mu g/m^3$ 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 NO_2 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, PM_{10} , SO_4^{2-} , 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 $10\mu g/m^3$), 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 $6.3\mu g/m^3$ 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 $10\mu g/m^3$ 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 $10.9\mu g/m^3$ 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 PM_{10} 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 $157\mu g/m^3$) 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 $89\mu g/m^3$ mass median aerodynamic diameter (MMAD) $3.9\mu m$, 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 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 400-500 $\mu\text{g}/\text{kg}$) 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 mg/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, exotoxin, 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 Happon 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- α 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/m ³ increase in PM ₁₀
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 sight 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 NOHSC:3003 (2005) <i>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 PM_{10} 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 PM_{10} standard of $50 \mu g/m^3$ (between 9 and 74 days in 2013) and the interim standard adopted by the Port Hedland Taskforce of $70 \mu g/m^3$ (between 2 and 16 days in 2013). Table 8 shows the summary statistics for the PM_{10} data including the number of exceedances at each monitorin location. The highest levels of PM_{10} 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

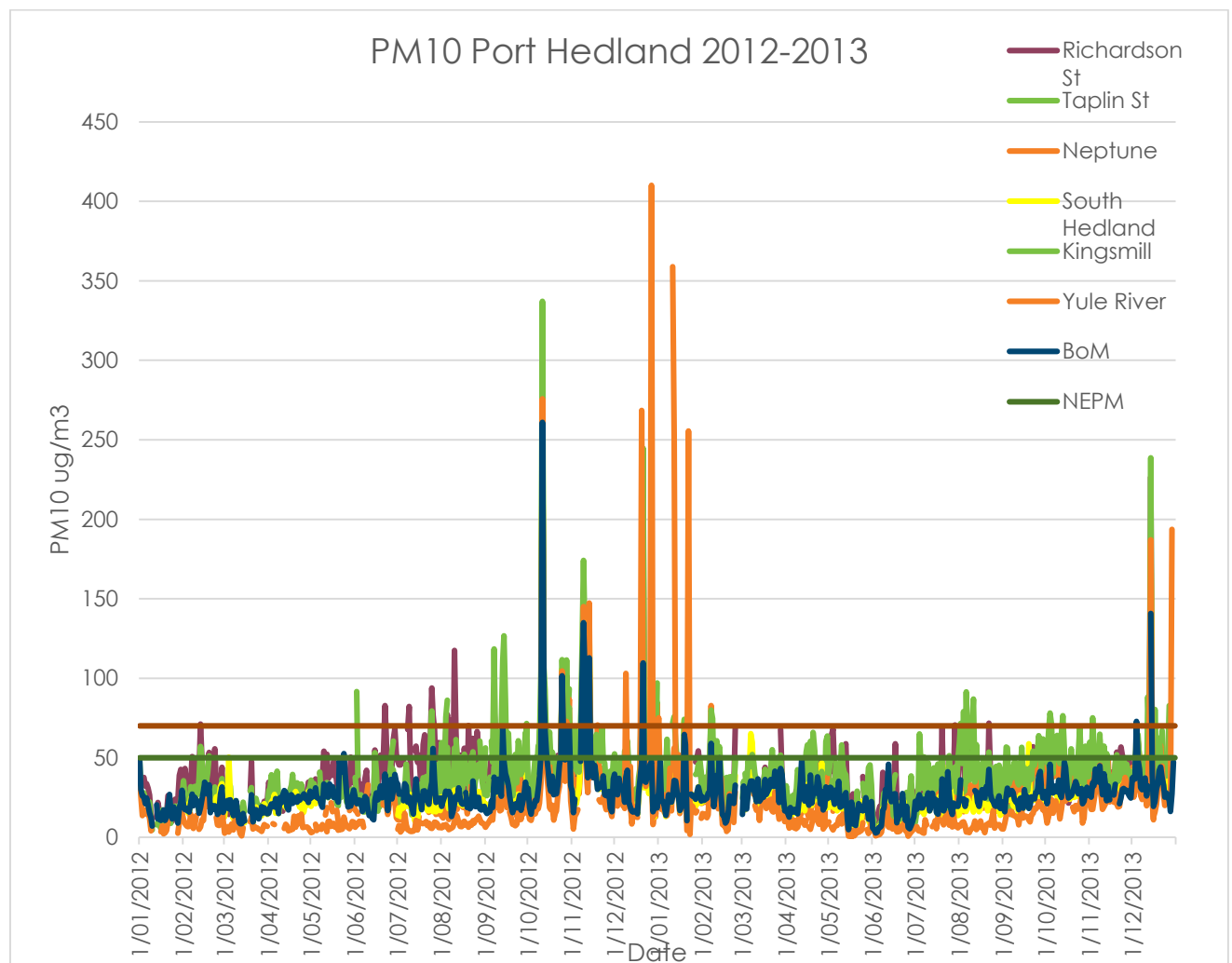
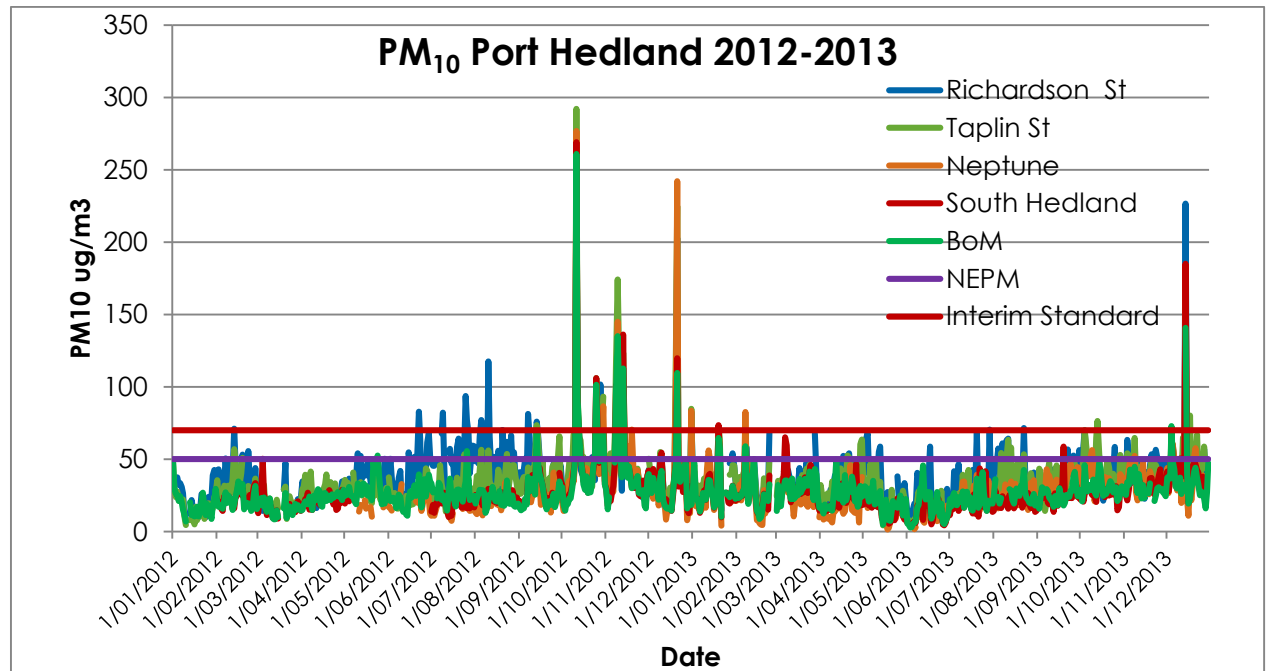


Figure 6 shows the PM₁₀ data for the Port Hedland sites only (minus Yule River). Examination of the PM₁₀ data for the Port Hedland sites only (minus Yule River) shows that there are numerous exceedances of both the NEPM and interim taskforce guideline at all sites during the monitoring period (see Table 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

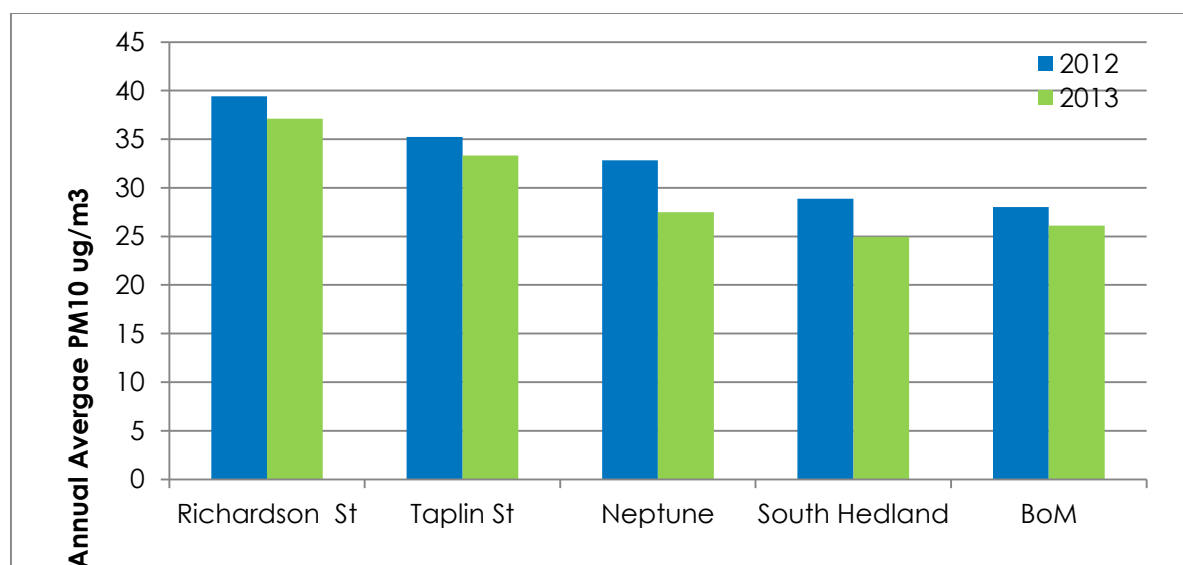
seen in **Figure 6**. What this indicates is that there is a source between South Hedland and the Richardson and Taplin St sites that is being transported by winds from the S-SE. If this was regional dust it would be seen across all sites.

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.

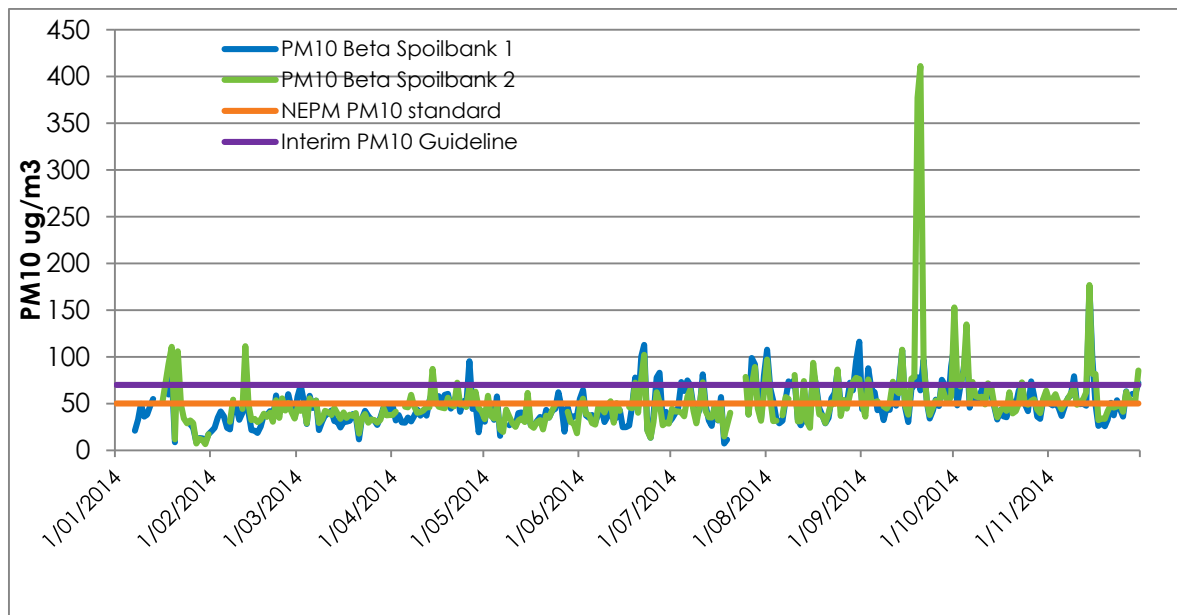
Figure 8: Location of Landcorp PM₁₀ Spoilbank monitoring locations.



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

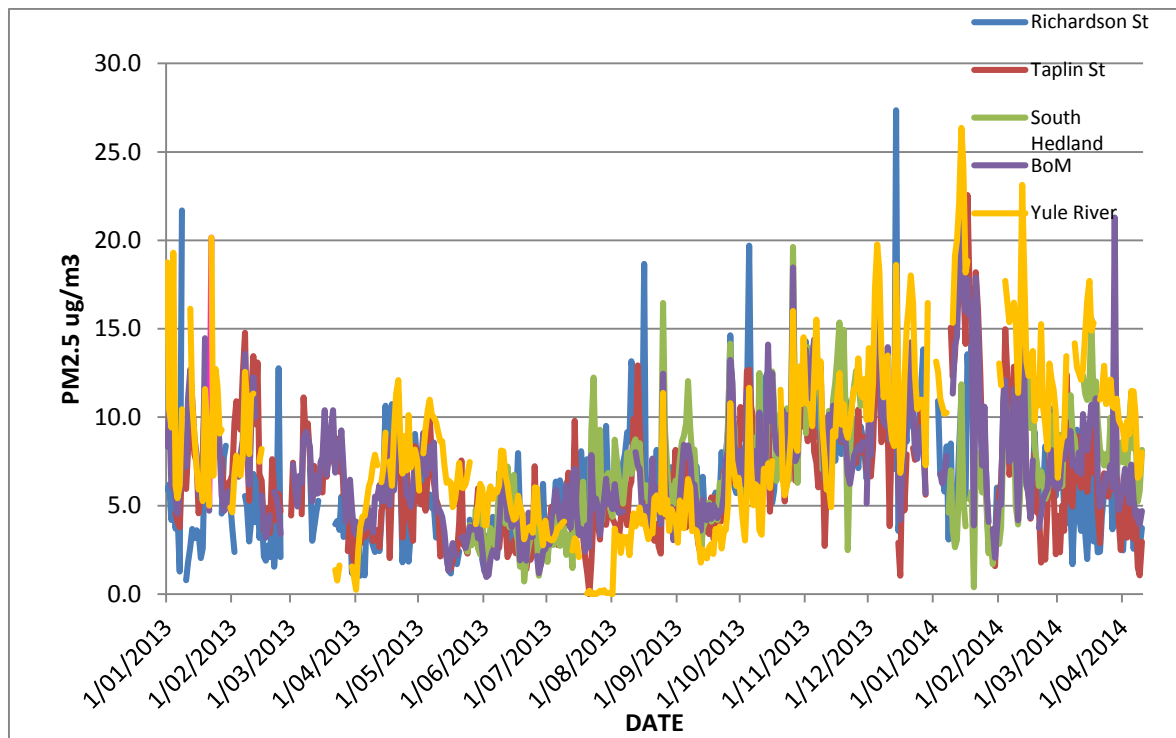
Figure 9 there are numerous exceedances (118 days during the monitoring period) of both the current NEPM standard of $50 \mu\text{g}/\text{m}^3$ and the interim guideline value of $70 \mu\text{g}/\text{m}^3$. In general the data at the two sites are similar although the data from Spoilbank 2 site is 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 $411 \mu\text{g}/\text{m}^3$.

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.

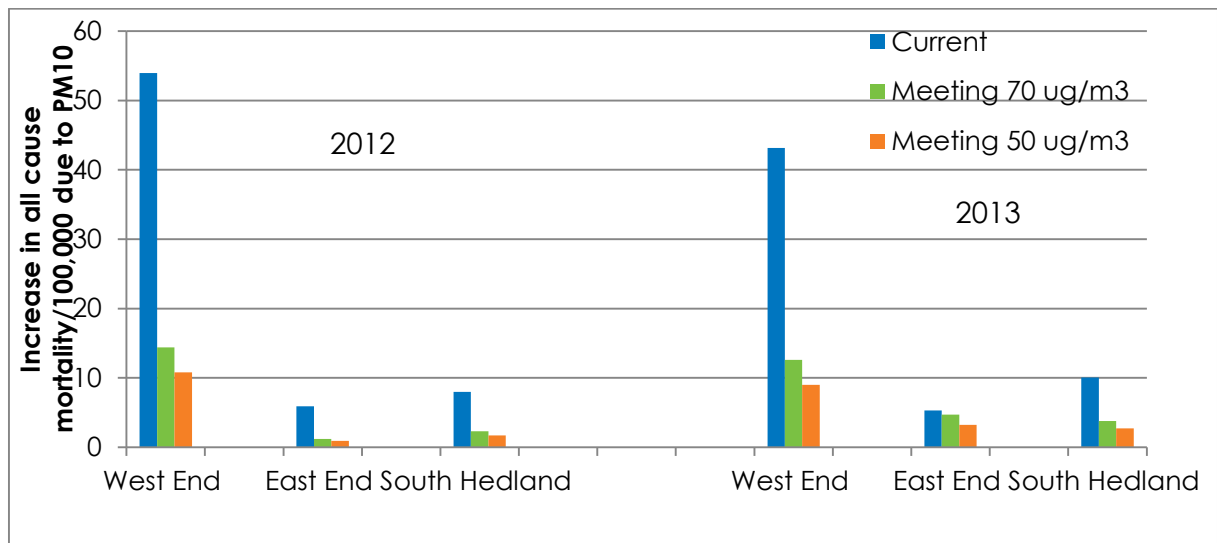
Table 13: Annual Health Outcomes Avoided on Meeting Alternative Standards

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
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				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 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 $\mu\text{g Mn}/\text{m}^3$. 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 $\mu\text{g}/\text{m}^3$ cut-off point. When using a 0.1 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{g}$, 9.5vs. 8.0 $\mu\text{g}/\text{L}$ 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 pneumoconiosis 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 due 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 bronchiolo-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 OEHA (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

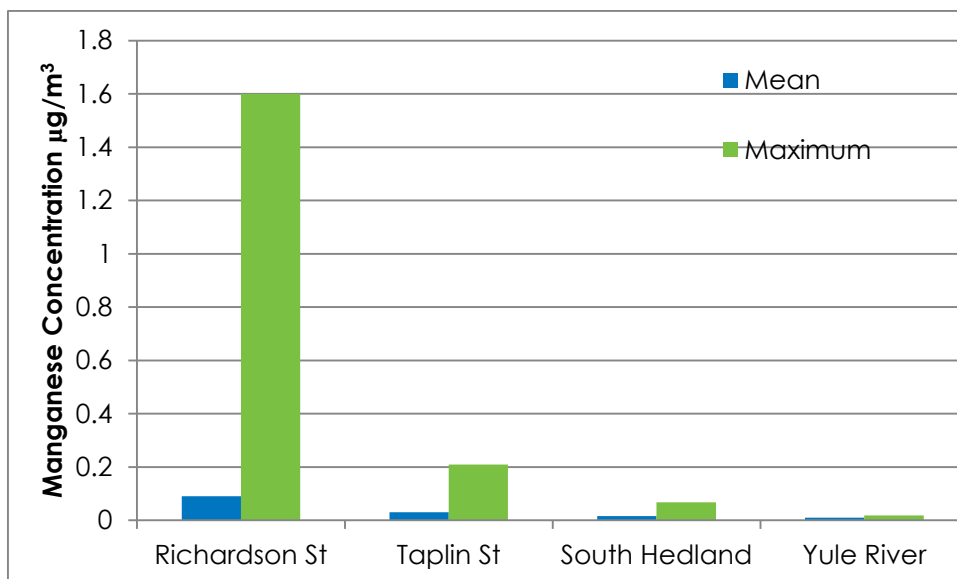
Summary Statistic	Dataset ($\mu\text{g}/\text{m}^3$)				
	2011	2012	2013	ChemCentre (Oct 2012-May 2014)	
				PM ₁₀	PM _{2.5}
Mean	Not sampled	1.49E+00	3.48E-01	5.28E-01	2.39E-01
90 th Percentile		2.46E+00	7.49E-01	8.24E-01	5.22E-01
95 th Percentile		4.98E+00	7.70E-01	1.05E+00	5.66E-01
Maximum		7.50E+00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

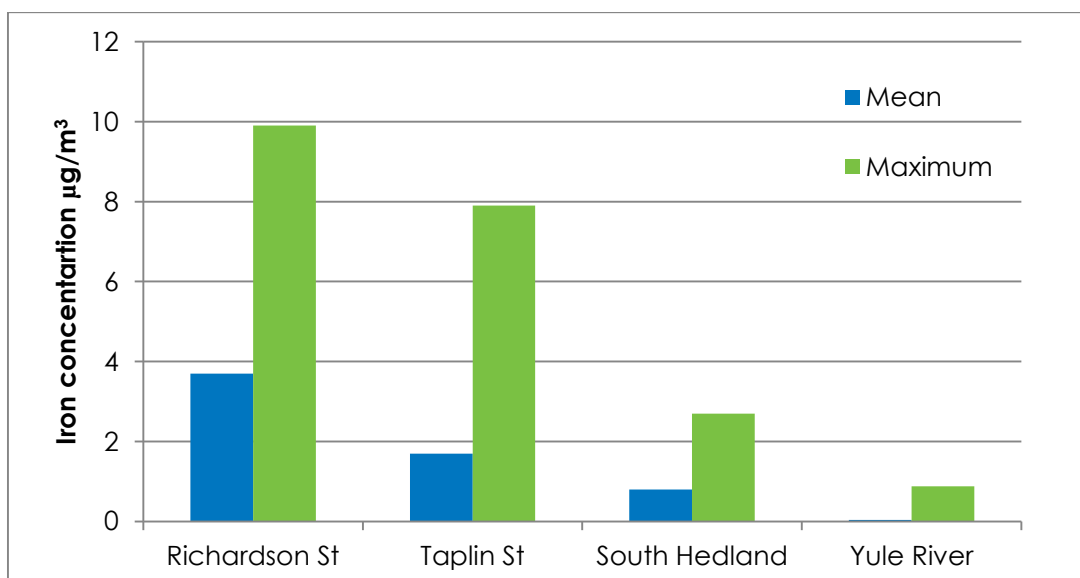
Comparison of mean and maximum values for each of these metals for all sites for 2013 (the most complete data set) is shown in **Figure 14**.

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



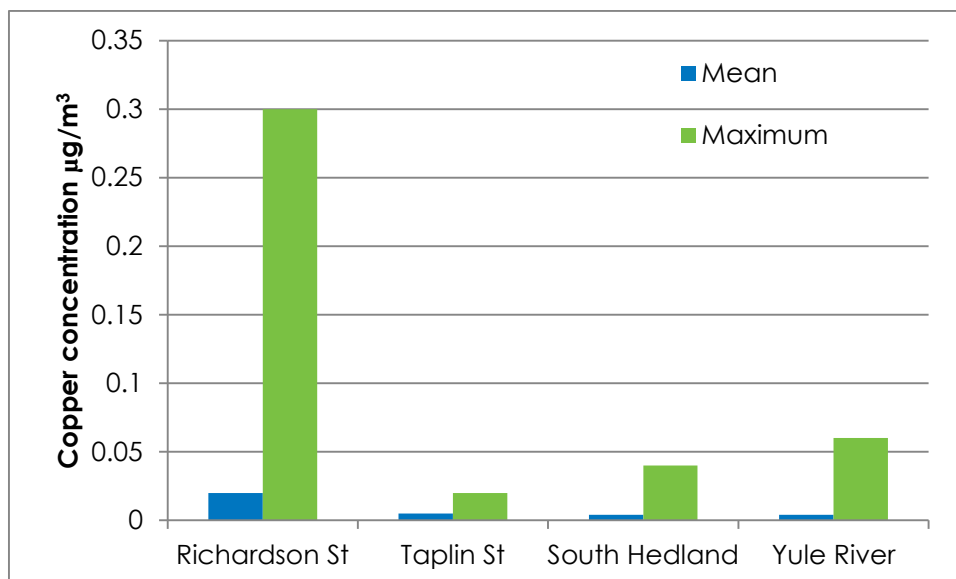
The data shown in Figure 13 shows a clear impact of Port operations on manganese levels at both Richardson St with much lower levels at Taplin St, South Hedland and Yule River. Manganese is stored at both the Port and Finucane Island.

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):

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

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

	HAZARD QUOTIENT		
RICHARDSON ST	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.

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

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 OEHA 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 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 lung function.

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 (0.026–0.040 ppm 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.

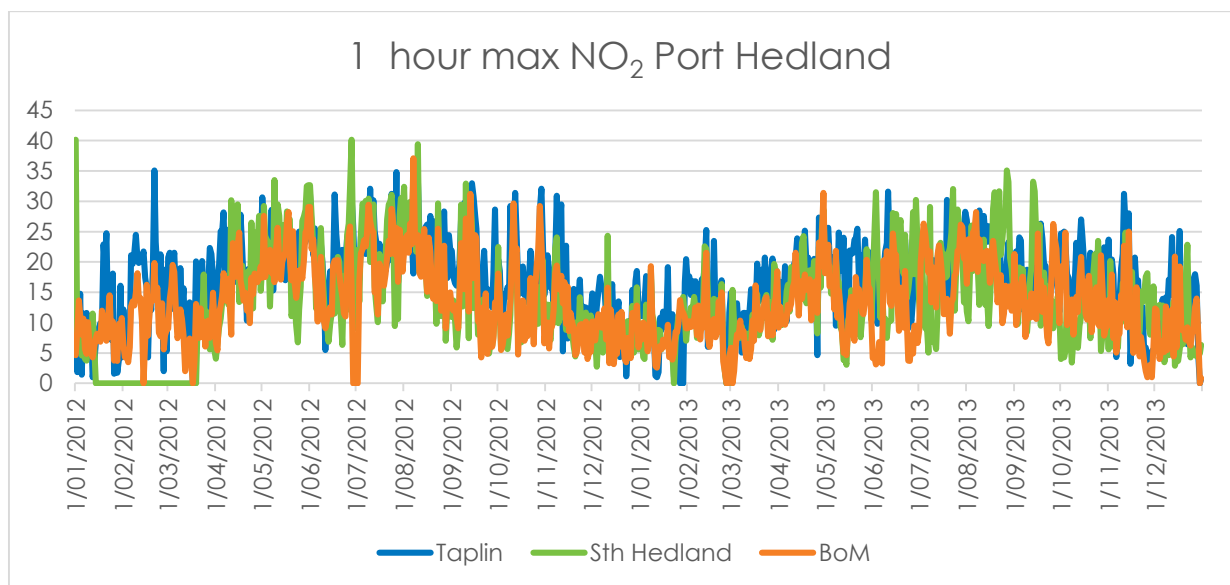
The results from several large U.S. and European multi-city studies and a meta-analysis study observed positive associations between short-term ambient NO₂ concentrations and risk of all-cause (non-accidental) mortality, with effect estimates ranging from 0.5 to 3.6% excess risk in mortality per standardized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

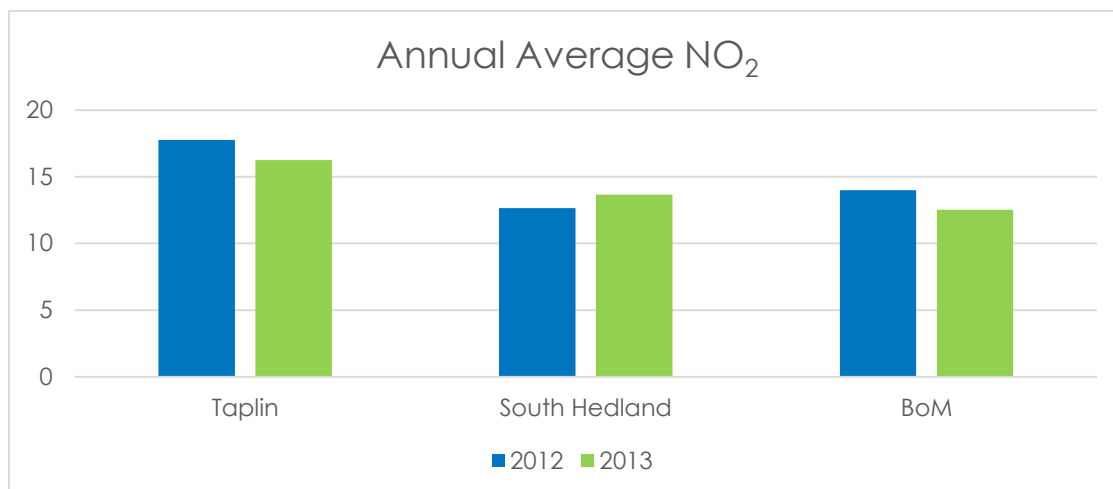
Monitoring for NO₂ was undertaken by PHIC at Taplin St, South Hedland and BoM for the period 2012-2014. **Figure 15** and **Figure 16** show the daily 1-hour maximum and annual average NO₂ levels for 2012-13 which are the most complete datasets. The data shown in **Figure 15** show that for 1-hour maximum 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.

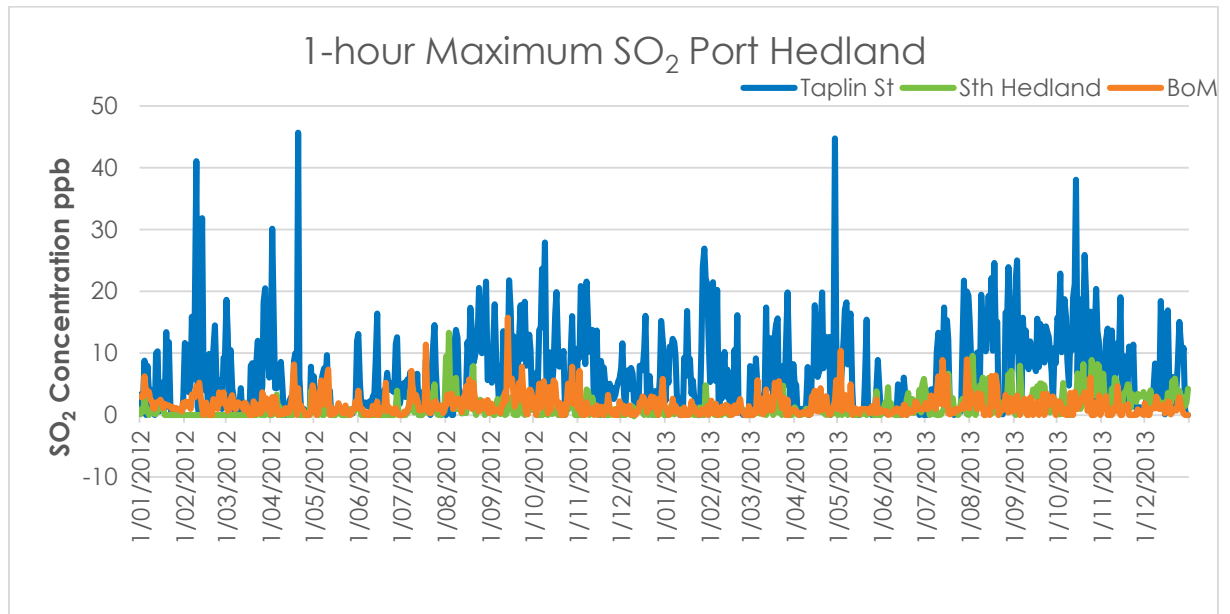
The studies reviewed indicate that short-term exposures of 5-15 minutes to sulfur dioxide are associated with a dose-response effect on lung function of exercising individuals with asthma. The controlled exposure study by Linn et al (1987) in exercising individuals with asthma is indicative of a LOAEL 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

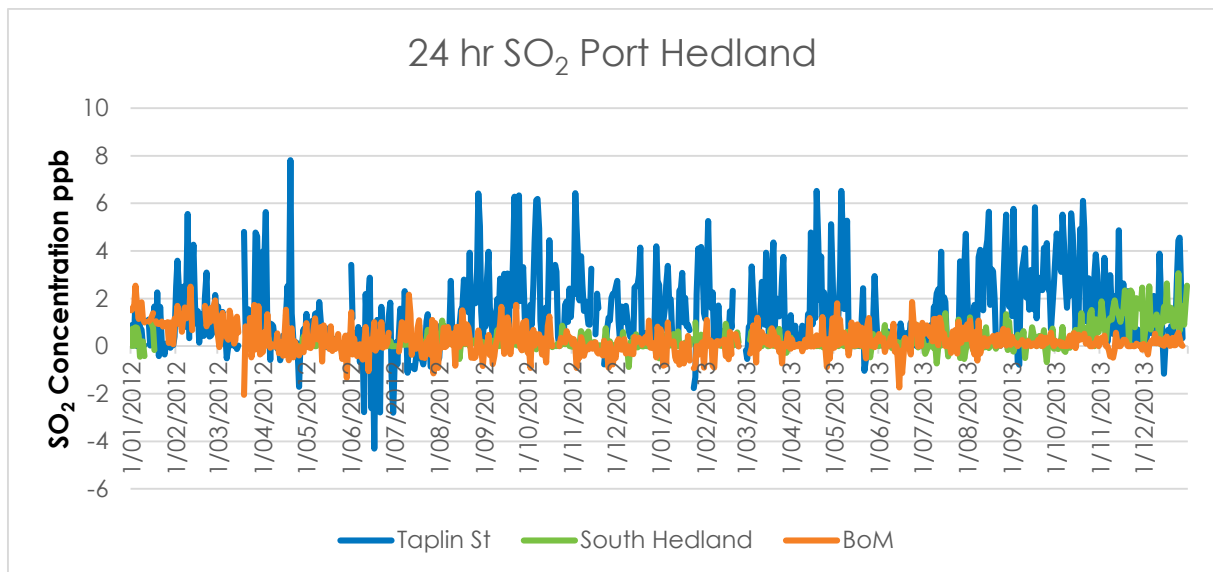
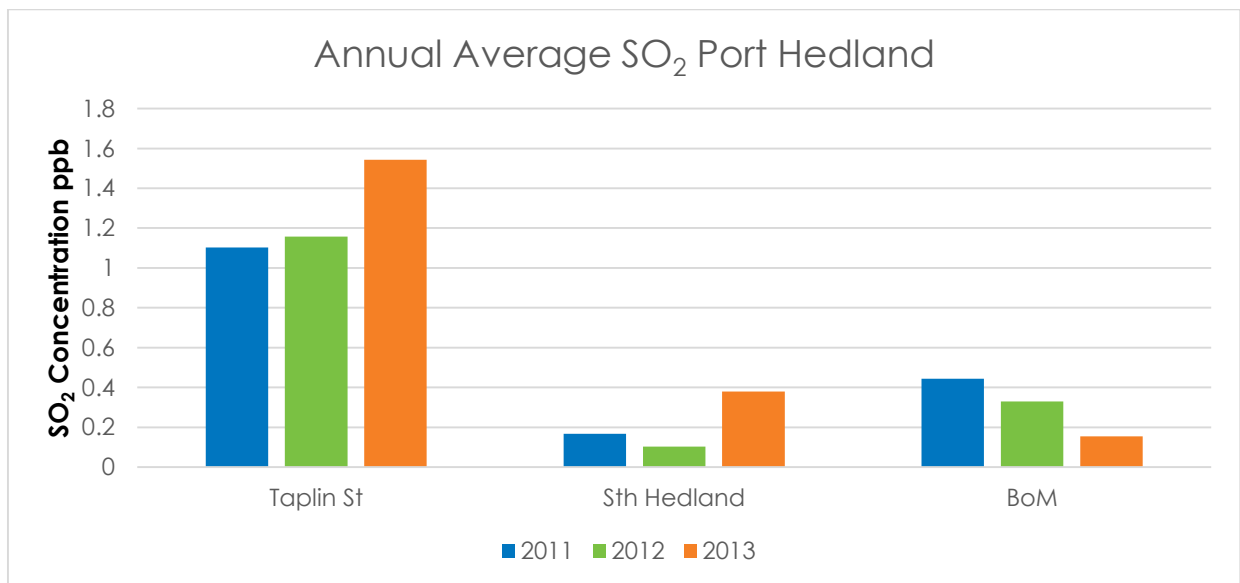


Figure 19: Annual Average SO₂ Levels



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 percentile? 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 HRA. 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:

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

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:

$$\text{Increase in annual all-cause mortality} = \frac{\text{exposure response function} \times \text{annual average PM}_{10} \times \text{baseline incidence rate}}{100,000 \text{ population}}$$

$$= 0.004 \times 37.1 \times 196$$

$$= 29/100,000$$

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:

$$\text{Increase in daily all-cause mortality} = \frac{\text{exposure response function} \times \text{daily average PM}_{10} \times \text{baseline incidence rate}}{100,000 \text{ population}}$$

$$= 0.002 \times 25 \times 0.54$$

$$= 0.027/100,000$$

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 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ x daily average $\text{PM}_{2.5}$ 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 NO_2

For the increase in risk due to NO_2 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 NO_2 value was $11.8 \mu\text{g}/\text{m}^3$. The exposure response function for daily hospital admissions for respiratory disease 15-64 years of age from Table 19 is 0.0002 increase per $1 \mu\text{g}/\text{m}^3$ increase in NO_2 . 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 $\mu\text{g}/\text{m}^3$ increase in NO_2 x daily 1-hour maximum NO_2 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 $9.9 \mu\text{g}/\text{m}^3$. The 24-hour average TRV adopted for the HRA from Table 2 is $120 \mu\text{g}/\text{m}^3$. Therefore the resulting hazard index is:

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

$$= 9.9/120$$

$$= 0.08$$

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.

APPENDIX 2

Port Hedland Dust Management Taskforce Report to Government

**(Department of Jobs, Tourism, Science and Innovation,
August 2016)**



Government of **Western Australia**
Department of **State Development**

PORT HEDLAND DUST MANAGEMENT TASKFORCE REPORT TO GOVERNMENT

August 2016

ADDENDUM

Machinery of Government Changes, 2017

In 2017, the Government initiated changes to State Government Departments as part of its Machinery of Government process. The process created a number of new amalgamated departments, affecting some of the departments participating in the Port Hedland Dust Management Taskforce and referenced in this report. The changes to the participating departments, effective from 1 July 2017, are as below.

Department of Environment Regulation (DER)

Office of the Environmental Protection Authority (OEPA)

The Department of Environment Regulation (DER) and the Office of the Environmental Protection Authority (OEPA) were amalgamated with the Department of Water to form the **Department of Water and Environmental Regulation (DWER)**.

Department of State Development (DSD)

The Department of State Development (DSD) was amalgamated with the Industry Development division of the former Department of Commerce; the Western Australian Tourism Commission; and the former Office of Science to form the **Department of Jobs, Tourism, Science and Innovation (JTSI)**.

Department of Planning (DoP)

The Department of Planning (DoP) was amalgamated with the Department of Lands and the State Heritage Office to form the **Department of Planning, Lands and Heritage (DPLH)**.

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1. INTRODUCTION

1.1 OVERVIEW

Port Hedland is the largest bulk handling port in the world, and makes a major contribution to the Western Australian and Australian economies. In 2014-15, the value of exports through the port was almost \$30 billion, accounting for 27% of Western Australia's merchandise exports and 12% of Australia's. Its users generate about half of the Western Australian Government's royalty income - \$2.4 billion in 2014-15.

Export volumes through the port have risen rapidly in recent years, in response to strong demand in China for iron ore. Iron ore exports rose from 167 million tonnes in 2010 to 424 million tonnes in 2015.

Rising exports have helped to support broader growth in the Port Hedland. The estimated residential population increased from 12,800 to 16,400 between 2001 and 2015. Social and economic infrastructure have improved, including the upgrade of recreational facilities (such as the South Hedland Aquatic Centre), the South Hedland Town Centre Revitalisation Project and major road upgrades linking Port Hedland and South Hedland. The Port Hedland Council and residents aspire to further population growth and development of economic infrastructure and social amenity.

The town and port developed in an era before modern planning principles on buffers and the separation of industrial, residential and commercial activity, and the health risks of exposure to dust were not well known. The port's industrial facilities are close to homes and businesses in Port Hedland's West End. Areas close to the port sometimes record elevated dust levels due to port activity.

In 2009, the Environmental Protection Authority raised concerns about the possible effects of dust on the health of Port Hedland residents, prompted by reports of high dust levels, mounting scientific evidence that high levels of dust concentrations can harm human health, and concerns that projected growth in throughput at the port could result in dust emissions rising.

In 2009, the Premier established the Port Hedland Dust Management Taskforce to co-ordinate and plan for dust management in Port Hedland. The Taskforce produced the *Port Hedland Air Quality and Noise Management Plan*, which included interim measures to limit exposure to dust by groups considered most at risk of health effects.

The Taskforce concluded that it needed three sets of data to make final recommendations on how to address dust issues – information on dust

levels and sources across Port Hedland; projections of how dust levels might change as the port developed; and information on the health effects of the types of dust found in Port Hedland. In response, the Port Hedland Industries Council established, and now maintains, a network of dust monitors in and around Port Hedland to collect data on dust types and levels. The Port Hedland Industries Council and the Department of Environment Regulation commissioned modelling to improve understanding of dust impacts arising from port developments. The Department of Health commissioned a Health Risk Assessment to evaluate the effects on human health of the types of dust found in Port Hedland.

All of these studies are now complete, and the Taskforce is in a position to make final recommendations to Government.

The Health Risk Assessment concluded that there is sufficient evidence of possible negative effects on human health from dust to warrant dust management controls and land-use planning measures to reduce community exposure to dust. Air quality monitoring data indicates that dust levels regularly exceed the Taskforce's interim guideline levels in the far West End (Richardson Street). Annual exceedances of the interim guideline at both Taplin Street and Neptune Street are around the recommended level of ten a year.

Dust levels have remained relatively stable in recent years, despite much higher export tonnages through the port. While port users have made substantial progress in reducing the intensity of dust emissions, these measures have not been enough to reduce dust levels to acceptable levels in the West End. If dust levels increase further, they will affect areas further east.

In reaching its recommendations, the Taskforce considered the conflicting and pressing priorities of different groups and interests. Residents are concerned that exposure to dust may affect their health. Property owners are concerned that planning measures to limit population levels and sensitive uses in dust-affected areas could reduce their properties' values and restrict their ability to improve and develop their properties in future.

The port and its users are major contributors to exports, taxes, royalties and employment. The port's continued operation and growth are important to the Western Australian and national economies.

The community aspires to live in a growing and vibrant town with good economic and social infrastructure and services, and improved amenity. The West End is an important part of that aspiration. The Town of Port Hedland needs certainty and predictability to manage its own planning and approvals processes and implement its Local Planning Strategy.

The Taskforce has framed its recommendations to meet the Health Risk Assessment objective of limiting the population exposed to relatively high dust levels in a way that takes all of these interests into consideration. Its main recommendations are:

- Exporters should continue to be required to reduce the dust emissions arising from port activities, and should be subject to more stringent risk-based regulation aimed at reducing the number of days when standards are exceeded.
- A Special Control Area should apply over the West End of Port Hedland, aimed at preventing further residential population growth west of Taplin Street, and limiting sensitive uses between Taplin and McGregor Streets.
- The State Government and the Town of Port Hedland will work with the community to improve Port Hedland's amenity, including identifying and addressing dust sources other than the port.

The following section details all of the Taskforce's recommendations.

1.2 RECOMMENDATIONS

The Taskforce recommends:

Health Risk Assessment - Interim Guideline

Recommendation 1:

The Taskforce recommends that the current interim guideline of 24-hour PM₁₀ of 70 µg/m³ (+ 10 exceedances to accommodate natural events) continues to apply to residential areas of Port Hedland and that measures should be introduced to cap (and if possible, reduce) the number of permanent residents in dust-affected areas of Port Hedland.

Industry Dust and Noise Management Regulation

Air Quality Monitoring

Recommendation 2:

The Taskforce recommends that:

- The Port Hedland Industries Council continue operating and maintaining its air quality network, with responsibility for oversight of the network, including data verification, storage and publication, transferred to the Department of Environment Regulation. The Taskforce notes that the Department of Environment Regulation will consider a number of options, including regulations, to implement this recommendation.

Industry Regulation

Recommendation 3:

The Taskforce recommends that:

- 3.1** The Department of Environment Regulation implements a coordinated risk-based review and assessment approach to managing dust and noise in Port Hedland through a review of all port premises licences under Part V, Division 3 of the *Environmental Protection Act 1986*.
- 3.2** Where premises are subject to Ministerial Statements, the Department of Environment Regulation will provide the findings and recommendations of its risk-based review and assessment to the Environmental Protection Authority and the Office of the Environmental Protection Authority.
- 3.3** The Environmental Protection Authority and the Office of the Environmental Protection Authority will consider the Department of Environment Regulation's assessments, and the appropriateness of conditions in Ministerial Statements.
- 3.4** Where the Environmental Protection Authority inquires under section 46 of the Environmental Protection Act 1986 into the conditions within Ministerial Statements, the Environmental Protection Authority will provide the Minister for Environment with a report on whether the conditions in the Statement/s should be changed.
- 3.5** The Department of Environment Regulation finalises and implements dust management guidelines for bulk handling port premises, outlining its expectations in relation to the assessment of dust impacts, dust control and monitoring requirements from these premises.

Noise

Recommendation 4:

The Taskforce recommends that:

- 4.1** The Department of Environment Regulation assesses unacceptable noise levels and assesses whether additional controls can be introduced as part of its review of all port premises licences under Part V, Division 3 of the *Environmental Protection Act 1986*.
- 4.2** The Town of Port Hedland uses the Port Hedland Cumulative Noise Study to inform its land use planning for the West End of Port Hedland.

Land-use planning

Recommendation 5:

The Taskforce recommends that:

- 5.1** The Minister for Planning asks the Town of Port Hedland to implement a Special Control Area westwards from McGregor Street as part of its Town Planning Scheme No. 5.

- 5.2** The Special Control Area prohibits new permanent residential development and other sensitive land uses, including aged care and child care premises, west of Taplin Street.
- 5.3** Low-density (R20) residential development be permitted in the predominantly residential area between Taplin and McGregor Streets, but higher-density residential development and other sensitive land uses be prohibited.
- 5.4** The zoning in the Special Control Area aligns with the Town of Port Hedland Local Planning Strategy's Precinct 1, taking into consideration the findings of the Health Risk Assessment.

Local Government and Community

Recommendation 6:

The Taskforce recommends that:

- The Town of Port Hedland works with key stakeholders to identify and mitigate dust from non-industry sources, with a focus on:
 - Identifying and implementing dust mitigation options for the spoil bank;
 - Sealing unsealed roads and undertaking regular and effective street sweeping operations;
 - Considering greening options, including coastal dune revegetation and the establishment of a green belt around the port; and
 - Reviewing and improving the efficacy of municipal services associated with dust control.

Governance

Recommendation 7:

The Taskforce recommends that:

- 7.1** The Taskforce continues to operate, with a focus on sharing information and co-ordinating agency activities when needed.
- 7.2** The Taskforce reports annually to the Minister for State Development on progress in implementing the recommendations in this report and on the overall status of dust and noise management in Port Hedland.

2. BACKGROUND

Port Hedland¹ is 1,300 kilometres north of Perth and falls within the Town of Port Hedland local government area, which also includes South Hedland, Wedgefield and some Aboriginal communities (See Figure 1). The environment is naturally dusty, with high levels of background dust from fires and other natural sources.

Port Hedland has grown rapidly in recent years, and exports through the port have risen substantially. This has contributed to high dust levels in the western areas, even though industry and the Pilbara Ports Authority have significantly improved the monitoring and management of dust levels.

In 2012-13, Port Hedland had 4,590 residents, of which 529 were in the West End². The West End population represented about 4% of the total local government population of 13,772. The Town of Port Hedland's Local Planning Strategy (the Pilbara's Port City Growth Plan) identifies the West End as a major tourism precinct.

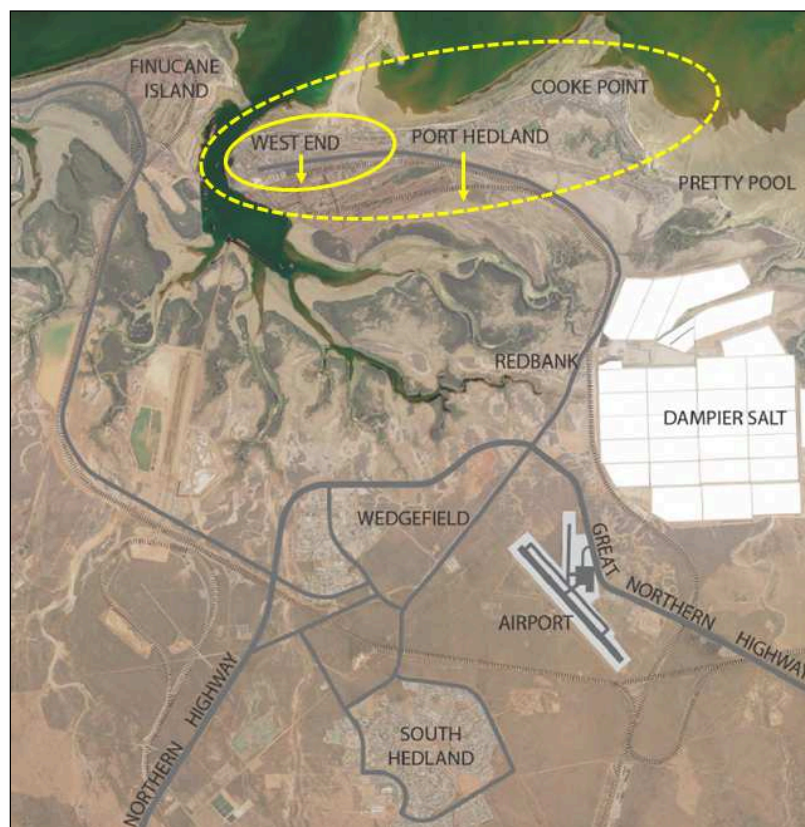


Figure 1: The West End in Port Hedland (Source: Town of Port Hedland, 2012: 15)

The Port of Port Hedland, adjacent to the West End, is the largest bulk minerals port in the world. Between 2010 and 2015, iron ore exports from

¹ The term 'Port Hedland' refers to the peninsula west of Pretty Pool and the term 'West End' refers to the area west of Taplin Street.

² Toxikos, 2015 - <http://ww2.health.wa.gov.au/Reports-and-publications/Port-Hedland-Health-Risk-Assessment>

the port increased from 167 million tonnes to 424 million tonnes. Other exports include salt, manganese, chrome and copper concentrates, and cattle.

The value of exports from the port was \$29.7 billion in 2014-15, accounting for 27% of Western Australia's merchandise exports and 12% of Australia's merchandise exports. Iron ore accounted for 95% (\$28.3 billion) of Port Hedland's merchandise exports in 2014-15, followed by copper (3% or \$996 million). In 2014-15, 57% of Western Australia's iron ore exports were exported from Port Hedland.

Port Hedland's exports of iron ore, copper and other base metals contributed an estimated \$2.4 billion of royalties to Western Australia in 2014-15, accounting for 48% of Western Australia's total royalty receipts (excluding North West Shelf Grants). The Pilbara Ports Authority recorded before-tax profit of \$283.8 million in 2014-15, and returned a dividend to the State of \$164.5 million.

The rapid development of this major industrial port in close proximity to an established residential area has created challenges that require careful management. In 2009, the Environmental Protection Authority expressed concerns about dust levels in Port Hedland, and the adequacy of planning controls to limit exposure to dust. At the time there was growing scientific evidence suggesting that dust can affect human health. The Authority proposed an integrated government and industry strategy to reduce and manage emissions. In response to these concerns, in 2009 the Premier established the Port Hedland Dust Management Taskforce (the Taskforce) to co-ordinate and plan for dust management in Port Hedland.

The Taskforce includes representatives of the Town of Port Hedland; the Pilbara Ports Authority; the Departments of Health, Planning, State Development and Environmental Regulation; the Environmental Protection Authority; and major port users (including BHP Billiton Iron Ore, Fortescue Metals Group and Roy Hill). The Port Hedland Industries Council is also a member of the Taskforce.

The Taskforce released the *Port Hedland Air Quality and Noise Management Plan* (the 2010 Management Plan) which the Government and Taskforce members endorsed. The 2010 Management Plan established an interim guideline measure for air quality in Port Hedland of 70 micrograms of PM₁₀ particles³ per cubic metre, with no more than 10 annual exceedances. The interim guideline applied pending the completion of a Health Risk Assessment and a new air quality model.

³ Dust, or particulate matter (PM), is material suspended in the air in the form of small solid particles, or liquid droplets. Particulate matter can be categorised as PM₁₀ or PM_{2.5}. PM₁₀ particles are 10 micrometres in diameter and smaller, while PM_{2.5} particles are 2.5 micrometres in diameter and smaller. The concentration of an air pollutant is given in micrograms (one-millionth of a gram) per cubic meter of air, or µg/m³.

The 2010 Management Plan also recommended measures for managing dust emissions and exposure in Port Hedland. These included a planning scheme amendment for the West End (Amendment 22, gazetted 27 April 2012), the development of an air quality monitoring regime and a Health Risk Assessment.

The Department of Health managed the Health Risk Assessment, which it released in February 2016. With the completion of this work, and the availability of other noise and air quality research, including several years of air quality monitoring data, the Taskforce has the information necessary to make recommendations for managing dust in Port Hedland.

This report outlines progress in implementing the 2010 Management Plan, the Taskforce's response to the Health Risk Assessment and its recommendations concerning the future management of dust and noise emissions in Port Hedland.

3. HEALTH RISK ASSESSMENT

3.1 OVERVIEW

The 2010 Management Plan recommended a Health Risk Assessment to assess the risk posed to the health of Port Hedland residents from exposure to high dust levels.

Human health risk assessments assist regulatory agencies, industry managers and the public to formulate strategies to protect human health from substances of concern, but they are not the only source of information for making decisions. A human health risk assessment estimates the risk to a population arising from exposure to a substance of concern. The process considers the uncertainties in determining the risk, the characteristics of the substance of concern and the affected population. It identifies the ways people are exposed to a substance, and includes an assessment of acute (immediate or short-term) and chronic (delayed or long-term) health risks that might arise from the exposure. Incomplete information limits risk assessments because it is not possible to know everything about the substance of concern, or the population exposed. To counteract this, risk assessments use safety margins that overestimate the risk.

The steps of a human health risk assessment generally include issue identification, hazard assessment, exposure assessment, risk characterisation and risk management.

The Department of Health released its Health Risk Assessment report, including a detailed technical report prepared by consultants Toxikos, in February 2016. The summary report (Port Hedland Air Quality Health Risk Assessment for Particulate Matter), together with the Toxikos report (Health Risk Assessment Port Hedland) constitutes the Health Risk Assessment⁴.

The Health Risk Assessment determined that PM₁₀ particles (coarse particulate matter) could affect human health in Port Hedland. Port Hedland dust mainly consists of rock and mineral particles, such as iron oxide. Other particles include salt, manganese, copper and other minerals. Port Hedland dust is both naturally derived (crustal) and anthropogenic (the result of human activity, such as mining and transporting activities).

⁴ <http://ww2.health.wa.gov.au/Reports-and-publications/Port-Hedland-Health-Risk-Assessment>

3.2 FINDINGS

The Health Risk Assessment concluded:

- There is sufficient evidence of potential impacts on human health from dust, specifically PM₁₀, in the Toxikos Report to warrant dust management controls and strategic and land-use planning to reduce community exposure to dust.
- Most of the public health concerns about dust in Port Hedland arise from PM₁₀ concentrations over 70 µg/m³. Research suggests coarse particles (PM_{2.5-10}) are associated with an increase in all-cause mortality and hospitalisation for respiratory conditions. The areas affected are close to the Port.
- The number of affected individuals is very low because the population is small. With a larger population, the impact on health would be more visible and would necessitate more immediate regulatory control.
- A legacy of the rapid growth of Port Hedland is the close proximity of residential areas to commercial operations at Nelson Point and the port. This means that fugitive dust from port and commercial operations at Nelson Point and Finucane Island disperses over residential areas under certain meteorological conditions, despite good dust management control.

3.3 HEALTH RISK ASSESSMENT RECOMMENDATIONS

The Health Risk Assessment made the following recommendations:

a) Guideline and Exposure Reduction

- Introduce exposure reduction measures that include capping the number of permanent residents to current numbers in areas most impacted by dust currently to the west of Taplin Street. Because acceptable risk is based on population size, a strategy must be introduced now to manage and restrict future population growth in Port Hedland. The closer to the port and Nelson Point operations the tighter the restrictions. A long-term land-use planning strategy may offer a tool for gradually moving the residential areas away from the operations area. Since the aim of government is not to disadvantage anyone currently living in the area planning tools such as Amendment 22 may offer a means to manage exposure while also managing population growth.
- Current regulatory controls for managing dust from operations at Nelson Point and Finucane Island may be aided by declaring a buffer between the port and residential areas further to the east. Air

quality modelling can help define this area but should not be the sole decision making tool used for determining the buffer boundary.

- Apply the current interim guideline of 24-hour PM₁₀ of 70 µg/m³ (+ 10 exceedances to accommodate natural events) in residential areas of Port Hedland within a reasonable time frame that allows for local dust sources to be identified and managed (i.e. the spoil bank). A period of five years is suggested.
- The interim guideline can be applied to South Hedland and Wedgefield but it may also be possible to achieve the National Environment Protection Measure for Ambient Air Quality in South Hedland if the source of local exceedances can be identified and managed.
- A coordinated approach to reduce dust from all sources, not just industry, is required. Various government sectors (planning, transport, energy) may need to develop and implement long-term policies and strategies that reduce exposure.

b) Air quality monitoring

- An ongoing air quality monitoring program is vital to monitor exposure risk. This program should include the criteria National Environment Protection Measure pollutants (minus lead) and manganese. The program should have the capacity to include additional pollutants as indicated by the development of new industries or changes to existing industries.
 - Exceedances of the interim guideline should be investigated and reported to the Department of Environment Regulation.
- c) Impact assessments for new developments and future expansion of existing industry should include baseline air quality data and consider additional impacts on air quality on the Port Hedland air-shed.
- d) Promote and encourage existing efforts at continuous improvement among stakeholders. Even small reductions in overall particulate matter can have incremental benefits, which, at least theoretically, contribute to improved amenity, reduced potential health risks and improved health status on a population basis.
- e) Assist local government to promote a community awareness of the benefits of reducing exposure to particulate matter overall. This may include ways to reduce personal exposure during extreme events.

- f) Promote an all of government support for further research on the health effects of crustal dust and the importance of exposure reduction.

RECOMMENDATION 1

The Taskforce endorses the Department of Health's recommendation that the current interim guideline of 24-hour PM₁₀ of 70 µg/m³ (+ 10 exceedances to accommodate natural events) should apply to residential areas of Port Hedland and that measures should be introduced to cap the number of permanent residents in dust affected areas of Port Hedland.

The Taskforce's responses to the other recommendations in the Health Risk Assessment are included in the following sections.

4. INDUSTRY DUST AND NOISE MANAGEMENT AND REGULATION

4.1 OVERVIEW

The 2010 Management Plan recognised the need for a local air quality management plan in the West End, where dust emissions regularly exceeded the National Environment Protection Measure for Ambient Air Quality. The 2010 Management Plan also proposed environmental management controls, including air quality monitoring and regulation and noise management.

4.2 AIR QUALITY MONITORING

The Port Hedland Industries Council established an air quality monitoring regime to count exceedances of the interim air quality target set out in the 2010 Management Plan. The Port Hedland Industries Council developed an ambient air quality monitoring network with monitoring points throughout Port Hedland, Wedgefield, South Hedland, and background/reference monitoring sites at Port Hedland Airport (Bureau of Meteorology) and Yule River. The Port Hedland Industries Council has monitored particulate matter across its network since 2012 and has released three annual monitoring reports, which are publicly available⁵. The real-time data is also available to the public on their website⁶.

Iron ore exports have grown substantially since the release of the Taskforce's 2010 Management Plan (from 167 million tonnes in 2010 to 424 million tonnes in 2015), but there has not been a proportionate increase in dust emissions. Air quality in the eastern parts of Port Hedland has largely remained within the interim guideline set by the Taskforce, while dust emissions in the West End of Port Hedland remain high. Figures 2 and 3 summarise data for the past three years from the Port Hedland Industries Council's monitoring stations around Port Hedland.

Due to the naturally dusty environment, the monitoring network includes background monitoring locations at Yule River and the Bureau of Meteorology site. The 2014-15 monitoring report indicated that the Taskforce's air quality criteria were exceeded on 10 days at the Taplin Street monitor, and that three of the events were due to elevated background dust levels.

⁵ http://www.phic-hedland.com.au/phic/Annual_Report.htm

⁶ <http://phicmonitoring.com.au/esys/rt/realtime.jsp?siteId=371>

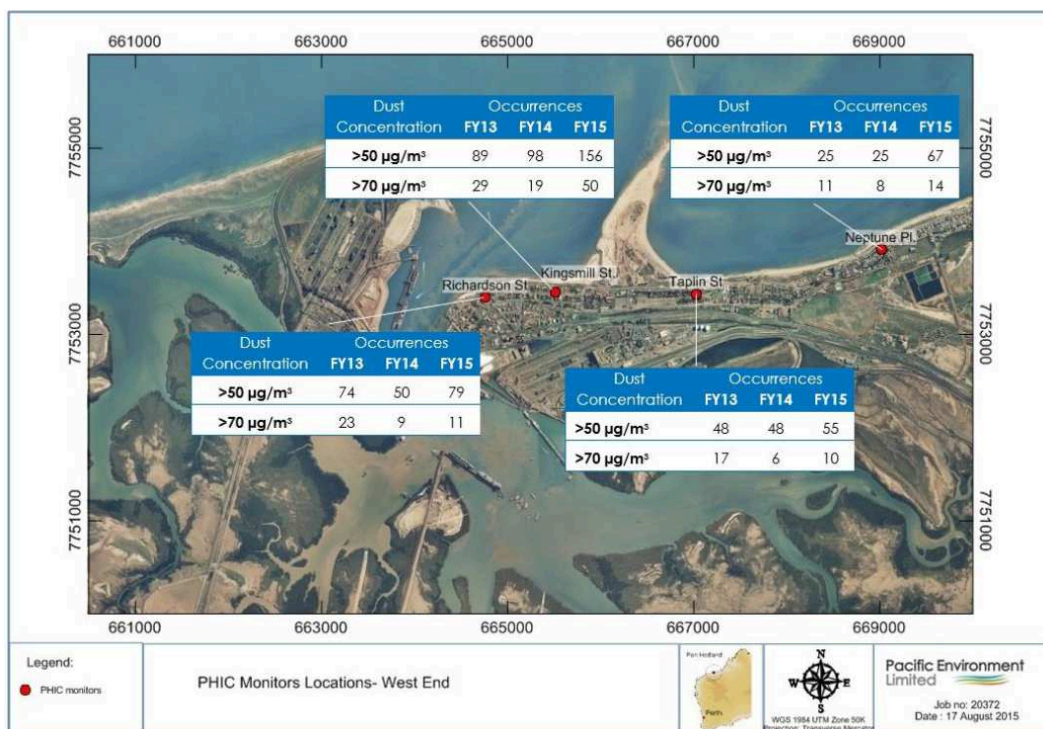


Figure 2: Port Hedland Industries Council Monitoring Station data around the West End in Port Hedland (Source: Port Hedland Industries Council, 2015: vii)

Wind direction information obtained from the monitoring network, and additional data from temporary monitors installed on the Port Hedland spoil bank, suggest that a number of sources contribute to elevated dust levels in the West End. Sources in addition to regional background dust and port-related activities may include the spoil bank itself, the Wedgefield industrial area (which has recorded significantly higher readings than the other monitoring locations), residential and commercial construction work in the West End, and traffic disturbance of dust build-up on roads. All of these sources should be addressed as part of a comprehensive response to managing dust in Port Hedland.

The Health Risk Assessment noted the importance of an ongoing air quality monitoring program to monitor dust exposure.

Some community members do not have confidence in the credibility of the air monitoring network data, given that industry funds, maintains and provides the data from the network. While the Taskforce has confidence in the accuracy of the data obtained from the network, it is important that the community shares this confidence. For this reason, the Taskforce recommends that oversight of the monitoring network, including the verification and publication of monitoring data, is transferred to the Department of Environment Regulation. The Department of Environment Regulation will consult with industry regarding options for implementing this recommendation.

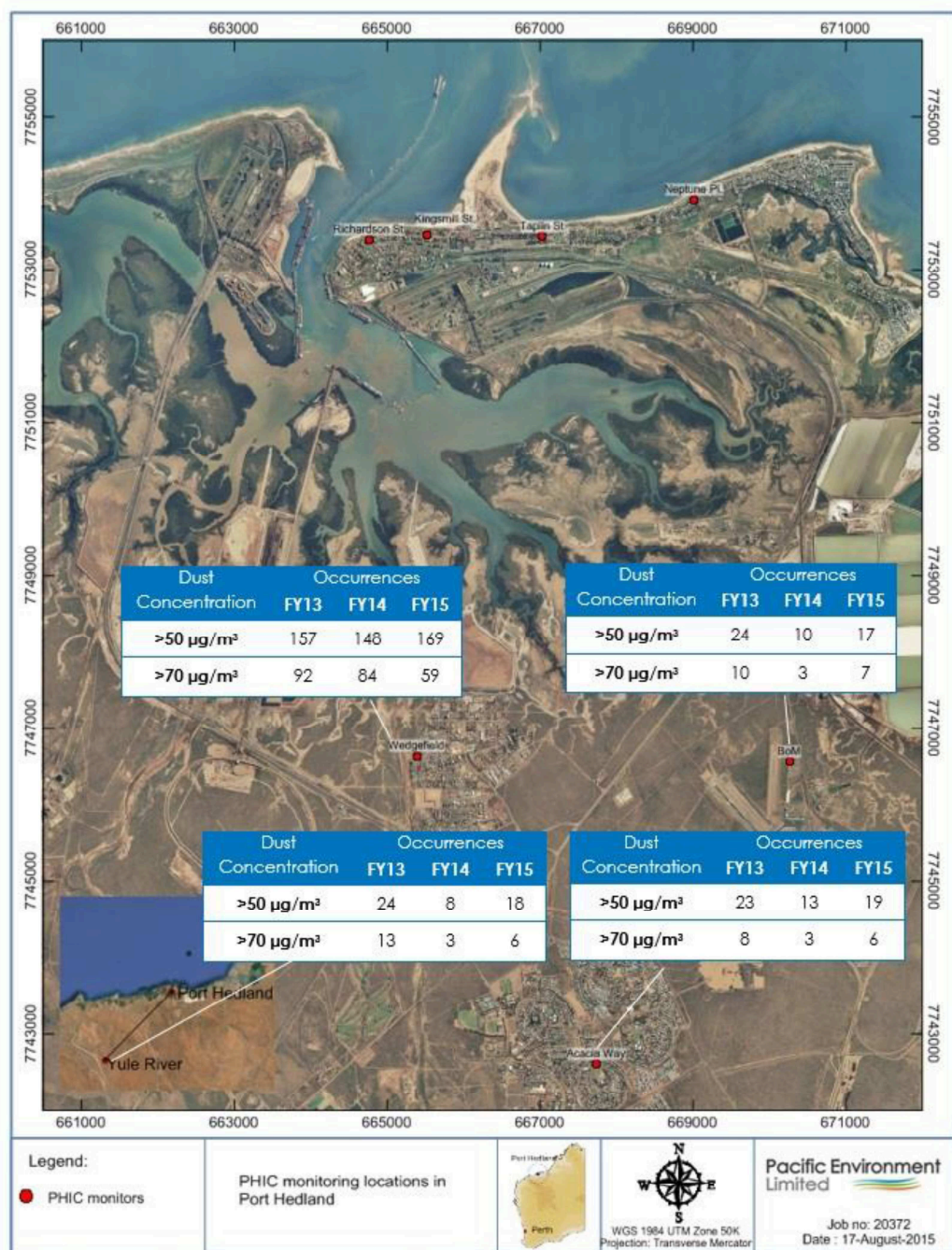


Figure 3: Additional Port Hedland Industries Council Monitoring Stations in Port Hedland (Source: Port Hedland Industries Council, 2015: viii)

RECOMMENDATION 2

The Taskforce recommends that:

- The Port Hedland Industries Council continue operating and maintaining its air quality network, with responsibility for oversight of the network, including data verification, storage and publication, transferred to the Department of Environment Regulation. The Taskforce notes that the Department of Environment Regulation will consider a number of options, including regulations, to implement this recommendation.

4.3 INDUSTRY REGULATION

Two agencies administer regulatory functions under the *Environmental Protection Act 1986* for major port premises within Port Hedland.

The Environmental Protection Authority undertakes environmental impact assessments of significant proposals and provides recommendations to the Minister for Environment on whether proposals may be implemented and, if so, any conditions which should be applied under Part IV, Division 1 of the *Environmental Protection Act 1986*.

The Minister for Environment determines whether proposals can be implemented and the conditions that apply, if any, through a Ministerial Statement issued under Part IV, Division 2 of the *Environmental Protection Act 1986*. The Office of the Environmental Protection Authority monitors compliance with Ministerial Statements. Most major port premises in Port Hedland hold a Ministerial Statement.

The Department of Environment Regulation regulates prescribed premises listed in Schedule 1 of the *Environmental Protection Regulations 1987*, including port premises under Part V, Division 3 of the *Environmental Protection Act 1986*. The Department of Environment Regulation licences and applies conditions on prescribed premises with regard to, and consistent with, issued Ministerial Statements, and seeks to avoid regulatory duplication. The Department also carries out compliance and enforcement actions in relation to pollution offences, noise, and the clearing of native vegetation.

Licences issued under Part V of the *Environmental Protection Act 1986* in the Port Hedland area include monitoring requirements. Some premises are required to undertake boundary monitoring and compare results with ambient air quality data from the Taplin Street monitoring station. In other cases, monitoring and reporting requirements are applied directly to exceedances at Taplin Street or, in the case of Fortescue Metal Group's Anderson Point operation, the Wedgefield monitoring station. Where exceedances of the air quality target are identified, these are reported to the Department of Environment Regulation and compared to ambient monitoring data. The Port Hedland Industries Council also publishes annual reports, which summarise the ambient air quality monitoring data from the monitoring network.

Similar dust management and monitoring requirements have been applied through Ministerial Statements issued under Part IV of the *Environmental Protection Act 1986* to major port premises within Port Hedland.

With the release of the Health Risk Assessment, the Department of Environment Regulation can now implement a coordinated risk-based review and assessment for the port premises based on the most

up-to-date information available. Its regulatory response will be informed by an analysis of data from boundary and ambient air quality monitoring networks, as well as an understanding of the responses by premises, based on meteorological forecasting.

Additionally, the Department of Environment Regulation's review of prescribed premises' licences (issued under Part V of the *Environmental Protection Act 1986*) will involve:

- an assessment of the effectiveness of dust controls applied by premises occupiers; and
- implementation or recommendation of suitable regulatory controls through outcome-based conditions where possible or specified infrastructure and/or monitoring programs.

The Department of Environment Regulation's regulatory response will also assess other impacts, including noise, and determine the most suitable controls if the Department concludes that these impacts have a moderate or higher risk to public health or the environment. The Department aims to review the licences of all licensed port premises by the end of 2016, and will apply its Regulatory Principles⁷ to ensure a risk-based regulatory approach.

Where a premise is also subject to a Ministerial Statement, the Department of Environment Regulation will provide a report outlining the findings and recommendation of their review and assessment to the Environmental Protection Authority and Office of the Environmental Protection Authority.

Any amendment to a Ministerial Statement will require an inquiry under section 46 of the *Environmental Protection Act 1986*. If the Minister for Environment considers that the implementation conditions relating to a proposal should be changed, section 46 enables the Minister to request the Environmental Protection Authority to inquire into the matter. On completing its inquiry, the Environmental Protection Authority will provide the Minister for Environment with a report on whether or not the implementation conditions should be changed.

The Minister for Environment, following consultation with other relevant Ministers, will then determine whether the conditions should be changed and, if so, issue a Ministerial Statement with the new conditions. The proponent of the proposal for which the implementation conditions are changed has a right of appeal. The Office of the Environmental Protection Authority manages this process.

⁷ Department of Environment Regulation, 2015. Guidance Statement – regulatory principles https://www.der.wa.gov.au/images/documents/our-work/regulatory-principles/Guidance_Statement_Regulatory_Principles.pdf

For port premises regulated by the Department of Environment Regulation and not holding a Ministerial Statement, all controls will be set out in the licences issued to port premises. The Department of Environmental Regulation will publish decision reports on its website explaining the risk assessment process and justification for the controls imposed on the licence.

The Department of Environment Regulation is also developing dust management guidelines specifically for bulk-handling port premises. These guidelines will outline its expectations in relation to the assessment of dust impacts from these premises and dust control and monitoring requirements. The guidelines will inform the establishment of appropriate and enforceable dust conditions for port premises licences. It is expected that port users will improve dust management practices in accordance with the Department of Environment Regulation's risk-based regulatory approach.

RECOMMENDATION 3

The Taskforce recommends that:

- 3.1 The Department of Environment Regulation implements a coordinated risk-based review and assessment approach to managing dust and noise in Port Hedland through a review of all port premises licences under Part V, Division 3 of the *Environmental Protection Act 1986*.**
- 3.2 Where premises are subject to Ministerial Statements, the Department of Environment Regulation will provide the findings and recommendations of its risk-based review and assessment to the Environmental Protection Authority and the Office of the Environmental Protection Authority.**
- 3.3 The Environmental Protection Authority and the Office of the Environmental Protection Authority will consider the Department of Environment Regulation's assessments, and the appropriateness of conditions in Ministerial Statements.**
- 3.4 Where the Environmental Protection Authority inquires under section 46 of the Environmental Protection Act 1986 into the conditions within Ministerial Statements, the Environmental Protection Authority will provide the Minister for Environment with a report on whether the conditions in the Statement/s should be changed.**
- 3.5 The Department of Environment Regulation finalises and implements dust management guidelines for bulk handling port premises, outlining its expectations in relation to the assessment of dust impacts, dust control and monitoring requirements from these premises.**

4.4 INDUSTRY INITIATIVES

Major port users have taken significant measures to manage their dust emissions since the release of the 2010 Management Plan, with overall dust levels remaining relatively unchanged despite substantial increases in tonnages through the port.

The Pilbara Ports Authority developed its Dust Management Leading Practice Guidelines⁸ to support the objectives of the 2010 Management plan. The Guidelines promoted a best-practice approach for the coordinated management of dust emissions from Port Hedland port operations, and focus on bulk material handling activities. The Taskforce notes that the Department of Environment Regulation is preparing guidance on bulk material handling at premises regulated by the Department under Part V of the *Environmental Protection Act 1986*.

Since 2010, the Pilbara Ports Authority has relocated all external stockpiles from the eastern side of the harbour to the Utah Point facility. All bulk material handling on the eastern side under the management of the Pilbara Ports Authority is now undertaken within closed sheds or by using containers. The Pilbara Ports Authority monitors dust at Utah Point and its eastern operations using what it considers are best practice technologies and processes.

Dust control measures include the use of water cannons and mister sprays, and requiring all product delivered to the site to be at the correct moisture levels to limit dust generation. Conveyor systems and transfer stations are all sealed to limit dust emissions on site. The condition and quality of product delivered to the Pilbara Ports Authority's Utah Point and eastern operations is the responsibility of the shippers using these facilities. However, if this is not done properly, the Pilbara Ports Authority takes action to limit, and in some cases refuses, the delivery of product until the correct quality objectives can be met.

Major port users have implemented dust mitigation measures for their operations. In 2010, BHP Billiton Iron Ore relocated its crushing and screening facilities, which historically were some of the larger dust contributors, to its mines. It has also increased the use of direct-to-ship loading, reducing the need to double-handle product at the port. Fortescue Metals Group's dust management plan covers a range of construction and operational stage dust mitigation measures. Dust mitigation for construction activities includes measures affecting vegetation clearing (for example, use of water carts and staged clearing) and earthworks (for example, minimising areas to be cleared). Operational dust mitigation measures cover railcars (enclosing key components of rail car dumpers), conveyors and transfer points (moisture control and enclosing transfer

⁸ [https://www.pilbaraports.com.au/PilbaraPortsAuthority/media/Documents/PORT%20HEDLAND/ENVIRONMENT%20AND%20HERITAGE/Dust-Management-Leading-Practice-Guidelines-\(A232535\).pdf](https://www.pilbaraports.com.au/PilbaraPortsAuthority/media/Documents/PORT%20HEDLAND/ENVIRONMENT%20AND%20HERITAGE/Dust-Management-Leading-Practice-Guidelines-(A232535).pdf)

points), stackers (spray heads), reclaimers (water sprayers), stockpiles (using coarse stockpiles to protect fines stockpiles) and ship loading (water sprayers).

While industry has achieved significant improvements in dust management in recent years, further improvements are required to ensure dust emissions are not increased, and where practicable reduced, if increases in port throughput occur.

4.5 CUMULATIVE AIR DISPERSION MODELLING

As part of the recommendation in the 2010 Management Plan for establishment of a best practice air quality management regime in Port Hedland, the Taskforce, through the Port Hedland Industries Council and the Department of Environment Regulation, commissioned a cumulative air model for Port Hedland. The intention was for the model to assist Taskforce decision-making by producing contours of exceedances of the interim air quality standard under port growth scenarios. Despite considerable progress, the Taskforce notes that the model developed has significant limitations affecting its usability for decision-making.

A peer review of the model in 2016 confirmed that the model, in its current form, is not suitable for informing land-use decisions. The model only uses emission estimates from port industries and accounts simplistically for other sources such as bushfires, dust storms and localised sources (such as town vehicle dust, construction dust and windblown dust from beaches, roads and spoil banks), through the use of a single factor called “background dust”. In addition, the uncertainties associated with the model outputs are large owing to uncertainties in emission estimates, meteorology and in the background variation.

The Taskforce notes that the preferred model is better suited for assessing relative changes in dust concentrations, rather than the total dust concentration. The Department of Environment Regulation has advised that models are comparative assessment tools and that monitoring data provides a better estimate of the actual impact currently occurring in Port Hedland. The model will therefore serve as a useful tool for addressing the Health Risk Assessment recommendation that “Impact assessments for new developments and future expansion of existing industry should include baseline air quality data and consider additional impacts on air quality on the Port Hedland air-shed”.

4.6 NOISE

The 2010 Management Plan also addressed concerns around noise caused by industrial activity in Port Hedland. High noise levels may affect

the amenity of adjacent residential areas. Noise emitters in Port Hedland include port facilities and a number of rail and road operators.

Cumulative noise emissions from industries in Port Hedland exceed the assigned noise levels in the *Environmental Protection (Noise) Regulations 1997*. The 2010 Management Plan recommended a noise regulatory strategy based on the:

- development of a cumulative noise model;
- definition of noise sensitive zones;
- clarification of planning measures; and
- clarification of building standards.

In 2014, SVT Engineering Consultants completed the Port Hedland Cumulative Environment Noise Study (Noise Study), commissioned by the Port Hedland Industries Council and the Department of Environment Regulation. The study produced noise models for emissions from port facilities and rail and road usage. The models covered 'current', 'near future', 'intermediate future' and 'ultimate capacity' scenarios for Port Hedland. The noise predictions provided by the study account for a worst-case scenario for operations, that is all equipment operating simultaneously, and weather conditions.

The study predicted exceedances of noise levels for port and industrial facilities (as assigned through the *Environmental Protection (Noise) Regulations 1997*) across Port Hedland's residential areas. Noise levels in South Hedland under the 'current' scenario were modelled to be below assigned levels, but exceedances were predicted under the 'near future', 'intermediate future' and 'ultimate capacity' scenarios.

Some exceedances of the noise targets set in State Planning Policy 5.4 from road usage were predicted in the West End of Port Hedland under all scenarios. The rail noise model did not predict any exceedances at the noise sensitive receivers used in the modelling.

The Department of Environment Regulation is considering a number of regulatory strategies, including:

- Individual approvals under Regulation 17 of the *Environmental Protection (Noise) Regulation 1997*, where approval to exceed or vary from the Regulations can be granted where noise emissions cannot reasonably and practicably comply with the prescribed noise standard.
- A precinct-based Regulation 17 approval, whereby Regulation 17 approvals could be granted to specific entities – for example, to the Pilbara Ports Authority and other port operators, to cover all the emitters within the port precinct.
- Precinct noise regulations, involving the development of a new noise regulation within, or alongside, the *Environmental Protection (Noise) Regulations 1997* that would set different allowable noise levels for a defined 'Port Hedland precinct'.

- A Ministerial exemption order exempting activities from the prescribed standards specified in the *Environmental Protection (Noise) Regulations 1997* (under section 6 of the *Environmental Protection Act 1986*).

The Town of Port Hedland can also use the Noise Study when reviewing land-use planning measures in the West End.

RECOMMENDATION 4

The Taskforce recommends that:

- 4.1 The Department of Environment Regulation assesses unacceptable noise levels and assesses whether additional controls can be introduced as part of its review of all port premises licences under Part V, Division 3 of the *Environmental Protection Act 1986*.**
- 4.2 The Town of Port Hedland uses the Port Hedland Cumulative Noise study to inform its land-use planning for the West End of Port Hedland.**

5. LAND-USE PLANNING

5.1 OVERVIEW

The 2010 Management Plan recommended progression of an amendment to the Town of Port Hedland Town Planning Scheme No.5 to address residential and sensitive land uses in dust-affected areas of Port Hedland. The subsequent Amendment 22, gazetted in April 2012, established the West End Residential zone in the most dust-affected areas around the port. The objectives of the West End Residential zone are to:

- restrict the form of residential development so that long-term residency by families with children or elderly persons is discouraged;
- add vibrancy to both the subject land and the nearby commercial area;
- maximise opportunities for workers in nearby employment nodes to reside close to work; and
- provide opportunities for commercial, entertainment facilities and short stay accommodation.

Building design and performance standards to address exposure to dust were included in Amendment 22, as recommended in the 2010 Management Plan. These included the use of deflection screens, building orientation requirements and the location of operable windows on the western and southern facades only.

The 2010 Management Plan also recommended the preparation of a development plan for the Port Hedland area. The Western Australian Planning Commission endorsed the *Pilbara's Port City Growth Plan* (Growth Plan) in 2012. The Growth Plan functions as the Town of Port Hedland's Local Planning Strategy and was formulated through an inclusive planning process, which included a comprehensive stakeholder consultation process.

Consistent with the precautionary approach recommended in the 2010 Management Plan, the Growth Plan recommends the progressive transition from permanent residential uses under the current West End Residential zone towards a longer term land use scenario with no permanent residential (short-stay accommodation only) west of Acton Street.

5.2 PROPOSED LAND USE PLANNING CHANGES

The Health Risk Assessment recommends the introduction of measures to cap the number of permanent residents in dust-affected areas of Port Hedland. The Taskforce supports this recommendation. Port operations are expected to expand in the future (the Pilbara Ports Authority estimates total exports to increase to 481 million tonnes in 2016-17, up from 433 million tonnes in 2015-16⁹), and the port has an important role in the economic growth of the Pilbara and Western Australia. With increasing throughput, it will be difficult to reduce dust emissions even with continued improvements in dust generated per tonne of exports.

Dust levels in the West End of Port Hedland are expected to remain high. Because acceptable risk is based on population size, a strategy must be introduced to manage and restrict future population growth in Port Hedland to reduce the overall population exposed to high levels of dust. The Health Risk Assessment recommends that “the closer to the port and Nelson Point operations, the tighter the required restrictions” and notes that “current regulatory controls for managing dust from operations at Nelson Point and Finucane Island may be aided by declaring a buffer between the port and residential areas further to the east”.

Dust monitoring data indicate that Taplin Street, the current eastern boundary of the West End precinct, remains the most appropriate boundary for limiting residential land use in proximity of the port. However, dust levels at the Neptune Street monitoring station east of Taplin Street and McGregor Street are also around the recommended threshold levels.

There is now sufficient evidence for the State and Local Governments to consider additional planning measures to further limit residential land use. The Taskforce recommends that the Town of Port Hedland and the Department of Planning collaborate to implement measures limiting permanent residential developments and other sensitive land uses in dust-affected areas of Port Hedland.

The Taskforce recommends the establishment of a Special Control Area over land west of Lukis and McGregor Streets, to the port (see Figure 4). The Special Control Area should prohibit new permanent residential development and other dust sensitive land uses, including aged care and childcare premises, in the area west of Taplin Street. New low-density (R20) residential developments would be permitted in the area between Taplin Street and Lukis/McGregor Streets, but higher density residential development and other dust sensitive land uses would be prohibited. The extension of planning controls to Lukis and McGregor Streets is a precautionary measure that allows for the possibility of increases in dust levels resulting from future port expansion.

⁹ Port Hedland Port Authority Port Development Plan 2012-2016

The proposed land-use measures will not affect the ability of property owners to carry out, or continue to enjoy, already approved developments on their property.



Figure 4: Proposed amendment area.

The Taskforce proposes that the Special Control Area align current zonings with the Growth Plan, taking into consideration the Health Risk Assessment. The Growth Plan defines the West End (Precinct 1) as the commercial and cultural core of Port Hedland with predominantly mixed land uses, including short stay accommodation (see Figure 5).

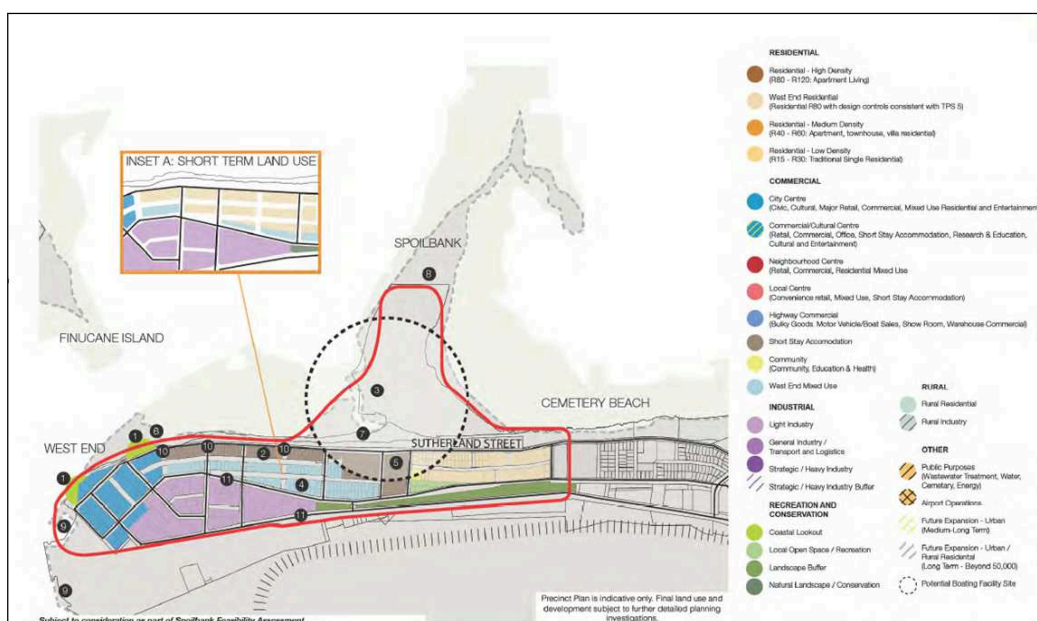


Figure 5: Pilbara's Port City Growth Plan: Precinct 1 – West End (Source: Town of Port Hedland, 2012:87)

The land-use planning measures recommended by the Taskforce can be incorporated into the Town of Port Hedland's Local Planning Scheme through a scheme amendment process prescribed under the *Planning and Development Act 2005*, and the associated *Planning and Development (Local Planning Schemes) Regulations 2015*.

RECOMMENDATION 5

The Taskforce recommends that:

- 5.1 The Minister for Planning asks the Town of Port Hedland to implement a Special Control Area westwards from McGregor Street as part of its Planning Scheme No. 5;**
- 5.2 The Special Control Area prohibits new permanent residential development and other sensitive land uses, including aged care and child care premises, west of Taplin Street;**
- 5.3 Low-density (R20) residential development be permitted in the predominantly residential area between Taplin and McGregor Streets, but higher-density residential development and other sensitive land uses be prohibited; and**
- 5.4 The zoning in the Special Control Area aligns with the Town of Port Hedland Local Planning Strategy's Precinct 1, taking into consideration the findings of the Health Risk Assessment.**

5.3 BUILDING DESIGN AND PERFORMANCE STANDARDS

The Taskforce is not convinced of the efficacy or enforceability of the building design and performance standards implemented as part of Amendment 22. These standards, included in the 2010 Management Plan's Appendix 4, include aspects like window and door orientation and filtered air conditioning. The Local Planning Scheme's building design and performance standards can be reviewed as part of a scheme review or amendment.

6. LOCAL GOVERNMENT AND COMMUNITY

6.1 OVERVIEW

Dust monitoring data suggest that there are sources of dust in Port Hedland other than the port that require investigation and management. These sources include the spoil bank and the Wedgefield industrial area. The recent growth of Port Hedland, which saw a significant increase in construction activity and infrastructure development, such as roads, has also contributed to dust emissions in the town.

The Town of Port Hedland is committed to working with the State Government, industry and the community to improve Port Hedland's amenity and reduce ambient dust levels in the West End.

6.2 SPOIL BANK

The Taskforce notes that the Town of Port Hedland and the State Government are committed to developing a waterfront precinct in Port Hedland, and that the Town of Port Hedland favours the development of a marina at the spoil bank site.

In 2013, the Town of Port Hedland proposed Scheme Amendment 56, which sought to establish a Marina Development zone within the Town of Port Hedland Town Planning Scheme 5 provisions and zone land on the spoil bank generally north of Sutherland Street from its current reservation as Parks and Recreation to Marina Development zone. Broadly, the Marina Development zone would provide for public marina uses as well as tourist, commercial and residential development.

In 2014, the Environmental Protection Authority determined that Amendment 56 was incapable of being made environmentally acceptable and could not be implemented due to its residential component. The Minister for Environment subsequently advised the Town of Port Hedland that it could review its amendment proposal for the spoil bank in view of the findings of the Health Risk Assessment, once complete.

Current planning for the first stage of the proposed spoil bank marina does not include any permanent residential development. The proposed Special Control Area for the West End will therefore not prevent this project from proceeding, although it would prohibit any permanent residential development for future stages of the project.

As the spoil bank contributes to dust emissions in the West End, the development of the marina may reduce emissions from this source. If the marina project does not proceed in the near future, the Town of Port

Hedland should identify and implement other options for mitigating dust from the spoil bank.

6.3 GREENING AND OTHER DUST MANAGEMENT INITIATIVES

The Taskforce notes that the Town of Port Hedland recently resolved to identify a range of dust mitigation measures in response to the Health Risk Assessment, including sealing unsealed roads and collaborating with the Pilbara Ports Authority, port users, industry and key stakeholders to investigate and implement green belts of vegetation around the port and town¹⁰. The Taskforce supports this initiative.

Other areas that appear to warrant further examination include reviewing current practice around site dust management plans, especially compliance and enforcement, and the effectiveness of the Town's street sweeping program.

RECOMMENDATION 6

The Taskforce recommends that:

- **The Town of Port Hedland works with key stakeholders to identify and mitigate dust from non-industry sources, with a focus on:**
 - **Identifying and implementing dust mitigation options for the spoil bank;**
 - **Sealing unsealed roads and undertaking regular and effective street sweeping operations;**
 - **Considering greening options, including coastal dune revegetation and the establishment of a green belt around the port; and**
 - **Reviewing and improving the efficacy of municipal services associated with dust control.**

¹⁰ <http://www.porthedland.wa.gov.au/Profiles/porthedland/Assets/ClientData/Document-Centre/Minutes/2016/OCM25May/20160525Minutes.pdf>

7. GOVERNANCE

The 2010 Management Plan envisaged that the Department of Planning and the (then) Department of Environment and Conservation would report separately to the Premier on implementation of the Plan. However, the Premier subsequently agreed that the Taskforce would report to him through the Department of State Development, as chair of the Taskforce. Following a change in Ministerial portfolios in early 2016, the Taskforce now reports to the Hon. Bill Marmion MLA, Minister for State Development.

The main responsibility for implementing the recommendations in this report lies with planning and regulatory agencies.

RECOMMENDATION 7

The Taskforce recommends that:

- 7.1 The Taskforce continues to operate, with a focus on sharing information and co-ordinating agency activities when needed; and**
- 7.2 The Taskforce reports annually to the Minister for State Development on progress in implementing the recommendations in the report and on the overall status of dust and noise management in Port Hedland.**

GLOSSARY OF TERMS

Amendment 22	Town of Port Hedland Local Planning Scheme No. 5, Scheme Amendment 22
EP Act	Environmental Protection Act 1986
DER	Department of Environment Regulation
DSD	Department of State Development
DoP	Department of Planning
HRA	Health Risk Assessment
OEPA	Office of the Environmental Protection Authority
µg	Microgram
NEPM	National Environmental Protection Measure
Particulate Matter (dust)	Particulate Matter suspended in the air in the form of minute solid particles or liquid droplets, especially when considered an atmospheric pollutant
PM ₁₀	Particulate matter classified as having an aerodynamic diameter of 10 µm/m ³
PM _{2.5}	Particulate matter classified as having an aerodynamic diameter of 2.5 µm/m ³
2010 Management Plan	Port Hedland Air Quality and Noise Management Plan
ToPH	Town Of Port Hedland Council
West End	The Western of Port Hedland – the area generally west of Taplin Street

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