Toxikos

Preliminary screening health risk assessment and literature review

SIMTA

SYDNEY INTERMODAL TERMINAL ALLIANCE

Transitional Part 3A Concept Plan Application

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Preliminary screening health risk assessment and literature review

Moorebank Intermodal Freight Terminal, Moorebank NSW

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Toxikos Pty Ltd is a consulting company formed on December 1st 2000 to provide clients with independent excellence in toxicology and health based risk assessment. Its charter is to assist industry and government make science based decisions regarding potential effects and management of environmental and occupational chemicals. For over twelve years, prior to and since the establishment of Toxikos, staff have provided toxicology and health risk assessment advice to clients in a wide range of industries and government in Australia, New Zealand and South Africa.

About the authors: John Frangos is one of the Directors and Principal consultants of Toxikos Pty Ltd and provides toxicology, risk assessment and regulatory advice to a broad range of industries and government bodies. He has primary degrees in chemistry and pharmacology and a Masters degree in Toxicology. Mr Frangos has been a toxicology consultant to industry, Australian Federal and State Authorities. He is an expert in hazard classification of chemical substances and is currently a sessional lecturer at RMIT University teaching advanced risk assessment to postgraduate level students.

Executive summary

Hyder Consulting on behalf of the Sydney Intermodal Terminal Alliance is planning to develop an Intermodal Freight Terminal (IMT) at Moorebank, NSW (SIMTA proposal). An air quality impact assessment has been conducted to assist in gaining Concept Plan approval for the IMT as a transitional Part 3A project under the Environmental Planning and Assessment Act 1979. The SIMTA proposal will function as an intermodal rail-to-truck freight terminal with the capability to process up to 1,000,000 TEUs (twenty foot equivalents) per annum. It will be located at the site of the existing Defence National Storage and Distribution Centre (DNSDC), on Moorebank Avenue, Moorebank (the site). The site is well positioned to take advantage of existing infrastructure, being 5 km east of the M5/M7 Interchange, 2 km from the main north-south rail line and future Southern Sydney Freight Line and 0.6 km from the M5 motorway.

Toxikos were requested by Hyder Consulting to conduct a preliminary screening health risk assessment for a proposed Intermodal Freight Terminal (IMT) and Warehouse / Distribution Facility at Moorebank NSW. In addition Toxikos were requested to include a toxicology assessment on particulate matter considering in particular diesel emissions.

Air quality is one of the many parameters that can influence well being. Other parameters can include noise or social determinants of health such as the local job market, access to amenities and public spaces. Given the project is at the concept phase some components of a health impact assessment have been progressed in a preliminary manner including the potential direct effects on health of the local population due to air pollutants. The present health risk assessment seeks to predict whether the proposed intermodal transport terminal will significantly affect air quality of the inhabited area around the terminal and what the likelihood is for direct health effects should exposure to emissions occur. The assessment is of a preliminary nature as the air quality predictions for the facility are based on limited information and thus have been conservatively estimated.

Importantly the health risk assessments have been conducted in accordance with state, national, international guidelines and best practice. Because of the complexity of the assessments and the need for them to be predictive some aspects of the health risk assessments herein are necessarily of a screening nature. Assumptions have been made that bias the assessment towards protection of public health. Where a potential concern has been

identified as part of a screening process it has been subject to further refined evaluation if data allows.

The estimation of exposure in the present HRA is derived from an air quality assessment conducted by PAEHolmes (PAE 2010). The air quality assessment is preliminary in nature as the final development design, layout and operational details for the IMT are not available. The assessment therefore adopted a conservative approach assuming the IMT operated for 24-hours per day, 7-days per week continuously at the conceptual busiest hour of operations at the site. The conservatism in the emissions estimate is purposely intended to account for uncertainty in the operational details for the facility.

To achieve the above the screening HRA evaluated the following scenarios:

- Scenario 1 **(S1):** The effects of the emissions due to increased freight movements related to the proposed IMT. This is based on conservative estimations of emissions during the busiest hour of operation at maximum capacity. This is also referred to as the 'incremental' scenario. Scenario 1 is the primary scenario for assessing impacts of the IMT facility.
- Scenario 2 **(S2):** The combined effects of existing air quality (particulate matter and nitrogen dioxide) and emissions (particulate matter and nitrogen dioxide) from the proposed IMT. This is the 'cumulative' scenario.
- Although not a formal scenario a qualitative assessment of the impact of shifting freight to rail is provided within the HRA.

The basic method for characterising possible health risks has been to compare the predicted ground level concentrations for individual emission components from the IMT facility to an air guideline value established by a reputable regulatory agency for protection of public health. Air guideline values usually have large safety factors incorporated in them. The ratio of the ground level concentration to the air guideline value is called the hazard quotient (HQ). Predicted ground level concentrations (GLC) of pollutants at places where people live or gather (called receptor locations) were determined by air dispersion modelling, Toxikos was provided spread sheets of the predictions. Both the dispersion modelling, and the emission inventory data (for Scenarios 1 and 2) upon which it draws, is subject to independent peer review.

For effects that may occur from exposure to the IMT emission it has been assumed the health effects of individual components (particulate matter (PM) and nitrogen dioxide (NO_2)) may be additive with each other. This approach may inherently overestimate the overall impact on health. The health based guidelines for these emission components are based on epidemiological studies that measure the association between at least one specific pollutant (i.e. PM, NO_x, CO or O_3) and health outcomes. These specific components are usually highly correlated with other pollutants and are considered indicative of the complex pollutant mixture. It is unclear how much the associations reported in epidemiological studies represent the independent effects of specific pollutants. This correlation means that simply summing the pollutant-specific impacts can lead to an overestimation of the overall impact of air pollution on health.

Risk Assessment Results
Health impact of emissions from SIMTA Intermodal transport facility

The assessment undertaken for emissions from the IMT facility indicates that acute or chronic direct health effects are unlikely. The emissions of major importance for possible health effects are fine particulate matter ($PM_{2.5}$). Nitrogen dioxide does not contribute to the overall acute or chronic health risk estimated for the IMT facility. PM_{10} , $PM_{2.5}$ or NO_2 released from the IMT facility have negligible impact on the surrounding area, either on their own or in combination.

Cumulative health impacts of emissions from IMT in combination with existing air quality data:

Detailed operation plans have yet to be developed for the SIMTA IMT facility necessitating the emissions to be estimated using highly conservative assumptions. Existing air quality data for the Moorebank air shed are available from a nearby air monitoring station in Liverpool. Information on the IMT facility emissions has been combined with the existing data from the Liverpool air monitoring station. Not all substances in the emissions from IMT facility have been assessed (in particular criteria pollutants such as ozone and organic compounds such as PAH) nor has the veracity of the emissions estimates been assessed. Based on the available data and the substances that have been assessed it can be concluded there is low likelihood for cumulative acute or chronic health effects.

Individual concentrations of NO_2 and PM_{10} and for the most part $PM_{2.5}$ are individually below their respective health guidelines. However on rare occasions (one occasion in the period assessed excluding dust storms or other extreme weather events in Sydney) the accumulation

of particulate matter and nitrogen dioxide (mainly related to PM) can exceed the combined standards. This does not mean health effects are probable or imminent in the vicinity of the IMT facility. In reaching this conclusion it is noted the air dispersion modelling may have over-estimated ground level concentrations.

Regional health impacts of emissions from IMT:

Intermodal transport facilities in metropolitan Sydney are expected to reduce long term environmental impacts from land based container transport activities given the increased proportion of containers transported by rail (SKM 2005, Walls 2008). The SIMTA IMT will contribute to this target by reducing the levels of fuel consumption due to a reduction in the growth of container truck movements (PAEHolmes 2010). Although this will accompany and increase in locomotive emissions overall the type and quantity of emissions from fuel consumption are expected to be reduced and hence regional air quality would improve. An overall reduction in diesel related particulate emissions will likely reduce background fine particulate matter in southwestern Sydney and lead to improved health outcomes.

Contents

Acronyms and Abbreviations

AGV	Air Guideline Value
AQIA	Air Quality Impact Assessment
ATSDR	Agency for Toxic Substances and Disease Registry
CDB	Central Business District
CO	Carbon monoxide
COPD	Chronic Obstructive Pulmonary Disease
CSIRO	Commonwealth Scientific and Industrial Research Organisation
CV	Cardiovascular
[$\Delta PM_{2.5}$]	Daily Change in $PM_{2.5}$ concentrations
DNSDC	Defence National Storage and Distribution Centre
EA	Environmental Assessment
EU	European Union
enHealth	The Environmental Health Committee (subcommittee of the Australian Health Protection Committee)
FEV	Forced Expiratory Volume
GLC	Ground Level Concentration
GMR	Greater Metropolitan Region
HI	Hazard index
HQ	Hazard Quotient
HRA	Health Risk Assessment
IARC	International Agency for Research on Cancer
IMT	Intermodal Freight Terminal
IPCS	International Program on Chemical Safety
LOAEL	Low Observed Adverse Effect Level
LOEL	Low Observed Effect Level
NEPM	National Environment Protection Measure
NHMRC	National Health and Medical Research Council
NO_2	Nitrogen Dioxide
NOAEL	No Observed Adverse Effect Level
NOEL	No Observed Effect Level
NO_x	Oxides of Nitrogen
NSW DECCW	New South Wales Department of Environment, Climate Change and Water
NSW OEH	New South Wales Office of Environment and Heritage (formerly NSW DECCW)
O_3	Ozone
OEHHA	Office of Environmental Health Hazard Assessment
PAH	Polyaromatic Hydrocarbon
PM	Particulate Matter
$PM_{2.5}$	Particulate Matter less than 2.5µm in size
PM_{10}	Particulate Matter less than 10µm in size
REL	Reference Exposure Level
RIVM	The Dutch National Institute of Public Health and the Environment
S1	Scenario 1
S2	Scenario 2
SIMTA	Sydney Intermodal Terminal Alliance
STP	Standard Temperature and pressure
TEOM	Tapered Element Oscillating Microbalance
UK	United Kingdom
WHO	World Health Organization

1.0 Introduction and scope

The Sydney Intermodal Terminal Alliance (SIMTA) is consortium of Qube Logistics and QR National. The SIMTA Moorebank Intermodal Terminal Facility (SIMTA proposal) is proposed to be located on the land parcel currently occupied by the Defence National Storage and Distribution Centre (DNSDC) on Moorebank Avenue, Moorebank, south west of Sydney. SIMTA proposes to develop the DNSDC occupied site into an intermodal terminal facility and warehouse/distribution facility, which will offer container storage and warehousing solutions with direct rail access to Port Botany.

Toxikos were requested by Hyder Consulting to conduct a preliminary screening health risk assessment for a proposed Intermodal Freight Terminal (IMT) and Warehouse / Distribution Facility at Moorebank NSW. In addition Toxikos were requested to include a toxicology assessment on particulate matter considering in particular diesel emissions.

PAEHolmes were engaged to prepare an Air Quality Impact Assessment (AQIA) to form part of the Environmental Assessment (EA) to assist with the concept phase approval. The PAEHolmes report identified infrequent exceedences of fine particulate matter. Given that air quality is one of a range of important variables to the health of the local population this report focuses on direct health effects due to air quality. It does not address risk versus benefits nor is it a health impact assessment.

1.1 What is a health risk assessment?

NSW Health (2009 – Healthy Urban Checklist and supporting documentation) defines ‘environmental health’ as “the interaction between the environment and the health of populations of people” and "those aspects of human health determined by physical, biological, and social factors in the environment”.

The following are examples of determinants of health and well-being (enHealth 2001, enHealth 2004, Harris et al. 2007, WA Health 2007, Mahoney et al. 2004, NSW Health 2009):

- 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).

According to enHealth (2004) all developments have a potential impact on health. Some will have positive health impacts by providing jobs, attracting health services to an area, and improving overall economic well being of a community etc. Other projects may have negative impacts such as increased risk of disease, social disruption, increased noise etc. Many developments will have both positive and negative aspects. The potential influence of the proposed expansion on local area economic factors, social disruption through changed traffic patterns and other such factors are addressed in various sections of the regulatory planning and approval processes (NSW Health 2009).

Air quality is one of the many parameters influencing well being. Given that the IMT facility involved the storage, handling and transport of up to 1,000,000 containers per annum one of the potential direct impacts on health is air pollution.

The **health risk assessment** seeks to predict, whether the proposed intermodal transport terminal will significantly affect air quality of the inhabited area around the terminal and what the likelihood is for direct health effects should exposure to emissions occur. The assessment is of a **preliminary nature** as the air quality predictions for the facility are based on limited information and thus have been **conservatively estimated**.

A prospective assessment of the holistic nature of health as per the WHO definition is usually termed a **health impact assessment**. These are most often done during the policy and planning cycle after a draft proposal has been developed and before it is implemented (Harris et al. 2007). A health risk assessment (HRA) is an analysis that uses information about hazardous (perceived or real) substances (e.g. diesel particulates) to estimate a theoretical level of risk for people who might be exposed to defined levels of these substances in the future. The information comes from scientific studies and measurement data of emissions. The risk assessment helps regulatory officials, facility managers and the public determine strategies that will ensure overall protection of human health and the environment should the proposed development proceed. In other words the risk assessment is undertaken to help define the boundaries/conditions under which the proposed development may obtain approval. This usually only occurs when the regulatory authorities are satisfied that the appropriate conditions have been put in place to ensure the future safety of the public.

It is important to note that a prospective risk assessment does not measure the actual health effects that hazardous substances may have on a community because the development project has not yet taken place. Risk assessments are often conducted by considering possible or theoretical community exposures predicted from air dispersion modelling of 'known' concentrations of emissions from a specific point of release at the intermodal terminal. Conservative safety margins are built into a risk assessment analysis to ensure protection of the public. Therefore people will not necessarily become sick even if they are exposed to materials at higher dose levels than those estimated by the risk assessment. In other words, during the risk assessment analysis, the most vulnerable people (e.g., children and the elderly) are carefully considered to make sure all members of the public will be protected.

The risk assessment helps answer common questions for people who might be exposed to hazardous compounds in the environment, in this case components of the emissions from the Moorebank intermodal terminal. These include:

- Under what circumstances might I and my family and neighbours be exposed to hazardous substances from this proposed development?
- Is it possible we might be exposed to hazardous substances at levels higher than those determined to be safe?
- If the levels of hazardous substances are higher than regulatory standards, what are the health effects that might occur?

1.2 Description of the intermodal transport facility

The SIMTA site is located in the Liverpool Local Government Area. It is 27 kilometres west of the Sydney CBD, 17 kilometres south of the Parramatta CBD, 5 kilometres east of the M5/M7 Interchange, 2 kilometres from the main north-south rail line and future Southern Sydney Freight Line, and 0.6 kilometres from the M5 motorway.

The SIMTA site, approximately 83 hectares in area, is currently operating as a Defence storage and distribution centre. The SIMTA site is legally identified as Lot 1 in DP1048263 and zoned as General Industrial under Liverpool City Council LEP 2008. The parcels of land to the south and south west that would be utilised for the proposed rail link are referred to as the rail corridor. The proposed rail corridor covers approximately 37 hectares and adjoins the Main Southern Railway to the north. The rail line is approximately 2.5 kilometres in length and includes two connections to the SSFL, one south and one north.

The proposed rail corridor is owned by third parties, including the Commonwealth of Australia, RailCorp, private owners and Crown Land held by the Department of Primary Industries, and would link the SIMTA site with the Southern Sydney Freight Line. Existing uses include vacant land, existing rail corridors (East Hills Railway and Main Southern Railway), extractive industries, and a waste disposal depot. The rail corridor is intersected by Moorebank Ave, Georges River and Anzac Creek. Native vegetation cover includes woodland, forest and wetland communities in varying condition. The proposed rail corridor is zoned partly 'SP2 Infrastructure (Defence and Railway)' and partly 'RE1 - Public Recreation'. The surrounding Commonwealth lands are zoned SP2 Infrastructure (Defence).

The location of the site is shown in Figure 1.1. The figure identifies the location of the discrete receptor locations (7 receptors) chosen as part of the air quality impact assessment (PAE 2010). The locations were chosen as representative of the closest residential areas surrounding the site.

It is proposed that the Moorebank Intermodal Terminal will provide an integrated transport solution for the movement of freight to, from and within the Sydney metropolitan area. It is envisaged that this will alleviate urban congestion and address the critical shortage of freight handling capacity in Sydney. A staged redevelopment of the Intermodal Terminal is expected to commence in 2013 (subject to planning approval), with the first stage of the Intermodal Terminal expected to be operational by early 2016.

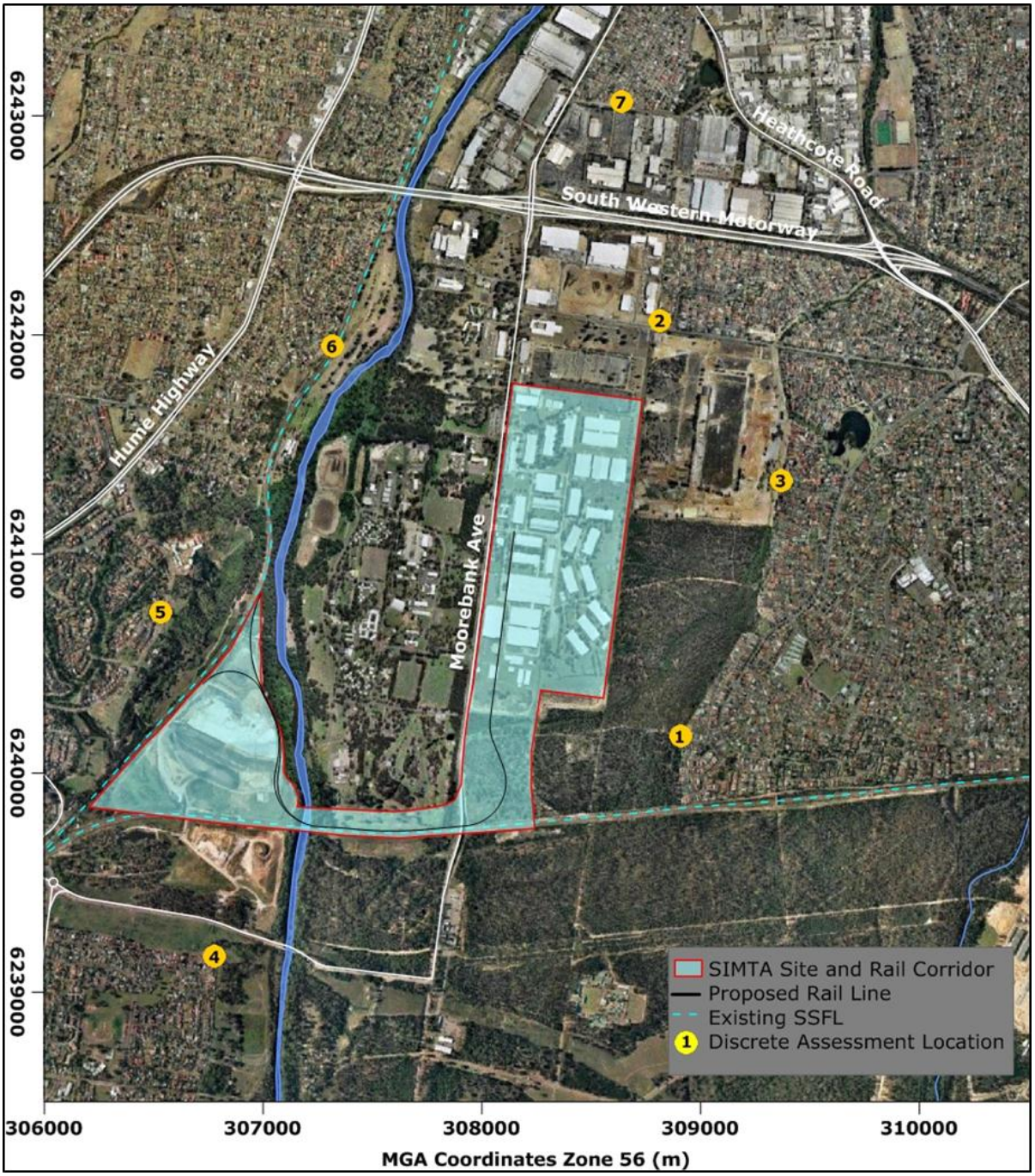


Figure 1.1: Local setting and location of receptors.

1.3 Scope of the risk assessment

The screening 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.

SCOPE:

A screening risk assessment.

It is preliminary because a detailed assessment of air quality impacts is not yet available. Thus emissions have been conservatively estimated.

Primarily for impact of emissions from the proposed transport hub, the IMT.

It includes a discussion of particulate matter health effects with a particular emphasis on diesel exhaust.

1.4 Description of emission scenarios

The primary purpose of the preliminary risk assessment is to evaluate emissions from the proposed IMT and warehouse/distribution facility. In addition it is important to understand whether or not the emissions from the proposed IMT are significant additions to the air shed surrounding the site. The HRA does not consider the impact of the IMT on reducing the overall transport related particulate emissions in metropolitan Sydney (PAE 2010), except to note that this is one of the reasons for the proposed project. To achieve the above the screening HRA considers impacts on health in two scenarios:

- Scenario 1 **(S1):** The effects of the emissions due to increased freight movements related to the proposed IMT. This is the 'incremental' scenario.
- Scenario 2 **(S2):** The combined effects of existing air quality (particulate matter and nitrogen dioxide) and emissions (particulate matter and nitrogen dioxide) from the proposed IMT. This is the 'cumulative' scenario.

As air dispersion modelled data related to emissions from the proposed IMT are only available for particulate matter and nitrogen dioxide this HRA is focussed on the health risks due to these compounds: i.e.: the significance of these emissions to the overall air quality in the locality (i.e. IMT facility + background). It is beyond the scope of the present HRA to assess any current

potential health impacts of existing air quality in metropolitan Sydney or any benefits due to a reduction in overall emissions to the air shed.

1.5 What is included in the preliminary screening HRA

The HRA is referred to as a preliminary assessment for two reasons:

- The air quality impact assessment is based on concept phase information for the project. The final development layout for the site has not been determined, these are necessary to accurately predict emissions from the IMT. In the absence of these details conservative assumptions that are likely to overestimate emissions have been modelled. For instance:
    - The modelling has been conducted assuming the busiest hour of operation (86 trucks per hour assuming a 24 hour, 7 day a week operation).
    - The modelling has assumed conservative values for inputs related to fleet profiles, time spent idling, locomotive time in mode and average speeds.
    - The cumulative scenario (Scenario 2) does not consider the potential reduction in the emissions of particulate matter and nitrogen dioxide due to the reduction in heavy goods vehicle traffic using the M5 corridor particularly during peak times.
- Not all emission components have been included in the air dispersion modelling for the IMT. In particular ozone, sulphur dioxide, carbon monoxide and polycyclic aromatic hydrocarbons are potentially important emission components to consider. The air quality impact assessment included air dispersion modelling for particulates and nitrogen dioxide. To accommodate the absence of these substances a literature review of the health effects of diesel exhaust emissions has been included focussing on diesel exhaust particulates.

The HRA is referred to as a screening HRA as it compares air dispersion modelling results represented as predicted ground level air concentrations at seven discrete locations at close proximity to the Moorebank IMT directly to ambient air guideline values. It assumes that resident exposure can be attributed to the ambient air concentrations.

Although this risk assessment is quantitative there are aspects that are primarily of a screening nature due to the fact that it deals with risks for a person who is hypothetically exposed to the highest atmospheric emission concentration that is reasonably expected to occur at the nominated receptors.

The purpose of a screening risk assessment is to efficiently determine if, at the predicted exposures, health impacts are possible and if so discover the likely contributions of each causative agent. Thus the risk assessment herein uses a number of procedures to decide which of the emission components either on their own or as a mixture are potential threats to public health and hence important for further detailed assessment. By necessity, to ensure protection of public health the risk assessment is conservative, that is it errs on the side of safety by over predicting the likelihood for health risk. However to provide reality and contextual information in the assessment a qualitative discussion has been undertaken for the uncertainty inherently embedded in the assessment and the level of conservatism used to account for unknown and missing information. Although aspects of uncertainty are discussed within the section where a particular topic is discussed, they are drawn together in Section 7.

The potential for direct health effects has been evaluated for predicted short term and long term exposures. These are called the acute and chronic health effect evaluations respectively (Sections 6.1 & 6.2 respectively).

As described in section 4 the likelihood for the most sensitive health effect of an emission component has been characterised using a health based air guideline value sourced from authorities. Ambient air guidelines are established to protect the general population (inclusive of sensitive sub-groups) against the most sensitive health effect associated with the chemical. The most sensitive health effect is the one that occurs with the lowest level of exposure.

2. Issue identification

The Sydney Intermodal Terminal Alliance (SIMTA) intends to develop an Intermodal Freight Terminal facility (IMT) which is a significant change to the existing land use for the site. Although the SIMTA site is currently used as a distribution centre (Defence National Storage and Distribution Centre) the proposed IMT will have a much larger capacity to receive and transfer goods with the capacity to process up to 1,000,000 twenty foot equivalent containers per year.

The terminal facility operations will involve freight being loaded onto trains at Port Botany, directly transporting containers to Moorebank on a dedicated freight line, unloading the containers at Moorebank into warehouses on site or onto trucks for delivery to businesses and warehouses across southwest Sydney. This operation would also work in reverse, taking freight containers to Port Botany.

The expanded freight rail capacity that will be achieved through development of the Moorebank Intermodal Terminal is intended to support the New South Wales Government's target of increasing the share of freight that is transported from Port Botany to Sydney's intermodal facilities by rail to 40% by 2025, from 23% in 2009 (Urbis 2010).

During operation the proposed IMT will emit diesel vehicle exhaust (locomotives, trucks and container handling equipment). Fugitive emissions are also likely due to vehicle movements on and off the site (PAE 2010). On a regional scale however fugitive emissions are predicted to decrease.

A preliminary social impact statement for the proposed IMT provides a summary of key issues identified for the future development of a Social Impact Assessment as part of a concept phase environmental impact assessment. There are a number of issues that concern members of the local community. These are mainly focused on actual or perceived traffic increases on local roads and the associated safety, noise and air quality impacts. Although the proposal would lead to a relative reduction in heavy vehicle traffic across the Sydney metropolitan area, there may be some additional traffic movements on certain arterial roads close to the SIMTA IMT.

One of the key issues and concerns raised by the general community to-date include the impact of the IMT facility on air pollution and health of families (Urbis 2010).

In addition the Concept Phase approval air quality impact assessment report (PAE 2010) identified the potential for small exceedences to the $PM_{2.5}$ Australian advisory reporting standard. It is important to emphasise that the concept phase air quality report is based on worst case assumptions that will result in a conservatively high prediction of impact.

2.1 Demographics

The site is located in south west of Sydney in the suburb of Moorebank NSW. It is located approximately 27 kilometres south-west of the Sydney central business district in the local government area of the City of Liverpool. Moorebank features a mix of residential and industrial areas. The site is near two major arterial roads the South Western motorway (M5) and the Hume Highway.

The nearest residential area can be found within 0.2 km to the north east of the site on the corner of Anzac Rd and Delvin Drive. These residences are between the IMT facility and the M5. Residences within 0.5 to 2 km can be found to the east and west of the proposed Moorebank IMT (site).

Based on 2006 census data, the local suburb of Moorebank had a population of 7,599 persons (Urbis 2010). Table 2.1 summarises the age distribution for residents which is similar to the national average with the exceptions of persons aged 0-24 years (slightly higher proportion in Moorebank) and a higher proportion of people aged 55-64 (14.4% compared to 11% for Australia) (Urbis 2010).

Table 2.1: The age distribution for residents of Moorebank compared to the National Average

Age Group	Moorebank	National Average
0-4 years	8.10%	6.30%
5-14 years	12.70%	13.50%
15-24 years	13.20%	13.60%
25-54 years	41.80%	42.20%
55-64 years	14.40%	11.00%
65 years +	9.70%	13.30%

During specific periods of life (i.e. childhood and advanced age), individuals may be more susceptible to environmental exposures, which in turn can render them more susceptible to particulate matter related health effects. An evaluation of age-related health effects suggests that older adults have heightened responses for cardiovascular health effects with particulate matter exposure (US EPA 2010). Section 4 provides additional information.

2.2 Existing air quality

There are many sources of particles in the air, arising from both natural processes and human activity. The main sources of particle emissions from human activity in the Sydney region[1] are industrial activities, mobile sources (both on- and off-road) and domestic sources (primarily the use of domestic solid-fuel heaters). These emissions can be trapped below temperature inversions, particularly during winter. If this weather persists for several days, elevated concentrations of fine particles may occur.

The dominant source of PM_{10} in Sydney is from industrial premises (37%), domestic sources and on-road mobile sources make up a greater proportion of PM_{10} emissions in Sydney than they do in the GMR. The annual domestic sector contribution to PM_{10} emissions in Sydney comes largely from wood heating (93%). The geographic distribution of annual emissions of PM_{10} for the Sydney region is shown in Figure 2.1.

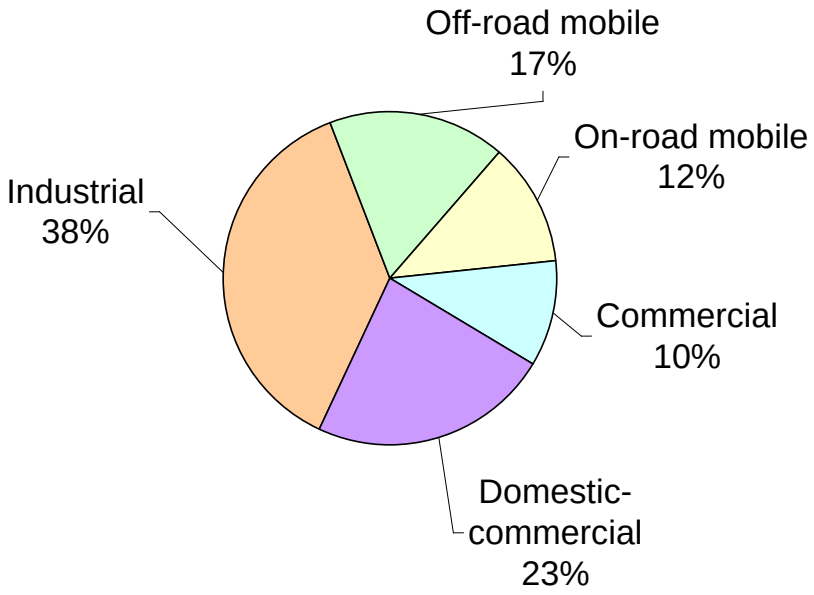


Figure 2.1: Major sources for anthropogenic particulate matter. From NSW DECCW (2010)

[1] NSW Government operates 14 measurement stations in the Sydney region.

Fine particle concentrations (PM_{10} and $PM_{2.5}$) in NSW are usually low. Despite the generally low concentrations of PM, Sydney does occasionally experience extreme PM concentrations due to bushfires and dust storms. Concentrations greater than the Air NEPM standard are uncommon (NSW DECCW 2010). Changes in daily PM_{10} from one day to the next by a concentration of 10 µg/m^3 or greater are very rare and generally these events are due to extreme events such as dust storms or bush fires (NSW DEC 2005, NSW DECCW 2010). The general trends for Moorebank NSW are consistent with those for Sydney.

The Liverpool air quality monitoring site is located in the Council depot, off Rose Street, Liverpool which is approximately 1.7 km from the site. Data from this monitor was considered in the present HRA and the way it is included is described in Section 3.

3. Exposure assessment

3.1 Exposure pathways and exposure estimations

Exposure to pollutants from the additional freight movement due to the proposed IMT is most likely to be by direct exposure. Inhalation is by far the most important exposure route and is the primary subject of this preliminary HRA.

Estimation of exposure to emissions at any given receptor location relies upon:

i) Determination of what is in the emissions;
ii) Determination of the concentration of emission components at point of release to atmosphere; and
iii) Dispersion modelling to predict the 'ground level concentration' of contaminant at locations where people may live or spend appreciable amounts of time.

The estimation of exposure in the present HRA is derived from an air quality assessment conducted by PAEHolmes (PAE 2010). The air quality assessment is preliminary in nature as the final development design, layout and operational details for the IMT are not available. The assessment therefore adopted a conservative approach assuming the IMT operated for 24-hours per day, 7-days per week continuously at the conceptual busiest hour of operations at the site. The conservatism in the emissions estimate is purposely intended to account for uncertainty in the operational details for the facility.

The air quality assessment, provided:

i) A description of the ambient receiving environment, including background criteria pollutant concentrations, prevailing meteorological conditions and nearby sensitive receptors;
ii) Quantified emissions to air for the operation of the IMT for particulate matter and nitrogen dioxide; and
iii) Consideration of the broader regional impacts from the project, in terms of improved freight handling in Sydney.

3.1.1 What is in the background?

The closest air quality monitoring station in the South-west Sydney region to the proposed IMT is situated at Liverpool. The monitoring equipment is located within the Liverpool council depot off Rose Street and approximately 1.7 km to the North-west of the nearest boundary for the

proposed IMT. Both sites are intersected by the Hume Highway and South Western Motorway and are in close proximity to receptors.

The background data from the Liverpool monitoring station was utilised to estimate background concentrations in ambient outdoor air for NO_2, $PM_{2.5}$ and/or PM_{10} at the discrete locations assessed within the present HRA. The data is monitored on-site using a Tapered Element Oscillating Microbalance (TEOM).

The background ground level concentrations for NO_2 and particulate matter (PM_{10} and $PM_{2.5}$) were downloaded from the Liverpool air monitoring station. Monitoring data was provided for the year 2009 by PAE (2012a, 2011). The averaging time of monitoring data corresponded to the requirements of air guidelines values for each pollutant based on the averaging times for the toxicity reference values. For $NO_2$1-hour averaging data were required while for particulate matter 24-hour averaging data was provided.

Extreme weather events have occurred in the Sydney Metropolitan during 2009 and resulted in elevated 24-hour PM concentrations. Regional dust storms in 2009 resulted in some of the highest ever recorded particulate matter readings in Sydney including at the Liverpool monitoring station which, on the 23rd of September 2009, recorded a 24-hour PM_{10} concentration of 1,580µg/m^3. Particulate matter data collected during dust storm events have not been included in this assessment (see section 6.1.3).

The concentrations for the PM_{10}, $PM_{2.5}$ and NO_2 fractions are provided in Appendix 2 for each of the percentiles at each receptor location. The background concentration percentiles are provided below in Table 3.1. The PM_{10}, $PM_{2.5}$ and NO_2 percentiles show a large decrease from the maximum GLC down to the 95th percentile and the 50th percentile that approximates the average.

Table 3.1: Percentiles for background data in µg/m^3 (NO_2, $PM_{2.5}$ and PM_{10})

Pollutant	Averaging Time	Percentile					
		50th	95th	99th	99.7th	99.9th	Max
NO_2	1-hour	18	47	59	72	88	109
$PM_{2.5}$	24-hour	7	14	19	20	25	27
PM_{10}	24-hour	18	33	41	43	44	44

3.1.2 What is in the emissions from the IMT?

Emissions from the proposed IMT and associated warehousing/distribution centre were attributed to particulate matter and NO_2 (PAE 2012). These pollutants are suspected to be primarily associated with vehicle emissions due to increased freight movement resultant from the IMT and warehouse operations.

PAE (2012) provided modelling data for NO_2 and particulate matter (size fraction not specified). Modelling data for NO_2 was calculated from NO_x transformations and was used in the current assessment as provided by PAE (2012). However modelling data relating to particulate matter did not specify the size fraction modelled and is assumed to represent PM_{10}. As such the modelled data provided by PAE(2012) for particulate matter was apportioned to the PM_{10} and $PM_{2.5}$ fractions before being used in the current assessment. This apportionment requires an understanding of the emissions associated with increased freight transport, including the proportion comprised of fine particles and that of coarse particles.

Dispersion modelling using a Gaussian plume model (Ausplume) was conducted in accordance with the DECCW Approved methods (NSW DEC, 2005) by PAEHolmes (2010).

Particulate matter is a mixture of solid particles and liquid droplets which vary in concentration, composition and size distribution. Particles generated by the build up of condensable vapour in the atmosphere act as nuclei for the formation of larger accumulate particles (Morawska, 2002). Fine particles (or particles with aerodynamic diameters smaller than 2.5µm; $PM_{2.5}$) are primarily generated from combustion processes and photochemical reactions followed by gas to particles conversion. While coarse particles are generated from mechanical processes including grinding, breaking and wearing of crystalline material and dust resuspension (Morawska, 2002).

Within the context of the current assessment, fine particles are likely to be largely comprised of vehicle exhaust emissions. According to Morawska (2002), particulate matter emitted from exhausts "*are mostly submicrometre agglomerates of solid phase carbonaceous material ranging in size from 30 to 500nm and residing mainly in the accumulation mode*". Vehicle exhausts are a major emitter of fine particles present in traffic influenced urban environments with the majority of the contribution from diesel engines that is two to three magnitudes higher than petrol vehicles (Morawska et al., 2005; Ristovski et al., 2005; Ristovski et al., 2006, Morawska 2002)

The particle size distribution and particle count of particulate matter emitted from engines running on different fuel are shown in Table 3.2 below. Particle distributions can be presented as number or mass distributions of particles size. When a single pollution source is investigated under steady state conditions the size distribution of particles is likely to have one distinctive peak and sometimes additional smaller peaks. These peaks are referred to as modes of the distribution (Morawka, 2002). In Table 3.2 the modes range from 0.011 to 0.085µm (i.e.: particle size at maximum peak height). Note that modes based on mass are likely to be higher than modes determined by particle counts. This is because the fine particles emitted by diesel exhausts are prolific in number however due to their size they have negligible mass.

Table 3.2: Modes (µm) for various fuel engines based on particle number

Emission	Major mode (µm)	Mode range (µm)	Source
Diesel Engines	0.05 0.085	0.01 to 0.3 0.02 to >0.2	Morawska (2002) Jayartne (2009)
Gas Engines	0.07 0.011	0.01 to 0.5 0.005 to >0.2	Morawska (2002) Jayartne (2009)
Petrol Engines	0.04	<0.01 to 0.3	Morawska (2002)

External factors such as wind speed (dispersion, dilution and/or resuspension of particles), precipitation (washout effect), humidity and temperature may affect the physical and chemical characteristics of particulate matter emitted from engines. Particles may transform after emission hence may exhibit different physical and chemical characteristics as well as concentration at a distant location to the source. These sources however continue to primarily contribute to the $PM_{2.5}$ fraction. The majority of vehicle emissions, even at distance, are less than 2.5µm in size and contribute most to the $PM_{2.5}$ fraction.

To a lesser extent fine particles may be comprised of particles generated on and off-site by vehicle mechanical processes that create brake dusts, mechanical repair and resuspension of road dusts. These dusts typically range from 1µm to above 10µm and hence contribute to both the $PM_{2.5}$ and PM_{10} fractions of particulate matter (Morawska 2008, CSIRO 2000). Such emissions are likely to represent the primary constituent of coast particulate matter in the current assessment. In addition, emissions from vegetation, wind-blown dusts, and sea-salt also contribute to background the particulate matter concentrations (Morawska 2008, CSIRO 2000).

CSIRO (2000) conducted a study on the chemical and physical properties of fine particulate matter in six Australian cities. The study provides data on the size distribution of automotive particulate matter monitored over five 6-day sampling periods from August 1996 to September 1996. Size distributions were constructed according to the mass of particles and indicated that the particle sizes for elemental carbon ranged from 0.04 to 20µm and exhibited trimodal characteristics (i.e.: three peaks). Each mode was identified within the $PM_{2.5}$ fraction. The major mode occurred at 0.6µm, with minor modes occurring at each 1.5µm and 0.09µm. The majority of the mass of particulate matter was located below 2.5µm on the size distribution (CSIRO, 2000).

Based on CSIRO monitoring data automotive particulate matter emissions are primarily comprised of $PM_{2.5}$. As such the current assessment conservatively estimates that 90% of the particulate matter emissions modelled by PAE (2012) are equal to or less than 2.5 µm with the remaining 10% assumed to be greater than 2.5 µm.

PAE Holmes (PAE 2012) provided Toxikos with spread sheets of predicted ground level concentrations at each receptor and relevant averaging times for particulate matter and NO_2 (data provided in Appendix 2). From this data, 90% of the particulate matter concentration was apportioned to the $PM_{2.5}$ fraction and 100% apportioned to the PM_{10} fraction. Ground level concentrations (GLC) for NO_2, PM_{10} and $PM_{2.5}$ were calculated for the maximum, 99.9th, 99.7th, 99th, 99th and 95th percentile for averaging times that matched the health guidelines for the compounds.

3.2 Where are people exposed?

The air dispersion modelling conducted by PAE (2010) provided probability estimates for ground level concentration frequencies (percentiles) at discrete locations listed in Table 3.3 below. In risk assessment terminology the locations are called receptors. Most of the locations were determined by PAE (2010) because they were the closest residential receptors surrounding the site.

Table 3.3: Receptor locations for which air dispersion for prediction of ground level concentrations of emissions was conducted.

Receptor ID	Receptor Type	Location	Distance and direction from nearest boundary
1	Residential	End of Yallum Ct	0.5 km southeast
2	Commercial	Cnr Anzac Rd and Delvin Dr	0.2 km north
3	Residential	end of Martindale Ct	0.5 km east
4	Residential	Goodenough St	2 km southwest
5	Residential	Leacocks Ln	1.5 km west
6	Residential	Buckland Road	0.8 km west
7	Residential	Church Rd	1.4 km north

[a] The receptor number corresponds to that on Figure 1.1.

3.3 How much are people exposed?

The extent people are exposed to background pollutants and emissions from freight movements related to the proposed IMT is determined by two major factors; how much is in the air and the behaviour of the person.

How much is in the air?

Concentrations of pollutants in the air at Liverpool are not constant; the concentration varies according to the direction and strength of the wind, time of day, how far away the location is from the emission source etc. Sometimes the pollutant concentration may be high for a short time but not present at other times and will be between these extremes for varying periods. Most of the time the concentration will not be zero but nonetheless tend to be below guideline values. Air dispersion modelling is used to predict the average concentration in the air for a specific time, for example 1 hour or 24 hours. These are referred to as the predicted 1 hour average or the 24 hour average ground level concentrations[2]. The dispersion modelling works by matching the patterns of emissions from a specific source with the variability of winds (the meteorology) that occur over a year in the general area. In this case the emissions are either 'background' air concentrations for this assessment, or the patterns of emissions from the proposed IMT which are determined by the extra freight movements related to the proposed transport facility.

[2] The terminology 'ground level concentration' is not actually the concentration at the ground but rather 2m above the ground level (an approximation for the breathing height of adults). To atmospheric scientists who are concerned about what happens some hundreds of metres in the air this is 'ground level'.

The dispersion modelling provides a statistical probability for the number of averaging periods in a year the concentration of a pollutant will be at a certain level. The concentrations of most interest are the high ones because these are the ones most likely to affect people. If the high concentrations are less than the exposures needed to cause a health effect then it logically follows the lower ones will also not be of health concern. The output of the dispersion modelling is a list of air concentrations and how often they occur during a year of typical meteorology. These are expressed as percentiles, and can be pragmatically regarded as code for the number of times a concentration will occur during the year, or the number of times during the year a person is likely to be exposed to a certain concentration if they are at the same spot at the same time the high concentration occurs. Table 3.4 provides the key to the code. Only the high percentile concentrations are used in the risk assessment. These represent maximum or near maximum exposures.

Table 3.4: Frequency percentiles and number of times they occur for a particular averaging time.

	Frequency Percentile				
	95th	99th	99.7th	99.9th	Max
Approx number of times [a] per year a **1 hour** average concentration might occur at a given percentile.	438	88	27	9	1
Approx number of times [a] per year a **24 hour** average concentration might occur at a given percentile.	19	4	1	1	1

[a] The number of times a percentile will occur has been rounded up to a whole number. For example the concentration at the 99.9th percentile for the 1 hour and 24 hour average occurs 8.76 and 0.36 times. An alternative way of interpreting percentiles is as the ranking of concentrations from high to low. For example the maximum and 99.9th 1 hour average concentrations are respectively the highest and the 9th highest concentrations that are predicted to occur.

Behaviour of the person:

Whether or not a person is affected by a pollutant in air from an industrial source requires them to be present at the location at the same time the high concentration occurs. However people do not spend all their time in one spot, for example an average adult only spends 1.5 hours outdoors per day (US EPA 1997). Given that people also move around during the time they spend outdoors, the chance of being present when a very high concentration of pollutant from a point industrial source occurs only a few times per year is therefore quite low.

In contrast the chance of being exposed is much more likely when the pollutant is from a number of sources in an area and the resultant pollution is spread over a wide area. This is what happens with the background particulate matter and nitrogen dioxide in the Sydney Metropolitan Area. Pollutants from various sources including vehicles becomes trapped by an

inversion layer in the air and cannot readily disperse. Instead it hangs in the air, and a relatively even concentration occurs over a wide area. In this situation, outdoor movement from one place to another does not decrease one's chance of being exposed.

To factor a person's behaviour (i.e. average daily movements) into a risk assessment is quite challenging, and is rarely done. Instead, an assumption is made that throughout their entire life a person is in a situation where they could be exposed to the highest concentrations predicted to occur by the dispersion modelling. This assumption adds conservatism (i.e. safety) into the risk assessment.

4. Hazard identification / toxicity

Appendix 1 includes health effects information for both particulate matter and nitrogen dioxide. In particular the information is centred on sensitive endpoints upon which the air guideline values were set as well as an overview of the basis for establishment of the air guideline value. The health effects of diesel particulates is summarised in the context of the effects of particulate matter in general.

The health effects review is based on toxicology profiles and electronic databases produced by competent agencies[3] rather than conducting a thorough toxicological evaluation for each chemical. The information in Appendix 1 does not always take into consideration the exposures necessary to cause the health effects stated.

Health effects in the present report are classified as either *acute* or *chronic* effects:

- Acute effects generally relates to effects occurring to single exposures of a short duration of exposure. In general short duration can range from a few minutes to 24 hours. For air pollution health effect conclusions are based on consideration of studies that have found associations (correlations) between community average exposures (i.e. not individual exposures) and deaths, hospital admissions, restricted activity days, exacerbation of conditions such as asthma or other respiratory or cardiovascular system symptoms (US EPA 2010, NEPC 2010, NSW DEC 2005). The information for acute effects for air pollution is also drawn from controlled human volunteer experiments and toxicological studies (i.e. controlled animal or cell culture scientific studies).
- Chronic effects refer to health effects associated with repeated and prolonged exposure to ambient (i.e. comparatively low levels) of air pollution. Conclusions about the health effects of air pollutants are drawn from similar studies to those for acute health effects but with a longer duration of exposure. For instance human studies investigating chronic effects normally rely on relationships drawn between community exposures defined as an annual average or animal toxicology studies conducted over an extended period of the animal's lifespan.

[3] National Environment Protection Council (NEPC), Australia; World Health Organisation (WHO)-International Programme for Chemical Safety (IPCS) & International Agency for Research on Cancer (IARC); Agency for Toxic Substances and Disease Registry (ATSDR), US Dept Health & Human Services; Office of Environmental Health Hazard Assessment (OEHHA), California EPA; The Dutch National Institute of Public Health and the Environment (RIVM); and the Integrated Risk Information System (IRIS), US EPA. Wherever it has been practical to do so, the hierarchal preferred reference list of enHealth (2004) has been used to source guidelines.

Where possible, the health effects information includes a description of sensitive subgroups in the population; for particulate matter and nitrogen dioxide these include:

- people with existing disease (mainly respiratory and cardiovascular),
- people with infections such as influenza and pneumonia,
- asthmatics,
- the elderly and
- potentially children.

Practically all of the information on air pollution (including particulate matter and nitrogen dioxide) is drawn from epidemiological studies. Epidemiology is the study of diseases in human populations. Epidemiological studies aim to identify correlations between ambient air pollution and human health so as to consider relationships that may be drawn between the level of exposure and response in the general population.

Epidemiology is an observational science and this is both a strength and weakness. It is able to evaluate health outcomes in real people, living in normal environments and exposed to typical concentrations of air pollution. However there are many confounding factors such as differing techniques and averaging times for measuring air pollutants in studies, differing individual exposures, different sources and contributions of individual pollutants within an air shed, lag time and latencies for health effects and differences in classifications and recording of health status both within and between studies.

The usual approach of epidemiological studies is to measure the association between at least one specific pollutant (e.g. particulate matter, nitrogen dioxide, ozone or carbon monoxide) and health outcomes. These specific components are usually highly correlated with other pollutants and are considered indicative of the complex pollutant mixture. It is unclear how much the associations reported in epidemiological studies represent the independent effects of specific pollutants. This correlation means that simply summing the pollutant-specific impacts could lead to an overestimation of the overall impact of air pollution on health (NSW DEC 2005).

For particulate matter, a threshold exists at the individual level. Realistically, most people are not at risk of severe acute health effects at current background levels. However, substantial evidence indicates that there is no threshold at the population level.

Information in Appendix 1 has been integrated in all sections and considerations in the HRA. For instance the selection of endpoints and their averaging times is dictated by the literature

review and the risk characterisation follows the health effects of each substance. For example the risk characterisation of of direct chronic health effects and the discussion around Figure 6.4.

5. Risk characterisation

5.1 Introduction to hazard quotients and the hazard index

For assessing the potential health impact of particulate matter and NO_2, predicted ground level concentrations are compared to individual health based ambient air guidelines generated to protect public health. This comparison is performed by calculating a hazard quotient[4] (HQ) which is the ratio of ground level concentration (GLC) to the ambient air guideline value (AGV)[5].

Thus a hazard quotient is calculated for each contaminant using the simple equation below:

$$HQ = \frac{GLC}{AGV} \qquad \textbf{Equation 1}$$

For assessing the potential effects of the combined exposure to NO_2 and particulate matter in the emissions it has been assumed individual components may have additive effects and an overall hazard index (HI) is calculated (US EPA 2000a). The hazard index (HI) is the sum of particulate matter ($PM_{2.5}$) and nitrogen dioxide hazard quotients determined from either the acute or chronic AGV[6], thus an acute and a chronic hazard index can be generated.

$HI_j = \sum HQ_{i \ldots j}$ **Equation 2**

[4] Some investigators call the 'hazard quotient' the 'hazard ratio' (e.g. Fox et al. 2004, Tam and Neuman 2004).

[5] The hazard quotient is commonly reported to one significant figure (US EPA 1989). For example, a hazard quotient of 0.13 is rounded to 0.1, and a hazard quotient of 1.6 is rounded to 2. In this risk assessment HQs and HIs have been calculated to two significant figures. This is not to imply there is a level of precision in the assessment; far from it, it has been done merely to allow proper accounting of the summing of HQ's in the spread sheets and better inform readers of the differences for individual receptors and scenarios.

[6] Health based guidelines inherently contain safety factors to protect against ill health being caused by exposure to the chemical. The hazard index is not an evaluation predicting whether health effects will/will not occur, but rather whether the health guideline value will/will not be exceeded. If the health guideline is not exceeded then it follows that health effects are unlikely to occur, if the health guideline is exceeded it does not naturally follow that health effects will occur. This is because of the conservatism embedded in the exposure estimate (i.e. the numerator of equation 1 which is the modelled GLC) and the uncertainty (safety factors) used to establish the health guideline value (i.e. the denominator of equation 1). The uncertainty factors used in the derivation of the health based air guideline value by competent agencies is included in Appendix 1 of this risk assessment, this information provides an appreciation of the margin between the AGV and the exposure that may actually be required to cause an effect.

Where HI_j is the sum of HQ's for all pollutants from i to j

This process assumes:

- there is a threshold level of exposure below which no adverse health effects will occur,
- either the toxicological effect of chemicals and/or the dose is additive, and
- multiple sub threshold exposures may result in an adverse health effect.

Urban air pollution is a complex mixture of many known and unknown substances. The total impact of air pollution on health is the sum of:

- all independent effects of specific pollutants
- the effects of mixtures, and
- the additional effects due to interactions between pollutants (that is, chemical reactions occurring in the air or in the course of inhalation, which may enhance or reduce the effects of individual pollutants (Kunzli et al., 1999 cited in NSW DEC 2005).

The usual approach of epidemiological studies is to measure the association between at least one specific pollutant (e.g. PM, NO_x, CO or O_3) and health outcomes. These specific components are usually highly correlated with other pollutants and are considered indicative of the complex pollutant mixture. It is unclear how much the associations reported in epidemiological studies represent the independent effects of specific pollutants. This correlation means that simply summing the pollutant-specific impacts could lead to an overestimation of the overall impact of air pollution on health.

In strict toxicological terms it is only valid to sum the effects and/or dose of chemicals if they have the same mode of toxicological action and affect the same target tissues. Similarly it would not be expected for substances in a mixture to have interactive health impacts if they were individually present at concentrations significantly below their biological threshold levels (i.e. below their true low observed effect level)[7]. Some investigators therefore prefer only to sum hazard quotients for pollutants that effect common organs, this yields effect-specific cumulative

[7] Because the true LOAEL cannot be readily established empirically, for public health purposes the experimental no observed adverse effect level (NOAEL) is often taken as being the threshold exposure level for eliciting an adverse health effect. Sometimes any meaningful biological effect, whether adverse or not, is taken as the threshold exposure, such an exposure level is called the no observed effect level (NOEL). It should be noted however that the NOEL, the NOAEL and the LOAEL are all influenced by the experimental design of toxicology studies, especially the dose spacing intervals. It should especially be noted that because air guideline values usually have large uncertainty/safety factors incorporated in them, that a HQ less than one signifies the GLC is much less than the biological threshold concentration for causing an effect.

HIs (Fox et al. 2004, Morello-Frosch et al. 2000). Others, while recognising that adding together HQs with different health end points will not give an accurate idea of the non-cancer HI nonetheless add all HQs together (Pratt et al. 2000, and this risk assessment), some investigators limit this latter practice to only those pollutants whose HQ is greater than unity (Tam and Neumann 2004), i.e. for substances whose concentrations may be nearing their biological thresholds.

This preliminary HRA focuses on two air pollutants, particulate matter and NO_2. Chemicals can have more than one toxicological effect often require different levels of exposure to become apparent and as indicated previously it is impractical to determine the all dose effect(s) relationships for all chemicals of concern. Hence it is difficult to identify with confidence all the substances that will have common sites of toxicological action. We have therefore adopted the pragmatic approach, regardless of the mode of toxicological action or site of adverse health effect, of generating overall acute and chronic non-cancer hazard indices for all chemicals of concern as if they were acting in concert. If the resulting composite HI is greater than the HI then the pollutants significantly contributing to the HI are examined in more detail to determine whether or not there is biological plausibility for the additive effects assumed in the calculation of the HI. At this stage the dose effect(s) relationships may be examined.

5.2 Interpretation of hazard quotients and indices

An ‘unacceptable’ risk, as defined by regulatory standards and requirements, is often determined as the exposure being larger than the air guideline value used to calculate the hazard quotient, i.e. the HQ>1. This definition of unacceptable risk does not equate with imminent adverse health effects or even high risk of adverse health effects. It simply means that the health guideline level has been exceeded.

The common practice of summing the HQ of all chemicals in screening (i.e. preliminary) risk assessments, regardless of biological mode of action or target tissue may grossly overestimate the risk estimation for systemic health effects from exposure to the emission mixture of chemicals. It is however a legitimate practice for assessment of end points such as irritation and odour where additive interactions between substances have been demonstrated to occur. Similarly it is not unreasonable to assume additive effects for pollutants that have direct effects on airways function.

Notwithstanding their use in this risk assessment, hazard quotients and hazard indices are relatively blunt tools used to assist in characterising and prioritising risks, great care must be

taken to the level of importance that is placed on the numerical value of the HI. Hazard indices should not be used in isolation of other pertinent data such as mechanistic information on the toxic mode of action and knowledge of the conservatism incorporated into the exposure assessment and toxicity values.

The HI calculation allows focus on components that are likely contributors to health risks either because their individual exposure levels exceed health guidelines, or because joint toxic action with other components, including additivity or interactions, may pose a health hazard. Generally mixture components whose hazard quotients are less than 0.1 (HQ<0.1) are considered unlikely to pose a health hazard due to interactions, and unless there are a relatively large number of components that act similarly, are not likely to pose an increased hazard due to additivity. The general rule of thumb for interpreting a hazard quotient and hazard index is that values less than 1 present no cause for concern; values greater than 1 generally also do not represent cause for concern because of the inherent conservatism embedded in the exposure portions of a preliminary risk assessment. However, it is usual to examine, and perhaps refine, the level of conservatism that has been assumed in the exposure assumptions. Hazard quotients and indices that are around 10 present some concern regarding possible health risks, in these circumstances it is usual to evaluate the extent to which the "safety margins" in the health guideline value used to compare estimated exposures may have been eroded in order to gauge whether concern is warranted. It is common that the risk assessment needs to be refined using site specific exposure information or additional analytical data when HI's are greater than unity.

Incremental data and background data for NO_2 and particulate matter have been obtained from PAE (2012a, 2011). The modelled emissions for the proposed IMT were calculated on a daily ($PM_{2.5}$) or 1-hourly (NO_2) basis and matched with background emissions for these substances at specific times using actual climatic conditions recorded in 2009[8]. For particulate matter data the extreme events coinciding with dust storms (PAE 2010) have been removed as discussed in Section 4. NO_2 modelled data was calculated from NO_X conversions that requires ozone (O_3) data. On days where O_3 data was not collected or returned negative values due to calibration shifts the data was rejected. The background and incremental (modelled) data are date and time matched and percentiles calculated. In many of the following figures information for acute health risks (as hazard indices) is provided for the maximum GLC as well as the 95th, 99th, 99.7th

[8] The modelled data for NO_2 have been converted from ppm to µg/m^3 by PAE (2011) using Standard Temperature and Pressure (STP) (0°C and 101.7kMpa). The Air Guideline Values used here have been done using the same STP conditions, i.e. the 1-hour AGV for NO_2 used was 246µg/m^3 and the annual AGV used was 62µg/m^3. Considering the conservatisms used in this assessment the use of AGV converted using STP conditions is not considered to have any impact the results provided.

and 99.9th percentile estimates of 1 hour average (NO_2) or 24-hour average (PM_{10}, $PM_{2.5}$) predicted GLC based on the data obtained.

As discussed in section 3.3 a good way to consider and interpret percentile GLC information is as the frequency, i.e. number of times per year that the hazard index will be equal to or above the target hazard index. The more frequent the event then the more likely health effects may be experienced at the receptor location for which the modelling was conducted. This is because it becomes more probable an individual may be present at the moment the predicted ground level concentration occurs. Table 3.3 provided a summary of the statistical percentiles used to predict 1-hour and 24-hour average GLC and the corresponding number of hours in a year the concentrations are anticipated to occur.

6.0 Risk characterisation results

Incremental data and background data for NO_2 and particulate matter have been obtained from PAE (2012a, 2011). The modelled emissions for the proposed IMT were calculated on a daily ($PM_{2.5}$) or 1-hourly (NO_2) basis and matched with background emissions for these substances at specific times using actual climatic conditions recorded in 2009. For particulate matter data the extreme events coinciding with dust storms (PAE 2010) have been removed as discussed in Section 3. NO_2 modelled data was calculated from NO_X conversions that requires ozone (O_3) data. On days where O_3 data was not collected or returned negative values due to calibration shifts the data was rejected. The background and incremental (modelled) data are date and time matched and percentiles calculated. In many of the following figures information for acute health risks (as hazard indices) is provided for the maximum GLC as well as the 50^{th} 95^{th}, 99^{th}, 99.7^{th} and 99.9^{th} percentile estimates of 1 hour average (NO_2) or 24-hour average (PM_{10}, $PM_{2.5}$) predicted GLC based on the data obtained.

As discussed in section 3.3 a good way to consider and interpret percentile GLC information is as the frequency, i.e. number of times per year that the hazard index will be equal to or above the target hazard index. The more frequent the event then the more likely health effects may be experienced at the receptor location for which the modelling was conducted. This is because it becomes more probable an individual may be present at the moment the predicted ground level concentration occurs. Table 3.3 provided a summary of the statistical percentiles used to predict 1-hour and 24-hour average GLC and the corresponding number of hours in a year the concentrations are anticipated to occur.

The screening HRA considers impacts on health in two scenarios:

- Scenario 1 **(S1):** The effects of the emissions due to increased freight movements related to the proposed IMT. This is the 'incremental' scenario. It considers emissions from the IMT facility only. Because the project is at a concept phase and detailed information is not yet available to model groundlevel concentrations (GLC) the estimates for the facility were derived (PAEHolmes 2010) in a conservative manner.

- Scenario 2 **(S2):** The combined effects of existing air quality (particulate matter and NO_2) and emissions (particulate matter and NO_2) from the proposed IMT. This is the 'cumulative' scenario. This scenario includes existing air quality data as it is important to consider the relative contribution of the facility to existing air quality.

The results are summarised according to acute (section 6.1) and chronic health risks (section 6.2). These sections include results for both S1 and S2. Appendix 2 contains all the calculations for the results presented in summary form in this section.

Receptors are only potentially exposed to emissions from the IMT when the wind is blowing in a particular direction. For instance Receptor 3 is located 0.5km to the East of the IMT hence when the wind direction is from the East the emissions from the IMT are being blown away from this receptor. However in this instance the emissions from the IMT are being blown towards receptors 5 and 6 that are located to the west of the IMT. Receptors 5 and 6 however are located further from the IMT at 0.8km and 1.5km respectively and GLC concentrations differ with distance to the source. Therefore receptors may not be exposed to emissions from the IMT at the same time and that the GLC are affected by climatic conditions such as wind direction and other influences including distance from the IMT. The actual locations for each receptor are provided in Table 3.4 and shown in Figure 1.1.

6.1 Direct acute health effects

Acute HQ for substances assessed ($PM_{2.5}$, PM_{10} and NO_2) and HI were calculated for Scenario 1 and Scenario 2. The HI was calculated by summing the HQ for $PM_{2.5}$ and NO_2. For assessing health effects from particulate matter the $PM_{2.5}$ fraction was selected ahead of PM_{10} as the representative fraction as $PM_{2.5}$ has been more closely related to hospital admissions and mortality (refer Appendix 1, Table A1.2). The use of $PM_{2.5}$ as the representative fraction is supported by the emissions from the IMT (discussed in section 3.1.2) that are likely to consist of particulate matter mostly in the $PM_{2.5}$ fraction.

6.1.1 Scenario 1

Scenario 1 is the incremental scenario that assesses emissions due to increased freight movements related to the proposed IMT only. For this scenario the target HI is not exceeded at any percentile or the maximum GLC as shown in Figure 6.1 below. The HI comprised of similar contributions from $PM_{2.5}$ and NO_2 with the HI approaching 0.6 for all receptors at the maximum predicted GLC. Based on the climatic conditions in 2009 the proposed emissions from the IMT would not have led to exposures that exceeded the target HI of unity on any day. These results suggest that a health risk is not likely due to emissions from the IMT. It is emphasised that the HI is conservative as the ground level concentrations derived from the facility emissions were based on conservative input assumptions (e.g. modelled based on the busiest hour of operation). It also assumes additivity between particulate matter and NO_2. The conservatism is

warranted given the preliminary information available to model the emissions and the absence of modelled data for all criteria pollutants.

Scenario 1 HQ for NO_2, $PM_{2.5}$ and PM_{10} are shown below in Figure 6.1 and a summary of acute hazard quotients for particulate matter (PM_{10} and $PM_{2.5}$) including percentile data is provided in Table A2.1, Appendix 2.

Evaluation of the health impact of NO_2: The highest maximum one hour GLC at any representative receptors for Scenario 1 was 85µg/m^3 (Receptor 5), well below the one hour air guideline value of 226µg/m^3. Thus for all percentiles evaluated the HQ for NO_2 was well below unity (0.35).

Evaluation of the health impact of PM: The HQ calculated for $PM_{2.5}$ or PM_{10} did not exceed unity at any percentile or the maximum GLC. The NEPM guideline value for PM_{10} (50 µg/m^3) permits five exceedences per year. Given the 95th percentile represents 4 exceedences per year for 24-hour averaging times (see Table 3.4) it is the most appropriate percentile on which to make a comparison for PM_{10}. At this percentile the HQ for PM_{10} ranged from 0.018 (Receptor 4) to 0.11 (Receptor 3). Not surprisingly the HQs for $PM_{2.5}$ are greater than the HQs calculated for PM_{10}. The $PM_{2.5}$ HQ at the 95th percentile (i.e.: a representative comparison) was typically twice as large with a maximum of 0.2 (Receptor 3).

Summary of the health impact of Scenario 1: The HI for scenario 1 (IMT emission only) was well below unity indicating that under the conditions evaluated, the facility is unlikely to cause adverse health effects even for susceptible persons at the closest residential receptors.

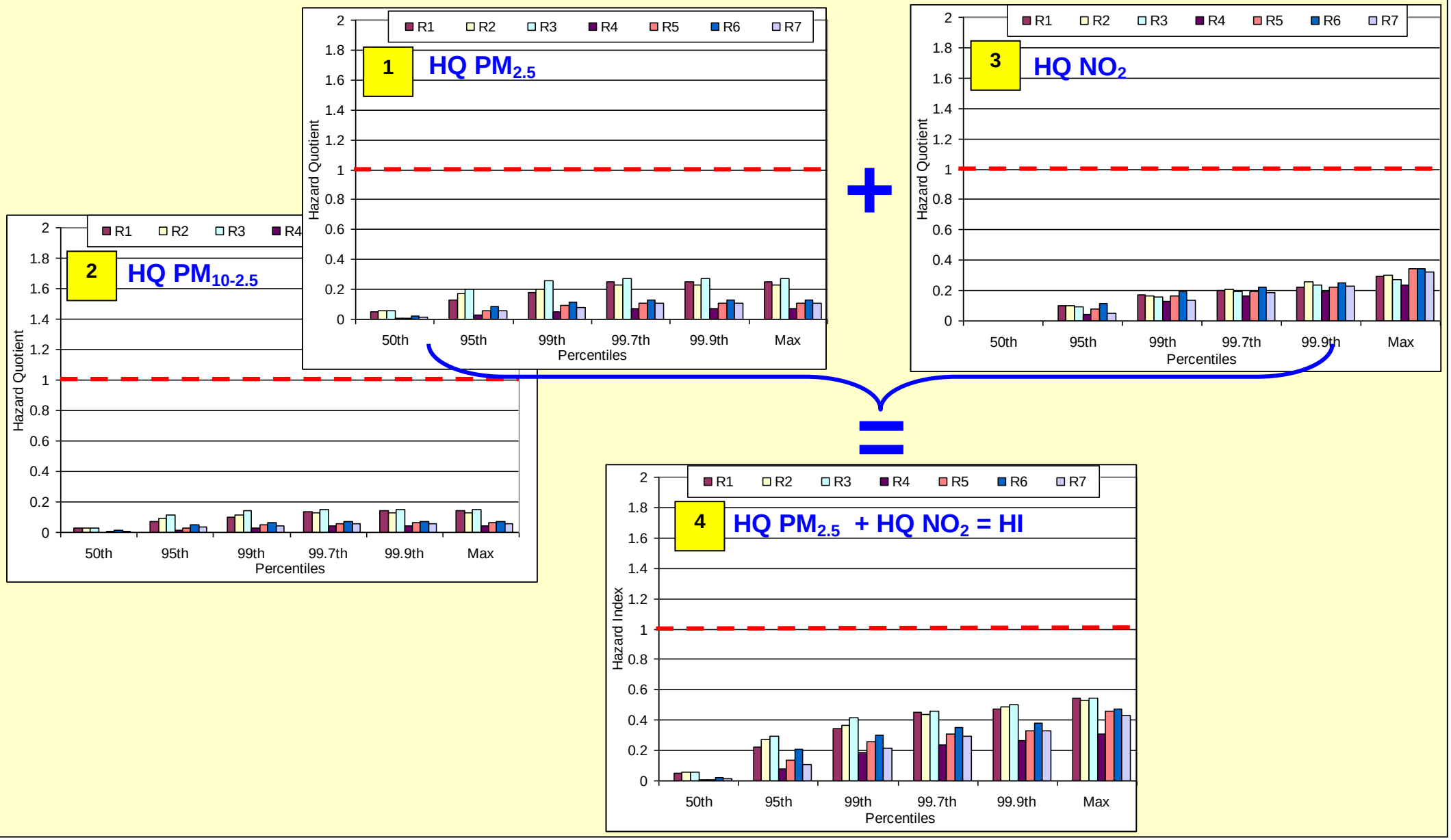
Figure 6.1: Hazard Indices for Scenario 1: Incremental Scenario

1: HQ $PM_{2.5}$, 90% of emissions from the IMT facility are assumed to be fine and ultrafine particles therefore $PM_{2.5}$ is most relevant fraction

2: HQ PM_{10}, The HQ calculated for PM_{10} were equivalent or lower than the HQ calculated for the $PM_{2.5}$ fraction.

3: HQ NO_2, The NO_2 HQ for each percentile and receptor were of similar magnitude.

4: HQ $PM_{2.5}$ + HQ NO_2 = HI, The incremental HI (S1) calculated for emissions from the site at all receptors were below 1.



6.1.2 Scenario 2

Scenario 2 is the cumulative scenario that combines existing air quality data for Liverpool with those from the proposed IMT facility. Figure 6.2 summarises the HQ for $PM_{2.5}$, PM_{10} and NO_2 as well as the HI. A summary of acute hazard quotients for each percentile evaluated is provided in Table A2.5 Appendix 2. The HI for Scenario 2 at the 95th percentile was below 1 (range 0.8 to 0.9) and approximately at or slightly above 1 at 99th percentile (range from 1.0 to 1.1). The HI was exceeded at the 99.7th (range from 1.1 to 1.4) and 99.9th (range from 1.4 to 1.5) percentiles as well as at the maximum GLC (range from 1.5 to 1.7). The majority of the HI was due to existing air quality (up to 93% refer to Figure 6.3) with background particulate matter contributing 60-70% of the total HI depending on the percentile. The contribution from background particulate matter and the significance of these results is discussed below.

6.1.2.1 Contribution of background to S2 results

Figure 6.3 provides the contribution of background emissions to the HI for Receptor 3, the residential receptor located the closest to the proposed IMT and consistently amongst the receptors with the highest HI. Figure 6.3 show that background NO_2 and $PM_{2.5}$ levels contribute the majority of the HI (81-93%) and that contribution from the proposed IMT (incremental NO_2 and $PM_{2.5}$) is low (7-19%).

The contribution of background is particularly important at the higher percentiles (99th, 99.7th, 99.9th) indicating that on rare occasions during the year background air quality in the region exceeds relevant NSW advisory air monitoring standard for $PM_{2.5}$ (i.e. one day in the year evaluated).

Evaluation of the NO_2 contributions to HI: The HQ for NO_2 is below unity and practically all of the hazard quotient (96-100%) is attributable to existing air quality (i.e. background – refer Figure 6.3). There is a consistent reduction in the contribution in NO_2 at higher percentiles hence it is evident that NO_2 from the IMT facility has a minimal impact on cumulative HI.

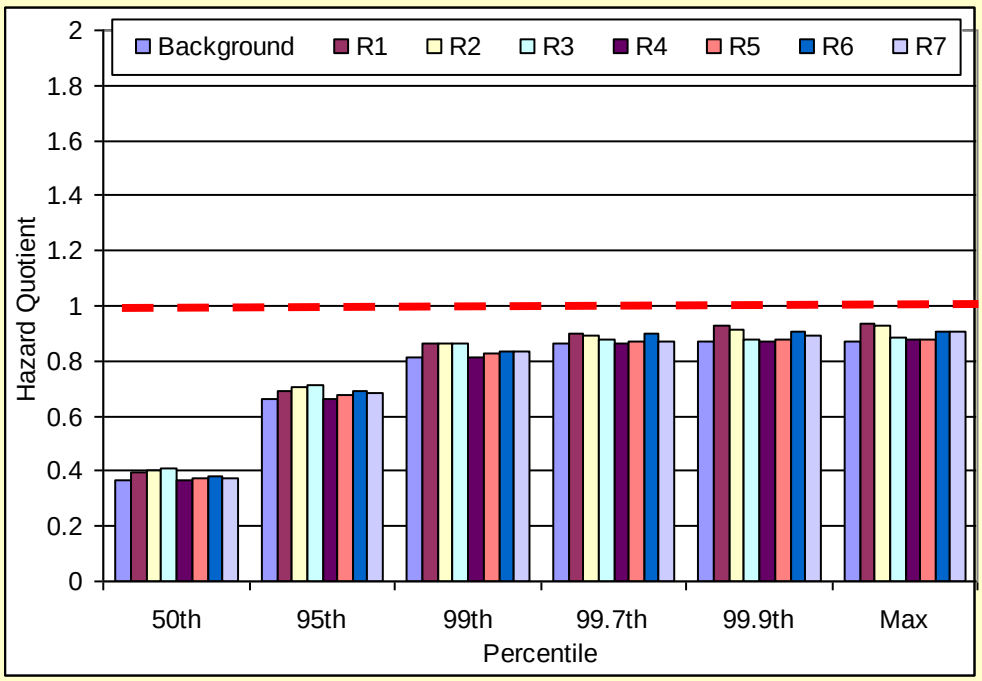
Evaluation of the $PM_{2.5}$ contributions to HI: Background $PM_{2.5}$ accounts for 60-71% of the total acute HI in Scenario 2. The proposed IMT accounts for 18-27% of the total acute HI for Scenario 2. Generally the proportion attributable to the proposed IMT increases slightly at higher percentiles (i.e. it is 21% at the 95th percentile and 24% at 99.9th percentile). This translates to one event (one 24 hour period) per year.

Figure 6.2a: Scenario 2: Acute Hazard Quotients (HQ) for PM_{10} & NO_2

1: HQ PM_{10}, the conservatively predicted 24 hr mean respirable particulate matter (PM_{10}) in combination to background PM_{10} concentration does not exceed the air guideline value for PM_{10} at any receptor locations in the vicinity of the Moorebank IMT.

2: HQ NO_2 the conservatively predicted 1 hr mean nitrogen dioxide in combination with background concentrations does not exceeds the air guideline value at any receptor location in the vicinity of the Moorebank IMT.

1 **HQ PM_{10}**



2 **HQ NO_2**

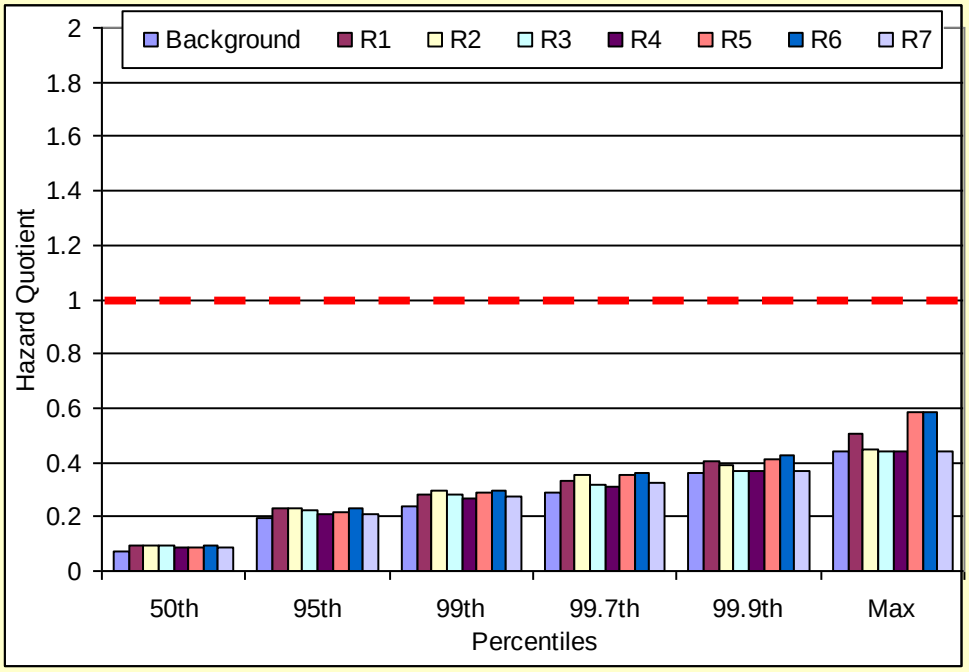
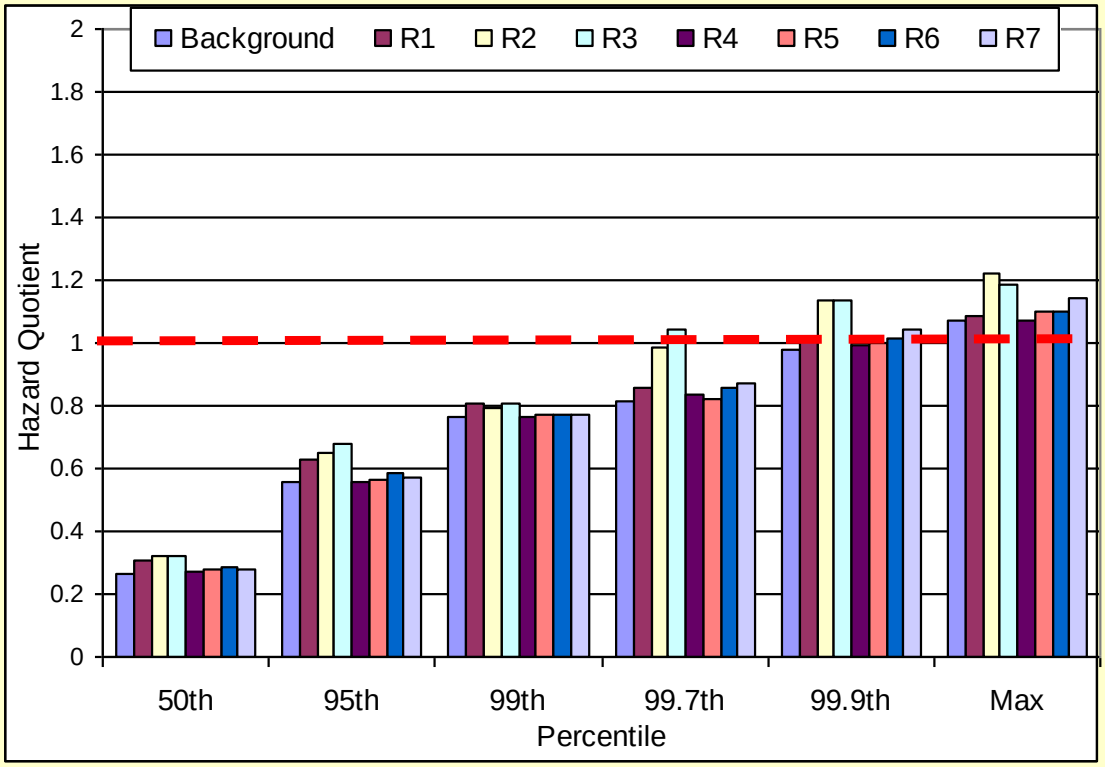


Figure 6.2b: Scenario 2: Acute Hazard Quotient (HQ) for $PM_{2.5}$

3: HQ $PM_{2.5}$, . the conservatively predicted 24 hr mean respirable particulate matter ($PM_{2.5}$) in combination to background $PM_{2.5}$ concentration exceeds the air guideline value on rare occasions. When these exceedences occur they are almost entirely due to high background $PM_{2.5}$ concentrations (see table within Figure 6.2b). Further analysis is provided for Rece

Data Table: The table shows that $PM_{2.5}$ exceedences are rare and are largely attributable to background concentrations (see also Figure 6.3). The exceedance HQ calculated for PM_{10} were equivalent or lower than the HQ calculated for the $PM_{2.5}$ fraction.

3 **HQ $PM_{2.5}$**

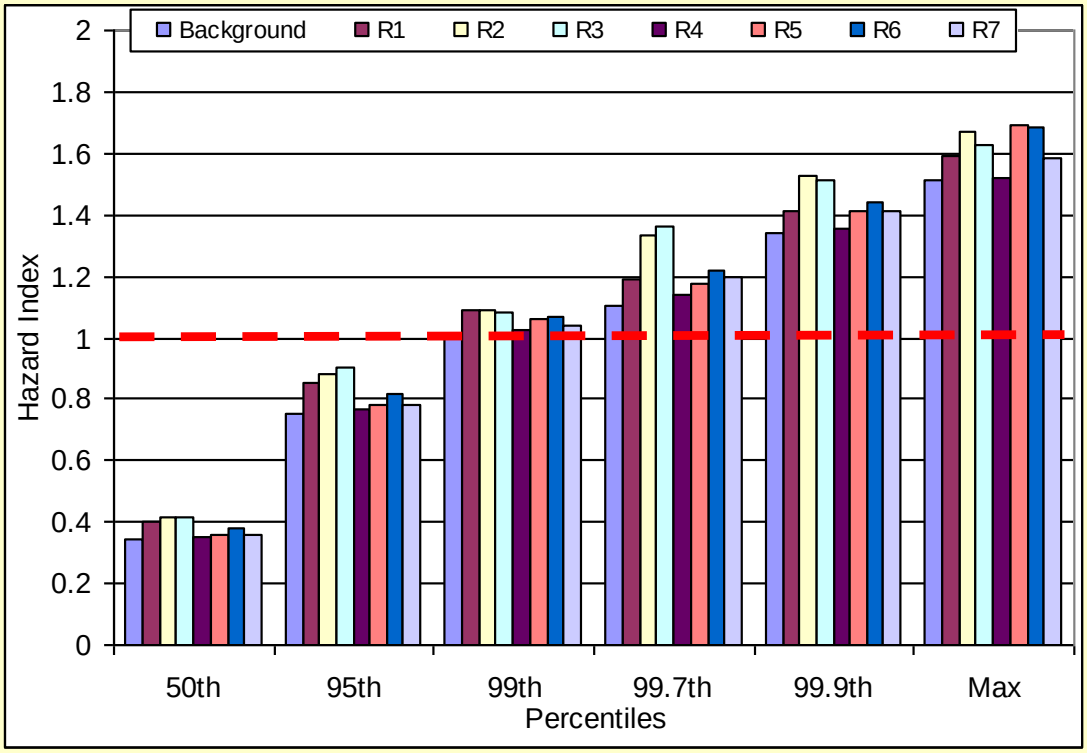


Percentile	Number of exceedances	Background µg/m³	R1 µg/m³	R2 µg/m³	R3 µg/m³	R4 µg/m³	R5 µg/m³	R6 µg/m³	R7 µg/m³
95th	0	17.5	19.2	19.8	20.4	17.8	18.0	18.5	17.9
99th	0	22.3	23.7	23.4	23.7	22.6	22.9	22.9	22.8
99.7th	1 d/yr	23.9	25.0	**28.1**	**29.7**	24.4	24.1	25.1	25.4
99.9th	1 d/yr	**28.2**	**28.9**	**32.2**	**32.2**	**28.5**	**28.8**	**29.1**	**29.9**
Maximum	1 d/yr	**30.5**	**31.0**	**34.3**	**33.4**	**30.7**	**31.3**	**31.2**	**32.3**

Figure 6.2c: Scenario 2: Acute Hazard Index (HI)

4: HQ $PM_{2.5}$ + HQ NO_2 = HI, The hazard index is exceeded on rare occasions (99th percentile and above) during the year (approximately 1 day per year). The relative contributions to the hazard index are described in Figure 6.3 and is mainly attributable to background $PM_{2.5}$. The significance of the exceedence is discussed in section 6.1.4 and Figure 6.4

4 **HQ $PM_{2.5}$ + HQ NO_2 = HI**



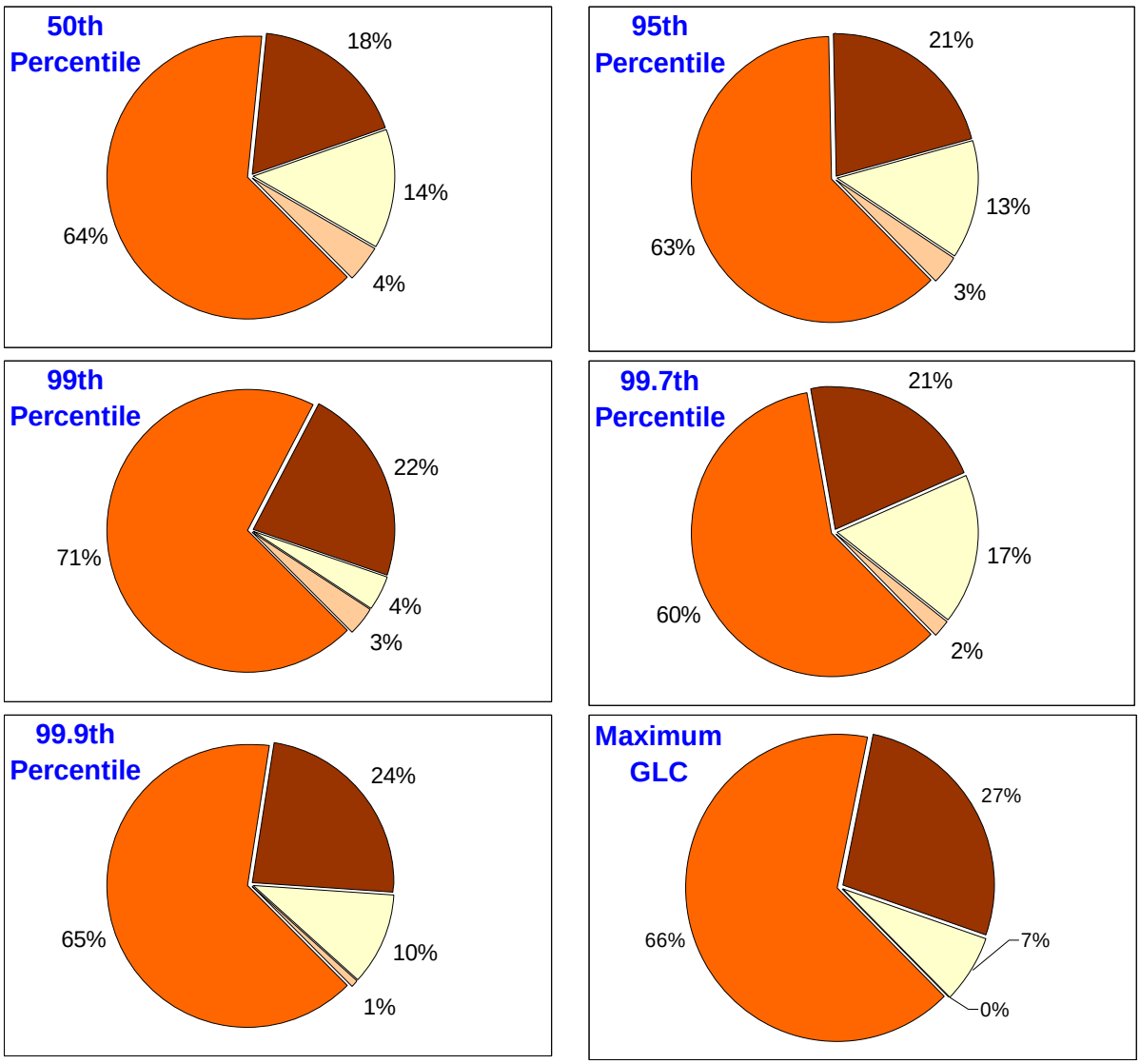


Figure 6.3: Contribution of background to the HI at the 95th Percentile (Scenario 2). The pie charts above show that the relative contribution to the HI from incremental NO_2 and $PM_{2.5}$ (ranging from 7 to 19%) is considerably lower than from background NO_2 and $PM_{2.5}$ (ranging from 81% to 93%)

6.1.2.2 Daily Change in $PM_{2.5}$ concentrations ([$\Delta PM_{2.5}$])

As described in Appendix 1 the health effects of fine particulate matter include respiratory and cardiovascular effects. Association between daily exposure and health effects have been drawn from epidemiology studies. Collectively it is agreed that exposure to a 10 µg/m^3 increase in mean 24 hour $PM_{2.5}$ on the previous day can result in adverse health effects. A recent Australian review (NEPM 2010) concluded that the evidence showed consistent positive associations between short-term exposure to $PM_{2.5}$ and all-cause, cardiovascular- and respiratory-related mortality. The evaluation of large cohort studies taking in data from multiple cities found that risk estimates for all-cause (non-accidental) mortality ranged from 0.29% to 1.21% **per 10µg/m^3 increase** in 24-hour average $PM_{2.5}$ from day to day. These effects were observed in study locations with mean 24-h average $PM_{2.5}$ concentrations 'as low as' 13µg/m^3 (i.e. the dataset in these studies is relevant to Sydney).

In order to consider the significance of the hazard quotient and hazard index for Scenario 2 Figure 6.4 considers the *change* in $PM_{2.5}$ ([$\Delta PM_{2.5}$]) from *one day to the next* for the available dataset (i.e. 2009 calendar year) provided for Receptor 3. This receptor was chosen for this analysis as it represents the closest residential receptors to the IMT and recorded the highest HQ for $PM_{2.5}$ emissions from the proposed IMT. The data set does not include dust storm events (refer section 6.1.3 below). The contribution of background and the IMT facility to the change in $PM_{2.5}$ is included in the figure. Background air levels resulted in one event (on the 12th September 2009) where $PM_{2.5}$ increased by 10µg/m^3 or higher. For receptor 3 on this day the emissions from the IMT would not have contributed to this change in $PM_{2.5}$ concentration. In 2009 there were no additional events where the additional emissions from the IMT facility would have increased the $PM_{2.5}$ concentration to more than 10 µg/m^3.

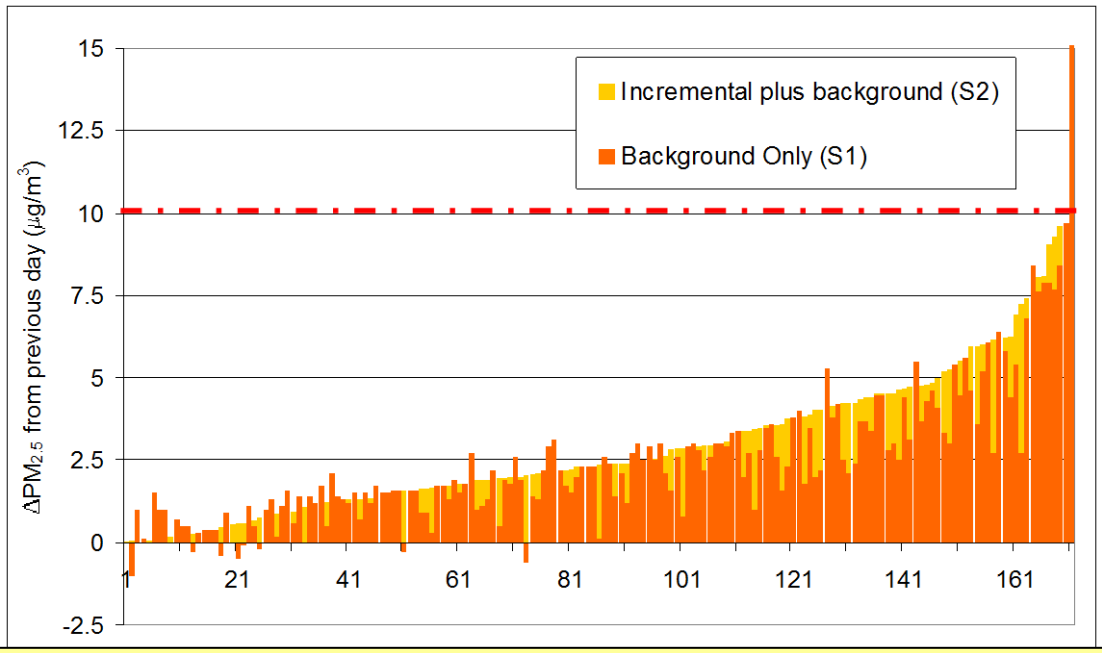


Figure 6.4: The [$\Delta PM_{2.5}$] as a result of additional modelled emissions from the proposed IMT. The change in background concentrations is compared with and without the additional modelled emissions from the proposed IMT for 2009. All but one point is below the measure of 10µg/m^3 indicated by the red dotted line.

6.1.3 Dust Storms

Due to Australia's dry climate it typically experiences 5 to 10 dust storms per year (Cohen et al. 2011). Meteorological conditions may result in these events that permit long range transport of dusts that have the potential to impact major population areas such as the Sydney Basin. Cohen et al. (2011) characterised contribution of various sources to $PM_{2.5}$ in Liverpool area of Sydney[9] and identified that wind blown soils typically make up 3.5% of the $PM_{2.5}$ fraction. However there was a number of soil outliers in the data with the majority of these (28 out of 31) related to dusts "originating from areas outside the Sydney region". The source of the dust was due to windblown soils from agricultural regions with the Riverina district the major contributor (33%) and desert dusts from Central Australia (Cohen et al. 2011).

During 2009 a number of these events were observed in the Sydney region and impacted on the particulate matter levels. The largest of these events was a dust storm on the 23rd of September 2009 that resulted in elevated PM_{10} concentration that exceeded 1,500µg/m^3. Dust storms were also evident in much of the last week of November with impacts on particulate matter concentrations due to isolated dust storms in the region seen on the 22nd, 27th, 28th and 29th of November. Other days during 2009 affected by dust storms include the 05th of March, the 15th and 16th of April and the 26th of September.

Dust from the September 2009 storms was attributed to the Lake Eyre basin as well as surrounding agricultural regions (Radhi et al 2009). Dust distribution was evident in the coarse mode with particle sizes distributed around 10µm. Characterisation identified the source of the dust to be primarily crustal (Radhi et al 2010). As the source of the dust is crustal which typically is a small component of $PM_{2.5}$ in the Liverpool area it is not expected that the adverse health effects associated with particulate matter that guideline values are based during days not affected by dust events are associated with crustal dust. Since these dust storms in September 2009 a monitoring network for dusts in the region has been reporting sightings of dust events (Dustwatch 2011). Advance warning of high particulate matter and air quality in Sydney metropolitan area can be obtained from the NSW OEH (2011). Dust storms are considered an extreme event and particulate matter data for these days have not been included in calculations performed in this assessment.

[9] Cohen et al. (2011) performed a long range study from the 1st of January 2001 to the 31st of December 2009 that characterised and source apportioned $PM_{2.5}$ in the Liverpool region of Sydney.

6.1.4 Conclusions for likelihood of acute health effects:

Overall the risk characterisation for the IMT facility show that it is unlikely to be detrimental to the health of residents in the vicinity over an acute duration of exposure. This conclusion is based on:

- The hazard quotients for particulate matter and nitrogen dioxide are below unity indicating that the emissions from the facility itself are unlikely to pose a health risk to nearby residents

- The HI for scenario 1 (IMT emission only) was well below unity indicating that under the conditions evaluated, the facility is unlikely to cause adverse health effects even for susceptible persons at the closest residential receptors.

- When existing air quality data was considered in combination with the air quality predictions for the proposed IMT facility (Scenario 2) the hazard quotient for PM_{10} and nitrogen dioxide is always less than 1. For $PM_{2.5}$ the highest hazard quotient was 1.2. The hazard quotients calculated for the maximum GLC and at the 99.7th and 99.9th percentile were 1.2, 1.1 and 1.0 respectively. This indicates that there is one day in the year (excluding extreme events) that the existing air quality exceeds the $PM_{2.5}$ advisory standard for monitoring purposes.

- The HI for Scenario 2 at the 95th percentile was below 1 (range 0.8 to 0.9) and approximately 1 at 99th percentile (range from 1.0 to 1.1). The HI was exceeded at the 99.7th (range from 1.1 to 1.4) and 99.9th (range from 1.4 to 1.5) percentiles as well as at the maximum GLC (range from 1.5 to 1.7). The majority of the HI was due to existing air quality (up to 93% refer to Figure 6.3) with background particulate matter contributing 60-70% of the total HI depending on the percentile. Although the hazard index is slightly greater than 1 adverse health effects are considered unlikely because the hazard index is predominantly based on existing air quality data..

- Adverse health impacts due to $PM_{2.5}$ are primarily related to sharp short term increases in daily average $PM_{2.5}$ concentrations. The proposed IMT does not contribute to such a change in daily average $PM_{2.5}$ concentrations.

6.2 Direct chronic health risks

The World Health Organisation (WHO 2006) recommends an air guideline value of 20µg/m^3 for PM_{10}, 10µg/m^3 for $PM_{2.5}$ and 56µg/m^3 for NO_2 as annual values or goals. An average of the air dispersion modelling results supplied by PAE (2012) shows that the annual average for these substances in 2009 anywhere in the air modelling domain to be less than the guideline value. A description of the basis of the air guideline value and the health effects of particular matter is provided in Appendix 1.

Scenario 1: Figure 6.5 summarises the chronic hazard quotients and indices for Scenario 1 for the selected receptors around the proposed IMT. For Scenario 1 the majority of the HQs calculated are less than 0.1 and well below unity indicating little likelihood of direct chronic adverse health effects due to emissions modelled from the proposed IMT. The highest HI of 0.26 was calculated for Receptor 3 well below the target HI of 1 therefore this result suggests that there is a low likelihood that susceptible persons at these residential receptors would exhibit adverse health effects arising from chronic exposure to IMT emissions for Scenario 1.

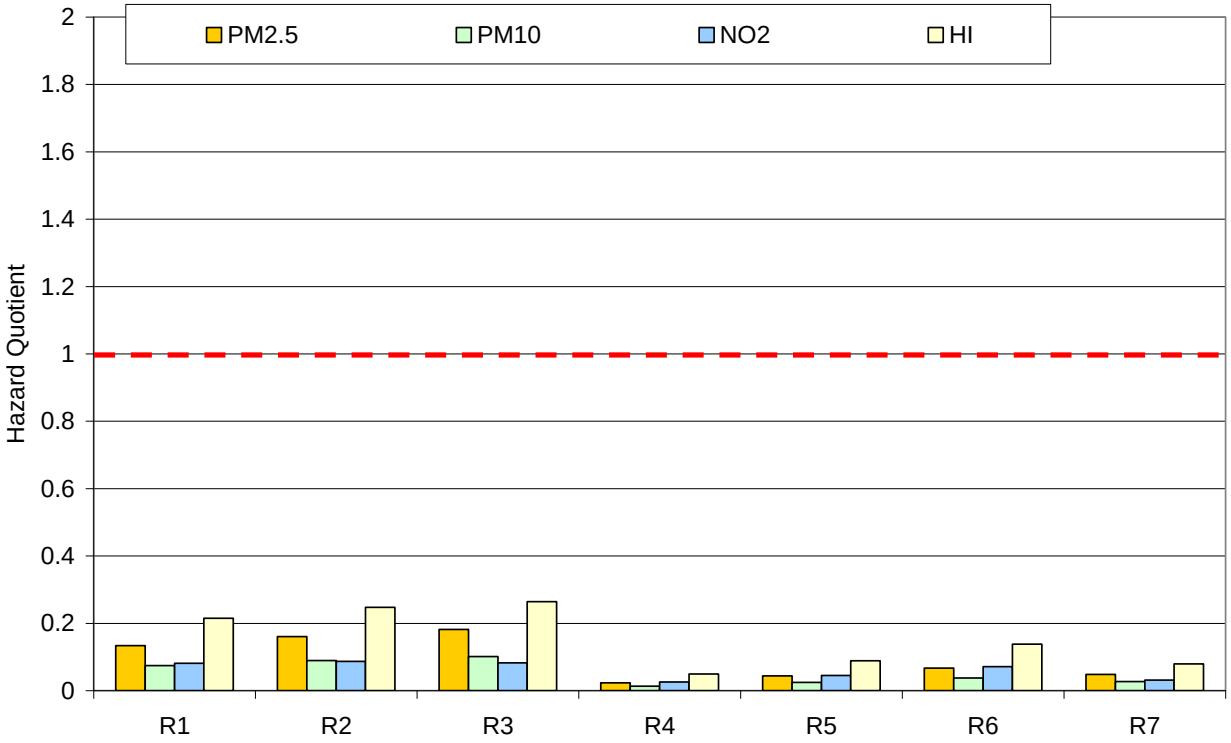


Figure 6.5: The Chronic Hazard calculated for Scenario 1. The HQ and HI calculated for scenario 1, the modelled emissions from the proposed IMT, are well below the target HI of 1 and would pose little risk.

Scenario 2: Figure 6.6 summarises the chronic hazard quotients and indices for Scenario 2 for the selected receptors around the proposed IMT. For Scenario 2 the majority of the HQs calculated are less than 1. Although the HQ's have been added to calculate a HI this is a very conservative approach. The health based guidelines for these emission components are based on epidemiological studies that measure the association between at least one specific pollutant (i.e. PM, NO_x, CO or O_3) and health outcomes. These specific components are usually highly correlated with other pollutants and are considered indicative of the complex pollutant mixture. It is unclear how much the associations reported in epidemiological studies represent the independent effects of specific pollutants. This correlation means that simply summing the pollutant-specific impacts can lead to an overestimation of the overall impact of air pollution on health. Given the HQs for each emission component is less than 1 it is unlikely that direct chronic adverse health effects due to emissions modelled from the proposed IMT in combination with background would be observed even in susceptible population. The highest HI of 1.3 was calculated for Receptor 3 above the target HI of 1 however rounding of the HI would result in a value of 1 that is at the target index. This result suggests that there is a low likelihood that susceptible persons at these residential receptors would exhibit adverse health effects arising from chronic exposure to IMT emissions for Scenario 2.

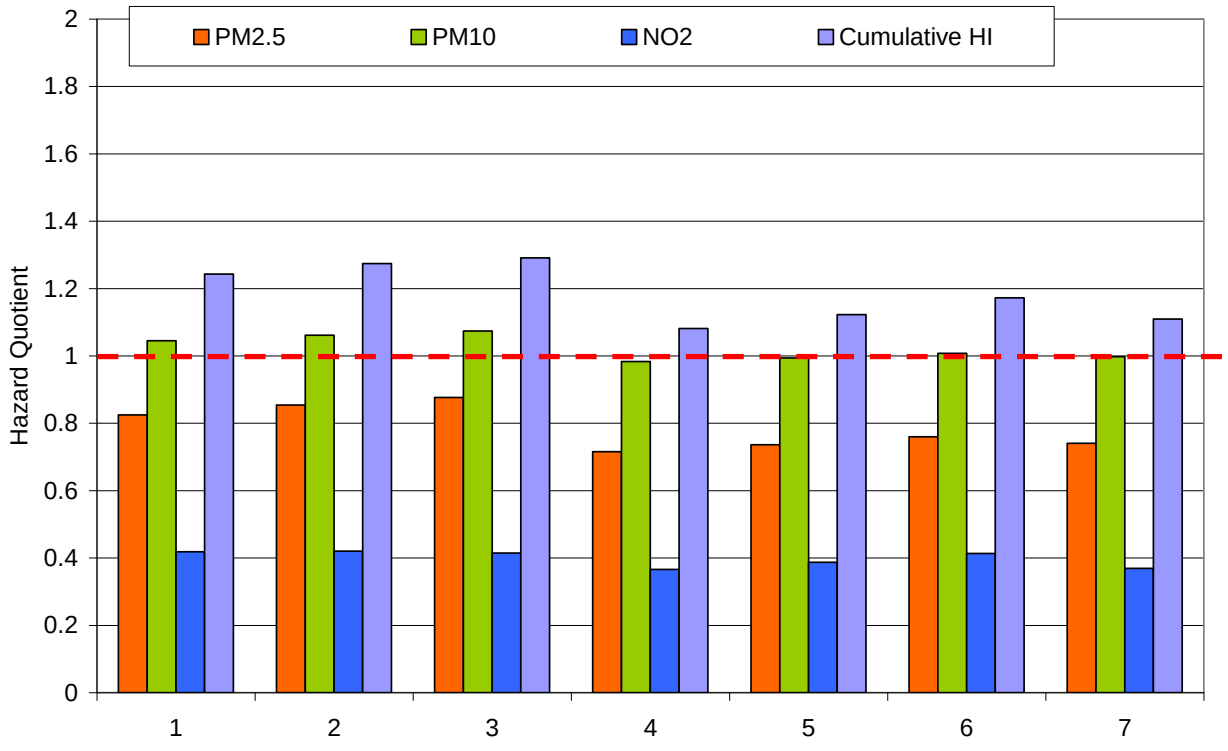


Figure 6.6: The Chronic Hazard calculated for Scenario 2. The HQ calculated for scenario 1, the modelled emissions from the proposed IMT plus background concentrations, are at or below the target HI of 1. The HI rounds down to 1 hence there is a low likelihood of risk.

6.3 Conclusions for systemic health effects

Health impact of emissions from SIMTA Intermodal transport facility
The assessment undertaken for emissions from the IMT facility indicate that acute or chronic direct health effects are unlikely. The emissions of major importance for possible health effects are fine particulate matter ($PM_{2.5}$). Nitrogen dioxide does not contribute to the overall acute or chronic health risk estimated for the IMT facility. PM_{10} , $PM_{2.5}$ or NO_2 released from the IMT facility have negligible impact on the surrounding area, either on their own or in combination.

Cumulative health impacts of emissions from IMT in combination with existing air quality data:
Detailed operation plans have yet to be developed for the SIMTA IMT facility necessitating the emissions to be estimated using highly conservative assumptions. Existing air quality data for the Moorebank air shed are available from a nearby air monitoring station in Liverpool. Information on the IMT facility emissions has been combined with the existing data from the Liverpool air monitoring station. Not all substances in the emissions from IMT facility have been assessed (in particular criteria pollutants such as ozone and organic compounds such as PAH) nor has the veracity of the emissions estimates been assessed. Based on the available data and the substances that have been assessed it can be concluded there is low likelihood for cumulative acute or chronic health effects.

Individual concentrations of NO_2 and PM_{10} and for the most part $PM_{2.5}$ are individually below their respective health guidelines. However on rare occasions (one occasion in the period assessed excluding dust storms or other extreme weather events in Sydney) the accumulation of particulate matter and nitrogen dioxide (mainly related to PM) can exceed the combined standards. This does not mean health effects are probable or imminent in the vicinity of the IMT facility. In reaching this conclusion it is noted the air dispersion modelling may have over-estimated ground level concentrations. Also the conclusion does not consider reductions in the background mass of PM, and NO_2 due to the replacement of road freight transport by rail.

Regional health impacts of emissions from IMT:
Intermodal transport facilities in metropolitan Sydney are expected to reduce long term environmental impacts from land based container transport activities given the increased proportion of containers transported by rail (SKM 2005, Walls 2008). The SIMTA IMT will contribute to this target by reducing the levels of fuel consumption due to a reduction in the growth of container truck movements (PAEHolmes 2010). Although this will accompany and increase in locomotive emissions overall the type and quantity of emissions from fuel consumption are expected to be reduced and hence regional air quality would improve. An overall reduction in diesel related particulate emissions will likely reduce background fine particulate matter in southwestern Sydney and lead to improved health outcomes.

7. Uncertainty analysis

In interpreting the calculated risks associated with assumed exposure to emissions from the refinery, uncertainties associated with the assessment need to be considered. The risk assessment process involves a number of steps (e.g. exposure assessment, toxicity assessment and risk characterisation), each of which incorporates the use of assumptions and simplifications to manage uncertainty or lack of knowledge about the correct value. Without such assumptions and simplifications it would not be possible to quantitatively evaluate the potential for health effects. Although uncertainties in the risk assessment may influence its accuracy, reliability and interpretation, the assumptions used to cope with the uncertainties err on the side of safety and therefore bias the evaluation to over estimation of health risk. This is appropriate for an assessment for possible impacts on public health. It must be realised however the conservatism regarding one value is at least additive, most times multiplicative, with other conservatisms such that the cumulative or compound conservatism incorporated into the assessment can be very large. This is especially so when gross, unrealistic default parameters are used in lieu of measured data.

The approach to characterising uncertainty and variability in this HRA is based on application of the WHO Guidance on Characterizing and Communicating Uncertainty In Exposure Assessment (WHO 2008). This guidance provides a four-tiered approach for characterizing uncertainty (and to a lesser extent variability) in the context of a risk assessment, with tiers ranging from qualitative characterization (Tier 1) to use of full-probabilistic Monte Carlo-based simulation (Tier 3). These tiers are described as:

- **Tier 0** – recommended for routine screening assessments, uses default uncertainty factors (rather than developing site-specific uncertainty characterizations);
- **Tier 1** – the lowest level of site-specific uncertainty characterization, involves qualitative characterization of sources of uncertainty (e.g., a qualitative assessment of the general magnitude and direction of the effect on risk results);
- **Tier 2** – site-specific deterministic quantitative analysis involving sensitivity analysis, interval-based assessment, and possibly probability bound (high- and low-end) assessment; and
- **Tier 3** – uses probabilistic methods to characterize the effects on risk estimates of sources of uncertainty, individually and combined. With this four-tiered approach, the WHO framework provides a means for systematically linking the characterization of uncertainty to the sophistication of the underlying risk assessment.

This section contains a general qualitative discussion of the major uncertainties and their potential influence on the screening health risk assessment (i.e. a Tier 1 assessment). The ‘big picture’ uncertainties fall into the following major categories.

- Those associated with exposure estimation.

- Receptor specific uncertainties.
- Contaminant specific uncertainties.

Questions commonly asked are:

- Have all emission components been identified?
- Are GLCs accurately predicted?
- What is the variability in emission levels?
- Have background exposures been taken into account?
- What is the effect on susceptible subpopulations?
- Are there emission components of unknown effects?
- Is there interaction between emission components for causing health effects?

For the IMT facility the above questions are addressed in Table 7.1, it presents a listing of the major areas of uncertainty for the IMT emissions only.

Elsewhere in the report, when particular risks, or health endpoints are discussed/assessed, additional specific information on the uncertainty is provided to enable the reader to integrate the uncertainties with the assessment that has been performed at that point in the report.

Table 7.1: Uncertainties in the risk assessment for IMT emissions and potential effect on screening health risk assessment outcome.

Uncertainty/Assumption	Comment	Effect on Risk Assessment
Exposure Estimation ***Identification & quantification of emissions.*** Some emission components may not have been identified or appropriately quantitated.	The Air quality assessment focussed on particulate matter and nitrogen dioxide. Carbon monoxide, sulphur dioxide, ozone and organic hydrocarbons were not modelled. Thus it was not possible to consider cumulative impacts due to these emission components.	The extent of emissions may have been underestimated. However the likelihood is considered to be low since 'missing' emission components will be minor constituents and predicted GLCs will be below relevant health based guidelines (PAEHolmes 2010).
There is uncertainty in sampling and analytical determination of emission components.	The emission rates used in the modelling are considered worst case as these were modelled at the busiest hour of operation.	It is likely that chronic hazard quotients and hazard index is overestimated. Emissions based on conservative worst case estimates are likely to overstate the annual average exposure.
	Sampling and analytical error bounds for organics are ~ ± 30% for dry to moderately wet sources, increasing to ± 50% for wet sources (>80% moisture). Most of the uncertainty (and variability) is in quantification of emission flows.	It is unlikely a compound was not assessed because the MDL was too high.
	In the absence of operational data for the Moorebank IMT assumptions on emissions from all activities were based on a similar facility at Enfield NSW.	This is likely to create bias towards overestimating emissions due to the conservative approach necessary to account for the lack of detailed information.
	Criteria pollutants in the emissions of IMT facilities are well known.	There is low confidence in the inventory for criteria pollutants. These are the substance likely to contribute the majority of health risks associated with the IMT facility. Air emission modelling was not conducted for some criteria pollutant

Uncertainty/Assumption	Comment	Effect on Risk Assessment
Not all sources of emissions from the IMT facility have characterised.	Some fugitive emission sources have not been considered.	Emissions of particulates include fugitive emissions thus these may be underestimated. Although it is considered unlikely marked underestimation of emissions has occurred we believe the conservatism built into the dispersion modelling and risk assessment will cater for possible underestimation.
Prediction of GLCs. There is uncertainty in the air dispersion modelling in its predictions of ground level concentrations of emission components at the receptor locations of interest. *Background Exposures* Inclusion of background data	Modelling techniques contain inherent uncertainty. It is assumed the model is predictive for dispersion of emission components. However input emission parameters to the model are apparently conservative because it has been assumed: -all emission points will be emitting together at the same time for 100% of the time. -during dispersion there is no loss of contaminant through gravimetric deposition, washout or atmospheric chemical reactions. Background data for particulate matter and nitrogen dioxide have been considered.	The estimates are conservative and are intended to be so to account for the operational data for Moorebank. For other emission components it appears the GLC may be over estimated and hence the risk over estimated. The extent is unknown. Background is considered in a cumulative emission scenario (scenario 2). The IMT facility on its own does not exceed acute & chronic risk estimates and the conclusions is minimal.
Receptor Uncertainty There may be people within the emission dispersion zone that are more susceptible than most to developing health effects if they are exposed to refinery emissions.	Public health air guidelines are established to account for the variability in human response and therefore largely compensate for lack of receptor characterisation in most HRAs. It is not usual to characterise the exposed population with respect to susceptibility in a HRA of this nature. Nevertheless the demographics and overall health status (for illnesses of interest to the SHRA) of the population around	Impact on the conclusions of the SHRA is minimal. However it is recognised there may be a very small, unlikely, possibility of an adverse health reaction if unusually sensitive individuals are exposed. This is no different than any other public health assessment using regulatory guideline values. As far as possible the possibility of highly sensitive responders has been catered for by inclusion of

Uncertainty/Assumption	Comment	Effect on Risk Assessment
	Collie is similar to other rural communities south of Perth. Hence the population potentially impacted by refinery emissions is not intrinsically more sensitive to air pollutants.	reasonable overestimation of exposures.
Contaminant Uncertainty *Defining toxicological potency of emission components.* Dose response relationships are not fully determined for all emission components.	The HRA relies on regulatory guidelines established to protect public health.	It is possible health guideline values used to characterise risk may not be protective of sensitive sub-groups in the exposed population. However given the large margins of safety between the NOEL and guideline for the majority of emission components (i.e. the use of safety factors in establishing guidelines) it is unlikely the guidelines used will fail to be protective of all or nearly all individuals. This is the very essence of the philosophy for creating public health guidelines.

References

Cohen, D. D., E. Stelcer, D. Garton and J. Crawford (2011). "Fine particle characterisation, source apportionment and long-range dust transport into the Sydney Basin: a long term study between 1998 and 2009." Atmospheric Pollution Research 2(2): 182-189.

CSIRO (2000). Chemical and Physical Properties of Australian Fine Particles: A Pilot Study. Final report. Division of Atmospheric research, CSIRO and the Australian Nuclear Science and Technology Organisation.

Dustwatch (2011). Dustwatch Australia. Available at: http://dustwatch.edu.au/

enHealth (2001). Health Impact Guidelines. Canberra: National Public Health Partnership, Commonwealth Department of Health and Aged Care.

enHealth (2011). Australian Exposure Factor Guidance. REVIEW DRAFT enHealth Council. http://www.health.gov.au/internet/main/publishing.nsf/Content/enhealth-public-comments.

Harris P., Harris-Roxas B., Harris E., Kemp, L. (2007). Health Impact Assessment: A practical guide. Sydney: UNSW Research Centre for Primary Health Care and Equity and NSW Health.

Jaenicke, R., 1993. Tropospheric aerosols. In: Hobbs, P.V. (Ed.), Aerosol-Cloud-Climate Change Interactions. Academic Press, San Diego, USA, pp. 1–31. As quoted in Morawska et. al (2008)

Jayaratne, E. R., Z. D. Ristovski, N. Meyer and L. Morawska (2009). "Particle and gaseous emissions from compressed natural gas and ultralow sulphur diesel-fuelled buses at four steady engine loads." Science of The Total Environment 407(8): 2845-2852.

Kunzli N, Kaiser R, Medina S, Studnicka M, Oberfeld G, Horak F (1999), Health Costs due to Road Traffic-related Air Pollution—An assessment project of Austria, France and Switzerland, Prepared for the Third Ministerial Conference for Environment and Health, London cited in NSW DEC (2005).

Mahoney, M., Simpson, S., Harris E., Aldrich, R., Stewart Williams, J. (2004). Equity focused health impact assessment framework. Newcastle: Australasian Collaboration for Health Equity Impact Assessment 2004.

Morawska, L., W. and Zhang, J. J. (2002). “Combustion sources of Particles. 1. Health relevance and source signatures.” Chemosphere 29 (2002) 1045-1058.

Morawska, L., W. Hofmann, J. Hitchins-Loveday, C. Swanson and K. Mengersen (2005). "Experimental study of the deposition of combustion aerosols in the human respiratory tract." Journal of Aerosol Science 36(8): 939-957.

Morawska, L., D. U. Keogh, S. B. Thomas and K. Mengersen (2008a). "Modality in ambient particle size distributions and its potential as a basis for developing air quality regulation." Atmospheric Environment 42(7): 1617-1628.

Morawska, L., Z. Ristovski, E. R. Jayaratne, D. U. Keogh and X. Ling (2008b). "Ambient nano and ultrafine particles from motor vehicle emissions: Characteristics, ambient processing and implications on human exposure." Atmospheric Environment 42(35): 8113-8138.

Morello-Frosch, R.A., Woodruff, T.J., Axerad, D.A. and Caldwell, J.C. (2000). Air Toxics and Health Risks in California: The Public Implications of Outdoor Concentrations. Risk Analysis. 20: 273-291.

NEPC (2010). Review of the National Environment Protection Measure (NEPM). National Environment Protection Council (NEPC).

NSW DEC (2005). “Approved Methods for the Modelling and Assessment of Air Pollutants in NSW”, August 2005.

NSW DEC (2005a). Air Pollution Economics Health Costs of Air Pollution in the Greater Sydney Metropolitan Region. NSW Department of Environment and Conservation.

NSW DECCW (2009) Action for Air. Department of Environment, Climate Change and Water NSW

NSW DECCW (2010) Current air quality in New South Wales A technical paper supporting the Clean Air Forum 2010. State of New South Wales and Department of Environment, Climate Change and Water NSW.

NSW Health (2009). Healthy Urban Development Checklist. A guide for health services when commenting on development policies, plans and proposals. NSW Department of Health.

NSW Health (2010). Population Health Division. The health of the people of New South Wales - Report of the Chief Health Officer. Sydney: NSW Department of Health. Available at: www.health.nsw.gov.au/publichealth/chorep/. Accessed June 2011.

NSW OEH (2011). Sydney AQI Forecast. Office of Environment and Heritage, New South Wales Government. Available at: http://www.environment.nsw.gov.au/aqms/aqiforecast.htm

PAE (2010). Air Quality Impact Assessment. Concept Phase Approval for Moorebank Intermodal Freight terminal. 17 August 2010. Job No. 5114. PAEHolmes.

PAE (2012a). Email correspondence between Ronan Kellaghan and John Frangos title “Emailing: Daily PM Background + Increment.xls” with attached excel spreadsheet “Daily PM Background + Increment.xls”.

PAE (2011). Email correspondence between Ronan Kellaghan and John Frangos title “Emailing: Ausplume_list_discrete_receptorsr.xls” with attached excel spreadsheet “Ausplume_list_discrete_receptorsr.xls”.

Pratt, G.C., Palmer, K., Wu, C.Y. et al. (2000). An Assessment of Air Toxics in Minnesota. Environ. Health Perspect. 108: 815-825.

Radhi, M., M. A. Box, G. P. Box and D. D. Cohen (2009). "Size-resolved chemical composition of the September 2009 Sydney Dust Storm." Air Quality and Climate Change 44(3): 25-30.

Ristovski, Z., Jayaratne, E.R., Lim, M., Ayoko, G.A., Morawska, L., 2006. “Influence of diesel fuel sulphur on the nanoparticle emissions from city buses”. Environmental Science and Technology 40, 1314–1320. As quoted in Morowska 2008b.

Ristovski, Z.D., Jayaratne, E.R., Morawska, L., Ayoko, G.A., Lim, M., (2005). “Particle and carbon dioxide emissions from passenger vehicles operating on unleaded petrol and LPG fuel.” Science of the Total Environment 345 (1–3), 93–98. As quoted in Morowska 2008b.

RIVM (2002). On health risks of ambient PM in the Netherlands. Netherlands Aerosol Programme, National Institute for Public Health and the Environment, Netherlands.

SKM (2005). Intermodal Logistics Centre at Enfield Environmental Assessment

Tam, B.N. and Neumann, C.M. (2004). A Human Health Assessment of Hazardous Air Pollutants in Portland, OR. J. Environ. Management 73: 131-145.

Urbis (2010) Moorebank Intermodal Terminal Community Issues Overview. Urbis Pty Ltd November 2010

US EPA (1989). Risk assessment guidance for Superfund. Volume I: Human health evaluation manual (Part A). Washington, DC: U.S. Environmental Protection Agency, Office of Emergency and Remedial Response. EPA/540/1-89/001.

US EPA (1997). Exposure Factors Handbook. Volume 1: General Factors. EPA/600/8-89/043, May 1989. Update to Exposure Factors Handbook. EPA/600/P-95/002Fa, August 1997. Office of Research and Development, National Center for Environmental Assessment, U.S. Environmental Protection Agency.

US EPA (2000a). Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures. Risk Assessment Forum, U.S. Environmental Protection Agency. EPA/630/R-00/002.

US EPA (2010). Integrated Science Assessment for Particulate Matter. United States Environmental Protection Agency. EPA/600/R-08/139F.
http://www.epa.gov/ncea/pdfs/partmatt/Dec2009/PM_ISA_full.pdf.

WA Health (2007). Health Impact Assessment Discussion Paper. In: Western Australian Department of Health, editor. Perth.

Walls (2008). Planning for Sydney's Intermodal Terminals. University of NSW, Thesis abstract.

WHO (2006). Air Quality Guidelines Update 2005. Particulate Matter, Ozone, Nitrogen Dioxide and Sulfur Dioxide. World Health Organisation Regional Office for Europe, Copenhagen.
http://www.euro.who.int/__data/assets/pdf_file/0005/78638/E90038.pdf.

Appendix 1: Health effects summaries

A1.1 Nitrogen dioxide

In recent years the health effects of ambient air exposure to nitrogen dioxide (NO_2) have been well studied and reviewed by national and international agencies (WHO 1997, 2000a, 2000b, 2006; NEPM, 1998, 2010; OEHHA, 1999). The critical health outcomes include respiratory disease and associated symptoms, and changes in lung function. Individuals with asthma and other chronic lung disease and cardiovascular diseases are recognised as being particularly vulnerable. Other susceptible populations include infants, children and the elderly (>65 years of age) (NEPM 2010).

Only very high concentrations of NO_2 (approximately 2,000 µg/m^3 (~1,050 ppb)) affect breathing in healthy people[10]. However small changes in lung function (< 5%) and changes in airway responsiveness have been reported in several studies of sensitive asthmatics or the elderly exposed to concentrations as low as 375-575 µg/m^3 (~200-300 ppb) over 20 minutes to 4 hours (Bauer et al., 1986; Bylin et al., 1988; Roger et al., 1985a & b; Morrow et al., 1992; Strand et al., 1996, 1997, Streeton 1997). These levels represent a clear low-observed-effect level (LOEL) for NO_2 based on increased responsiveness in mild asthmatics to bronchoconstrictors or in subjects with chronic obstructive pulmonary disease (COPD). The study by Bauer et al. (1986) did not find a significant change in pulmonary function when asthmatics were exposed to 560 µg/m^3 NO_2 when resting, with decreases recorded only after the subjects exercised. Similarly, testing asthmatics the day after exposure to 490 µg/m^3 NO_2 did not decrease lung function before allergen challenge (Strand et al., 1997).

The identification of an obvious no effect level is less clear but it seems to be around 200 µg/m^3 (approx 0.1 ppm). Studies have shown that effects can be detected in mild asthmatics following short-term exposure to 488-500 µg/m^3 (260-240 ppb) NO_2 and subsequent exposure to an inhalation challenge (Strand et al., 1996, 1997; Kraft et al., 2005). However, in a study where mild asthmatic subjects were exposed for 1 hour to 200 µg/m^3 (~0.1 ppm) NO_2 and then immediately exposed to a house dust mite challenge, the late asthmatic response (as tested using forced expiratory volume in one second; FEV_1) was found to be greater than when compared to air (NO_2 -7.76% vs. Air -2.85%), but the results were not found to be significant (Tunnicliffe et al., 1994).

According to Streeton (1997) there is an increasing body of evidence to suggest that longer term (years) ambient exposure to significantly lower concentrations of NO_2, of the order of 40 - 80 ppb (approx 75-150

[10] Conversions are performed at STP (0°C and 101.7kPa) consistent with data provided by PAE (2011). The conversion for NO_2 is: ppb = µg/m^3 x 0.49; µg/m^3 = ppb x 2.05. Many unit conversions in this section have been rounded.

µg/m^3) during early and middle childhood years can lead to the development of recurrent upper and lower respiratory tract symptoms, such as recurrent 'colds', a productive cough and an increased incidence of respiratory infection with resultant absenteeism from school.

Similarly, more recent studies of self-reported asthmatic individuals living in homes with flue-less gas heaters have shown significant effects of NO_2 exposures to those aged ≤14 years with chest tightness, breathlessness on exertion and asthma attacks experienced either the same day or with one day lag (Smith et al., 2000). The range of median indoor levels of NO_2 measured by positional passive samplers in homes during this study were indicated to be between 0-147 ppb (0-277 µg/m^3) with time weighted average levels measured by personal passive sampler of 0-1,760 ppb (0-3,300 µg/m^3)[11]. Subsequent investigations with flue-less space heaters in primary schools indicated that over the 12 week winter heating period asthma symptoms were significantly higher[12] in children exposed to gas combustion products with mean NO_2 levels of 47.0 ppb (88 µg/m^3) versus children in schools where a replacement intervention programme had removed or replaced the flue-less gas heaters, leading to a mean NO_2 level of 15.5 ppb (29.3 µg/m^3) (Pilotto et al., 2004).

Based upon a review of the literature, Streeton (1997) considered short-term ambient exposures to 200-300 ppb (375-565 µg/m^3) NO_2 and chronic exposures between 40-80 ppb (75-150 µg/m^3) capable of causing recurrent upper and lower respiratory tract symptoms, an increased incidence of respiratory infection and onset of symptoms in mild asthmatics. Streeton (1997) considered these effects as a low observed adverse effect levels (LOAEL) and has suggested that an uncertainty factor of 2 need apply to account for susceptible people within the population therefore establishing a short-term guideline in the range 100-150 ppb as a 1 hour average and a chronic guideline between 20-40 ppb for longer term exposures as an annual average (Streeton, 1997).

The recommendations of Streeton (1997) form the basis of the current air guideline for acute and chronic exposure to NO_2 in the National Environment Protection Measure (NEPM 1998). Standards set in the NEPM are 0.12 ppm (226 µg/m^3) measured as a 1-hour average for acute exposure to NO_2 and 30 ppm (56 µg/m^3) measured as an annual average for chronic exposure to NO_2.

The WHO (1997a, 2000a) took a different approach to reach a similar conclusion to that of Streeton (1997). The WHO noted the epidemiological studies suggesting human health effects associated with long-term NO_2 exposures however the WHO (1997a) state this is supported by animal toxicological

[11] The mean daily maximal 10 minute levels of pollutants measured outdoors were 9.8 ppb (19 µg/m^3) NO_2; 2.9 ppb (8.3 µg/m^3) SO_2; 17.5 ppb (37.5 µg/m^3) O_3.
[12] Unadjusted relative risks (RR) were 0.32 for difficulty breathing at night p=0.004; RR 0.41 for difficulty breathing during the day p=0.045; RR 0.45 chest tightness during the day p=0.008; RR 0.39 asthma attacks during the day p=0.034.

findings showing increased susceptibility to respiratory infections and impairment of host defences as a result of subchronic or chronic exposures to NO_2 concentrations near ambient concentrations (i.e. 20-60 µg/m^3; 11-32 ppb). On the basis of a background level of 15 µg/m^3 (8 ppb) as determined in Finland during the 1980s (Jaakkola et al., 1991) and the fact that significant adverse health effects occur with an additional concentration of 28.2 µg/m^3 (15 ppb) or more, which is an estimate of an increased risk of about 20% for respiratory symptoms and disease (Hasselblad et al., 1992; WHO, 1997), an annual guideline value of 40 µg/m^3 (22 ppb) was derived by the WHO (1997a). The WHO considers the guideline value will be protective of most serious effects. The fact that a no-effect level for subchronic or chronic NO_2 exposure concentrations has not yet been determined was emphasised.

Since their publication, both the NEPM and the WHO air quality guidelines for particles, ozone, nitrogen dioxide and sulfur dioxide have been subject to review (NEPM, 2010; WHO, 2006). In both instances review of the guidelines considered newly available information from various locations around the world, including Australia. The WHO concluded that the scientific literature has not accumulated sufficient evidence to justify revising the existing NO_2 guidelines. According to NEPM (2010) available epidemiological information indicates increased hospital admissions and emergency department attendance for respiratory symptoms, particularly in asthmatics and children, following short-term exposure to ambient concentrations from 0.018 to 0.036 ppm (24 hour average) (NEPM 2010). However, the available information remains under consideration by the NEPM and no changes to the standards have been made at this point in time (NEPM 2010).

Interactive effects with allergens in Humans

There is some evidence to suggest that NO_2 exposure can enhance the response of an asthmatic to allergens. Volunteers with mild asthma exposed to 400 ppb (820 µg/m^3) NO_2 for 1h and who then immediately underwent a fixed-dose house dust mite challenge displayed significant decreases in FEV_1 results for early (2h after allergen; -18.64%, $p<0.009$) and late (-8.13%, $p<0.02$) phase asthmatic responses compared to air (-14.92% & -2.85 respectively) (Tunnicliffe et al., 1994).

Similarly, subjects with mild asthma and allergies to birch or grass pollens, who were exposed on four consecutive days to 500 µg/m^3 (265 ppb) NO_2 for 30 minutes, had a significantly increased asthmatic response after exposure to NO_2 and allergen (non-symptomatic dose 4h after NO_2) with a fall in early phase (15 minutes following allergen exposure) forced expiratory volume in one second of -25% for NO_2 compared to -0.4% for air, which was still significant ($p=0.01$) 3-10h after allergen exposure (Strand et al., 1997; Strand et al., 1998). The delayed effect of bronchial responsiveness has been investigated and it was found that 110 µg (median) of histamine diphosphate (vs. 203 µg on air) was required as the

provocative dose 5h after 30 minutes exposure to 488 µg/m^3 (260 ppb)[13] NO_2 to cause 100% increase in specific airways resistance (Strand et al., 1996).

Conclusions:

- Concentrations of around 2,000 µg/m^3 (~1,000ppb) are needed to affect respiration of healthy people.
- The low effect level for increased bronchial reactivity in sensitive asthmatics is 375-575 µg/m^3 (~200-300 ppb) for exposures from 20 minutes up to 4hours.
- The no effect level for increased bronchial reactivity is ~200 ppb.
- The increased bronchial reactivity may remain for up to 10 hours after cessation of NO_2 exposure.
- Table A1 presents a summary of guideline values established relating to acute and chronic exposure to NO_2.

[13] Mean Temperature 25.5±0.6^{o}C

Table A1. Summary of national and international guideline values established relating to acute and chronic exposure to NO_2

Guideline [a] µg/m^3 / ppb			Derivation	Reference
Nitrogen dioxide (NO_2) Acute Guidelines				
246	120	Ambient air quality guideline 1 hr average	The Australian National Environmental Protection Council ambient air quality standard. It is based on a low observed adverse effect level (LOAEL) of 0.2 to 0.3 ppm derived from statistical reviews of epidemiological data suggesting an increased incidence of lower respiratory tract symptoms in children and aggravation of asthma. An uncertainty factor of 2 to protect susceptible people (i.e. asthmatic children) was applied to the LOAEL.	NEPC (1998), Streeton (1997)
217	106	Ambient air quality guideline 1 hr average	Lowest concentration causing small (~5%) changes in lung function in mild asthmatics is 560 µg/m^3. Some but not all studies show increased responsiveness to bronchoconstrictors at NO_2 levels as low as 376–560 µg/m^3. In other studies, higher levels had no such effect. Allergen challenges showed no effects at 190 µg/m^3. According to WHO there have been no studies of 1 hour exposures to NO_2 at 100µg/m^3.	WHO (2000b)
513	250	Inhalation reference exposure level (REL) 1 hr average	The REL is also the ambient air quality standard of California. It is the no observed adverse effect level in sensitive asthmatics for NO_2 mediated increased responsiveness to other bronchoconstrictors (e.g. exercising in cold air).	OEHHA (1999)
Nitrogen dioxide (NO_2) Chronic Guidelines				
62	30	Ambient air guideline Annual avg	A low observed adverse effect level (LOAEL) of the order of 40 - 80 ppb (approx 75-150 µg/m^3) during early and middle childhood years can lead to the development of recurrent upper and lower respiratory tract symptoms, such as recurrent ‘colds’, a productive cough and an increased incidence of respiratory infection with resultant absenteeism from school. An uncertainty factor of 2 was applied to the LOAEL to account for susceptible people within the population resulting in a guideline of 20-40 ppb (38-75 µg/m^3).	NEPC (1998), Streeton (1997)
43	21	Ambient air guideline Annual avg	WHO (1997a) reviewed the epidemiological studies suggesting human health effects associated with long-term NO_2 exposures. On the basis of a background level of 15 µg/m^3 (8 ppb) and the fact that significant adverse health effects could be expected occur with an additional level of 28.2 µg/m^3 (15 ppb) or more, an annual guideline value of 40 µg/m^3 (0.023 ppm) was derived by the WHO (1997).It is considered guideline will be protective of most serious effects. The fact that a no-effect level for subchronic or chronic NO_2 exposure concentrations has not yet been determined should be emphasized.	WHO (2000b), WHO (1997)

[a] Please note there are also standards set by the EC and the UK these are the same as those set by WHO.

References for A1.1

Bauer, M.A., Utell, M.J., Morrow, P.E., Speers, D.M. and Gibb, F.R. (1986). Inhalation of 0.30 ppm nitrogen dioxide potentiates exercise-induced bronchospasm in asthmatics. Am. Rev. Respir. Dis. 134(6): 1203 - 1208. (As cited in IEH, 1996).

Bylin, G., Hedenstierna, G., Lindvall, T. and Sundin, B. (1988). Ambient nitrogen dioxide concentrations increase bronchial responsiveness in subjects with mild asthma. Eur. Respir. J. 1(7): 606 - 612.

Hasselblad, V., Eddy, D.M. and Kotchmar, D.J. (1992). Synthesis of environmental evidence: nitrogen dioxide epidemiology studies. J. Air Waste Manage. Assoc. 42: 662 - 671.

IEH (1996). Assessment on Indoor Air Quality in the Home: Nitrogen Dioxide, Formaldehyde, Volatile Organic Compounds, House Dust Mites, Fungi and Bacteria. Assessment A2. Medical Research Council. Institute for Environment and Health. Leicester, United Kingdom. http://www.silsoe.cranfield.ac.uk/ieh/pdf/A2.pdf

Jaakkola, J.J.K., Paunio, M., Virtanen, M. and Heinonen, O.P. (1991). Low-level air pollution and upper respiratory infections in children. Am. J. Public Health 81: 1060 - 1063. (As cited in WHO, 1997).

Kraft, M., Eikmann, T., Kappos, A., Künzli, N., Rapp, R., Schneider, K., Seitz, H., Voss, J-U. and Wichmann, E-H. (2005). The German view: Effects of nitrogen dioxide on human health – derivation of health-related short-term and long-term values. Int. J. Hyg. Environ. Health 208: 305 – 318.

Morrow, P.E., Utell, M.J., Bauer, M.A., Smeglin, A.M., Frampton, M.W., Cox, C., Speers, D.M. and Gibb, F.R. (1992). Pulmonary performance of elderly normal subjects and subjects with chronic obstructive pulmonary disease exposed to 0.3 ppm nitrogen dioxide. Am. Rev. Respir. Dis. 145(2 Pt 1): 291 - 300.

NEPM (1998). Ambient Air Quality National Environmental Protection Measure (NEPM). National Environmental Protection Council (NEPC).

NEPM (2010). Review of the National Environment Protection (Ambient Air Quality) Measure: Discussion Paper Air Quality Standards. National Environmental Protection Council (NEPC).

OEHHA (1999). Nitrogen Dioxide Acute Toxicity Summary; Determination of Acute Reference Exposure Levels for Airborne Toxicants. Office of Environmental Health Hazard Assessment Californian Environmental Protection Agency. http://www.oehha.ca.gov/air/acute_rels/pdf/10102440A.pdf

Pilotto, L.S., Nitschke, M., Smith, B.J., Pisaniello, D., Ruffin, R.E., McElroy, H.J., Martin, J., Hiller, J.E. (2004). Randomized controlled trial of unflued gas heater replacement on respiratory health of asthmatic schoolchildren. Int. J. Epidemiol. 33(1): 208 - 214.

Roger, L.J., Horstman, D.H., McDonnell, W.F., Kehrl, H., Seal, E., Chapman, R.S. and Massaro, E.J. (1985a). Pulmonary effects in asthmatics exposed to 0.3 ppm NO_2 during repeated exercise. Toxicologist 5: 70. (As cited in IEH, 1996).

Roger, L. J., Kehrl, H. R., Hazucha, M. and Horstman, D. H. (1985b). Bronchoconstriction in Asthmatics Exposed to Sulfur Dioxide during Repeated Exercise. J Appl Physiol. 59: 784-791.

Smith, B.J., Nitschke, M., Pilotto, L.S., Ruffin, R.E., Pisaniello, D.L. and Willson, K.J. (2000). Health effects of daily indoor nitrogen dioxide exposure in people with asthma. Eur. Respir. J. 16(5): 879 - 885.

Strand, V., Rak, S., Svartengren, M. and Bylin, G. (1997). Nitrogen dioxide exposure enhances asthmatic reaction to inhaled allergen in subjects with asthma. Am. J. Respir. Crit. Care Med. 155(3): 881 - 887.

Strand, V., Salomonsson, P., Lundahl, J. and Bylin, G. (1996). Immediate and delayed effects of nitrogen dioxide exposure at an ambient level on bronchial responsiveness to histamine in subjects with asthma. Eur. Respir. J. 9(4): 733 - 740.

Strand, V., Svartengren, M., Rak, S., Barck, C. and Bylin, G. (1998). Repeated exposure to an ambient level of NO2 enhances asthmatic response to a nonsymptomatic allergen dose. Eur. Respir. J. 12(1): 6 - 12.

Streeton, J. A. (1997). A review of existing health data on six air pollutants. Prepared for the National Environment Protection Council. May. NEPC Service Corporation.
http://www.ephc.gov.au/pdf/Air_Quality_NEPM/6_pollutants_report_em_Streeton.pdf

Tunnicliffe, W.S., Burge, P.S. and Ayres, J.G. (1994). Effect of domestic concentrations of nitrogen dioxide on airway responses to inhaled allergen in asthmatic patients. Lancet 344(8939-8940): 1733 - 1736.

WHO (1997). Environmental Health Criteria 188, Nitrogen Oxides. Second Edition. World Health Organisation. Geneva. http://www.inchem.org/documents/ehc/ehc/ehc188.htm

WHO (2000a). Guidelines for Air Quality. World Health Organisation, Geneva.

WHO (2000b). Air Quality Guidelines for Europe 2nd Edition. World Health Organisation Regional Office for Europe (WHO Regional Publications, European Series Number 91).
http://www.euro.who.int/document/e71922.pdf

WHO (2006). Air Quality Guidelines. Global Update. 2005. Particulate Matter, Ozone, Nitrogen Dioxide and Sulfur Dioxide. World Health Organisation Regional Office for Europe, Copenhagen.
http://www.ephc.gov.au/sites/default/files/AAQ_DiscPpr__Review_of_the_AAQ_NEPM_Discussion_Paper_AQ_Standards_Final_201007.pdf

A1.2 Particulate Matter review of recent literature with emphasis on diesel emissions

Summary

Health based ambient PM air guidelines (summarised in Table A1.3) have been set using data from epidemiological studies conducted in large urban populations. These have demonstrated statistical associations between the concentration of fine (≤ 2.5µm) airborne particulates ($PM_{2.5}$) and a number of health effects. The data supporting health effects from exposure to coarse urban particulates ($PM_{10-2.5}$) is markedly less convincing but is suggestive of them being implicated in some health effects. Nevertheless the weight of evidence from recent studies indicates that in sufficiently exposed susceptible sub-populations, fine urban airborne particulates ($PM_{2.5}$) are markedly more detrimental to health than coarse urban particulates ($PM_{10-2.5}$). There is data associating urban PM_{10} with health effects but this PM fraction also contains $PM_{2.5}$.

There is general agreement in the scientific literature that there is a concentration-response relationship (with no indication of a threshold) between PM_{10}/$PM_{2.5}$ and various measures of population based health effects. The exact form of the relationship is unclear, depending upon the health measure some studies indicate the relationship to be linear while others suggest non-linearity (RIVM 2002). It is noted that people with compromised respiratory or cardiopulmonary function (either through disease or old age) are more susceptible to the effects of particulates.

What is particulate matter?

Fine particles ($PM_{2.5}$) are produced chiefly by combustion processes and by atmospheric reactions of various gaseous pollutants, whereas coarse particles ($PM_{10-2.5}$) are generally emitted directly as particles as a result of mechanical processes that crush or grind larger particles or by the resuspension of dusts. Sources of fine particles include, for example, motor vehicles, power generation, combustion sources at industrial facilities, and residential fuel burning. Sources of coarse particles include, for example, traffic-related emissions such as tyre and brake lining materials, direct emissions from industrial operations, construction and demolition activities, and agricultural and mining operations. Combustion-derived particles are components of fine PM ($PM_{2.5}$), typically among the most dominant components in the fine fraction of ambient air PM in urban areas. They are composed primarily of metals (and metal oxides), black or elemental carbon, primary and secondary organic compounds (e.g. PAHs), as well as sulphates, nitrates, ammonium and hydrogen ions (US EPA 2004).

Fine particles can remain suspended in the atmosphere for days to weeks and can be transported thousands of kilometres, whereas coarse particles generally deposit rapidly on the ground or other surfaces and are not readily transported across urban or broader areas. This generalisation is somewhat dependent also on wind speed (e.g. during dust storm events).

US EPA (2004, 2005, 2006, 2010) noted there are distinctions between:

- the character of the ambient mix of particles generally found in urban areas as compared to that found in rural areas, and
- the nature of the evidence concerning health effects associated with thoracic coarse particles generally found in urban versus rural areas.

The mix of thoracic coarse particles typically found in urban areas contains a number of contaminants that are not commonly present or not to the same degree in the mix of natural crustal particles that is typical of rural areas

Toxicological, controlled human exposure and epidemiological studies conducted for respirable particulate matter (PM) in ambient air are consistent as they show that there are a range of decrements on respiratory and cardiovascular health following both short term (acute) and long term (chronic) exposure.

While associations have been identified between PM and adverse health effects, considerable uncertainty remains with regards to the methods and approaches to understanding relationships between air pollution and health effects, which components (gas and/or aerosol) and/or sources are

most harmful, the mechanisms of actions of the pollutants and causal relationships, effect of confounding factors, and who are susceptible populations, especially for particulate matter since it is composed of many individual species.

Health effects of particulate matter

The relationships between ambient PM and adverse health effects is complicated and requires experts across a range of scientific disciplines from atmospheric sciences to exposure to health effects scientists.

There is differing amounts of evidence and the concentration responses are difficult to interpret due in part to different geographical and seasonal variations in particulate matter as well as the large diverse and growing body of literature on the health effects of PM. Recently the United States Environmental Protection Authority (US EPA 2010), and the World Health Organisation (2006) have published detailed reviews on the health effects of respirable particulate matter (PM).

Table A1.2 provides a summary overview of the short and long term effects of PM subcategorised as either $PM_{2.5}$ (fine) or $PM_{10-2.5}$ (coarse) respectively. It includes the US EPA conclusion on the available evidence for an effect (causal) in humans. Although the table is for PM from all sources additional comments have been included where the evidence suggests additional health endpoints for diesel emissions. The table shows that the strongest and most consistent evidence between PM and health effects occurs for $PM_{2.5}$.

Table A1.2: Summary of the evidence for health effects of $PM_{2.5}$, $PM_{10-2.5}$ [a])

		Causality[a]	Summary description of health effect
Fine Particulates ($PM_{2.5}$)			
Short term	Cardiovascular effects	Causal	Consistent positive associations predominantly for ischemic heart disease and congestive heart failure from epidemiologic studies relating ambient $PM_{2.5}$ concentrations with emergency department visits and hospital admissions. The epidemiologic evidence is supported by less consistent findings in controlled air pollutant volunteer studies and toxicology studies. An understanding of the suggested biological pathways linking PM exposure with CV disease is far from complete. It is thought that there are 3 generalised pathways; pulmonary oxidated stress and inflammation leading to direct actions that reduce supply of blood to the cardiac muscle and reduced blood flow, PM entering circulation leading to effects on vasculature and blood, and, changes in sympathetic nervous system. Susceptible subpopulations include the elderly, individuals with diabetes, patients with pre-existing coronary hearth disease, chronic lung disease or heart failure.
	Respiratory effects	Likely to be causal	There are consistent positive associations between short term ambient air concentrations and respiratory emergency department/hospital admissions for chronic obstructive pulmonary disease (COPD) and respiratory infections. There is also evidence that PM (particularly organic PM such as diesel PM) is related to for asthma emergency department/hospital admissions. The underlying mechanism Most of the data indicates a role for oxidative stress causing inflammation and immunotoxicity in airways and lungs, or a mechanism involving impairment of respiratory and cardiac neurological functions Additional information on diesel and its ability to act as an antigen is provided in the text below.
	Mortality	Causal	Epidemiologic studies predominantly in Europe and the US as well as individual city studies indicate a consistent positive association between short term exposure to $PM_{2.5}$ and daily mortality (all causes – non-traumatic, respiratory disease and cardiovascular disease. The underlying relationships are unclear with possible confounding by gaseous air pollutants, demographic, socioeconomic factors, seasonal and geographic variations.

		Causality[a]	Summary description of health effect
Long term	Cardiovascular effects	Causal	Large multicity US based epidemiologic studies provide consistent evidence of an association between long-term exposure to $PM_{2.5}$ and cardiovascular effects. Epidemiological studies examining sub-clinical markers show inconsistent findings. Toxicological studies provide evidence for accelerated development of atherosclerosis and have shown effects on coagulation, hypertension and vascular reactivity.
	Respiratory effects	Likely to be causal	Recent epidemiologic studies provide evidence of associations between long term exposure to $PM_{2.5}$ and decrements in lung function growth, increased respiratory symptoms and asthma development. Toxicity studies with diesel exhaust, controlled air pollutants and roadside dust have presented some evidence for altered pulmonary function, mild inflammation, immune suppression and histopathological changes (e.g. mucus cell hyperplasia). Exacerbated allergic responses have been demonstrated in animals exposed to diesel exhaust and wood smoke.
	Mortality	Causal	Epidemiologic studies support ambient exposure to $PM_{2.5}$ and cardiovascular (cardiopulmonary and ischemic heart disease) mortality. Associations have also been reported for lung cancer mortality but limited evidence exists for respiratory mortality.
	Reproductive and Developmental	Suggestive	Suggestive evidence is provided by epidemiologic studies for effects on low birth weight and infant mortality, especially due to respiratory causes during post neonatal period. The epidemiologic literature does not consistently report associations with preterm birth growth restriction, birth defects or decreased sperm quality. Mechanistic toxicological research is currently being investigated however to date there is little support for adverse birth outcomes such as low birth weight.

		Causality[a]	Summary description of health effect
	Cancer, mutagenicity, genotoxicity	Suggestive	Epidemiologic studies have shown a consistent positive association between $PM_{2.5}$ and lung cancer mortality but have not reported associations with lung cancer incidence. A number of studies have concluded that diesel exhaust particulates can be mutagenic and genotoxic. The International Agency for Research on Cancer (IARC) evaluated the evidence for the carcinogenicity of diesel exhausts as probably carcinogenic to humans (IARC Group 2A) based on: ○ limited evidence from epidemiology studies with workers (bus company and dock-side workers) and ○ sufficient evidence from animal studies (rats, mice Syrian hamsters and monkeys) showing benign and malignant lung tumours that were related to the exposure concentration. ○
Coarse Particulates ($PM_{10-2.5}$)			
Short term	Cardiovascular effects	Suggestive	Although positive associations have been identified with coarse particulates in single city studies, the results from multicity The available evidence provides a still equivocal answer to the question of a nonspecific role for particles in modulating toxicity and the extent to which size determines toxicity.
	Respiratory effects	Suggestive	
	Mortality	Suggestive	
Long term	Cardiovascular effects	Inadequate	Inadequate evidence is available because the are fewer (than $PM_{2.5}$) relevant studies to draw data from and there are limitations in available monitoring data characterizing ambient levels of $PM_{10-2.5}$ in prospective urban study areas. The US EPA (2009, 2010) concluded that uncertainties in characterizing risk for $PM_{10-2.5}$ are potentially significant enough at this time to limit the utility of those estimates.
	Respiratory effects	Inadequate	
	Mortality	Inadequate	
	Reproductive and Developmental	Inadequate	
	Cancer, mutagenicity, genotoxicity	Inadequate	

Although the detailed toxicological mechanism(s) by which particulate matter causes adverse health effects is not known, most of the data indicates a role for oxidative stress causing inflammation and immunotoxicity in airways and lungs, or a mechanism involving impairment of respiratory and cardiac neurological functions. As with all organs in direct contact with the external environment such effects are not uncommon to a wide variety of biological and non biological agents. These adverse health effects are particularly relevant to susceptible individuals (discussed below).

Studies reviewed by international agencies have shown inflammation to be central to producing the respiratory and cardiovascular health effects attributed to PM. PM can activate intracellular pathways and transcription factors leading to the up-regulation of genes responsible for inflammatory, immune and acute phase responses as well as genes responsible for antioxidant defence and xenobiotic metabolism (US EPA 2009; WHO 2006; NEPC 2010). Following PM exposure, transcription factor activation in macrophages and epithelial cells stimulate the production of soluble mediators involved in inflammatory and immune responses including cytokines, chemokines, proteases, and eicosanoids, which play a role in recruiting inflammatory cells such as neutrophils, monocytes, mast cells and eosinophils to the lung (US EPA 2009). The recruitment of these cells is often used as an indicator of an inflammatory response in experimental studies. It should be noted this is a normal adaptive or physiological response to foreign material, which can, under high exposure conditions or in compromised individuals, increase to a level of severity which may produce overt health effects. In the short term, inflammation can lead to airway hyperresponsiveness and in the long term may lead to morphological changes in the lung.

A number of epidemiology studies in humans have also found associations between acute and chronic ambient PM exposures and increases in cardiovascular-related deaths and/or morbidity indicators, such as heart rate variability. The emerging evidence in humans (epidemiological and controlled exposure studies with CAPs) indicates PM can affect cardiovascular function (e.g. measured by atrial fibrillation/flutter predictors), which could be of concern in susceptible individuals[14] (Section 9) (Liao et al. 2011; Liao et al. 2004; Whitsel et al. 2009; Devlin et al. 2003).

Huang and Ghio (2006) have suggested ambient pollution particle -induced vasoconstriction may be an important mechanism associated with cardiovascular morbidity in humans. One possible mechanism is that exposure to particles activate lung cells producing inflammatory mediators, which then trigger a wide array of vasoactive signals (resulting in systemic inflammation). Another possibility is that PM may exert direct effects on vascular reactivity. PM has been shown to constrict systemic arteries and increase blood pressure in humans, and decrease the diameter of pulmonary arterioles in rats and rabbits (e.g. concentrated ambient particles, motorcycle exhaust particles, ozone and CAPs, urban particles). Intratracheal instillation and aerosol inhalation studies in rats by Nurkiewicz et al. (2004, 2006, 2008, 2009) support a role for increased reactive oxygen and nitrogen species production in the microvascular wall following particle exposure as a mechanism for vasoreactivity.

Numerous studies have shown ambient PM (in particular diesel) can act as an adjuvant for allergic sensitisation (Alberg et al. 2009; Alessandrini et al. 2006, 2009; Archer et al. 2004; Granum et al. 2001)

[14] This may not be considered a significant effect in healthy individuals, but may constitute a shift in the number of individuals that are classed as having cardiovascular disease, with individuals at the sensitive end of a population distribution being affected.

with ultrafine particles having a greater effect than fine particles (de Haar et al. 2006; Li et al. 2009) and fine particles having a greater effect than course particles (Alberg et al. 2009; Dybing et al. 2004). Effect was generally found to increase with soluble organic (e.g. PAHs) or mineral content (e.g. Zn, Cu, Cd, SiO_2) (Diaz-Sanchez et al. 2000; Geng et al. 2006; He et al. 2010; Ichinose et al. 2008; Kang et al. 2010). Most of these studies have examined the effects of combustion-derived particles (e.g. ROFA) rather than those of geological origin (i.e. crustal).

Populations shown to be susceptible to the effects of airborne particles are primarily those with compromised health, especially respiratory and/or cardiopulmonary function. At risk groups include the elderly, people with existing respiratory disease such as asthma, chronic obstructive pulmonary disease (COPD) and bronchitis; people with cardiovascular disease; people with pulmonary infections such as pneumonia; and children (Streeton 1997). In relation to the data underpinning establishment of the national ambient air standard (NEPC 1998) most of the 'effects' due to particles are associated with exacerbation of existing disease states. The 'effects' observed with elevated PM_{10} concentrations are increased hospital visits and/or admissions for respiratory conditions, decrements in pulmonary function (especially in adults with obstructive airways disease but also in young children), increases in prevalence of pulmonary symptoms and increased mortality (Streeton 1997, RIVM 2002).

The health based ambient PM air guidelines available from competent agencies are summarised in Table A1.3. Some of these guidelines are not based on a threshold below which it is clear that health effects do not occur at a population level. It is important to note that thresholds are likely at an individual level however the underlying mechanisms of action for PM health effects are not fully understood and the information necessary to determine such thresholds is not currently available (US EPA 2009, NSW DEC 2005).

Table A1.3: Summary of derivation of guideline values for particulate matter

Guideline µg/m^3		Derivation	Reference
Particulates (PM_{10})			
50	Ambient air quality guideline 24 hour avg	The Australian National Environmental Protection Council ambient air quality standard was based on increased hospital visits and/or admissions for respiratory conditions, decrements in pulmonary function (especially in adults with obstructive airways disease but also in young children), increased prevalence of pulmonary symptoms and increased mortality.	NEPC (1998), Streeton (1997).
50	Ambient air quality guideline 24 hour avg	Based on increases in short-term mortality and the relationship between 24-hour and annual PM levels.	WHO (2006a,b)

Guideline µg/m^3		Derivation	Reference
50	Ambient air quality guideline 24 hour avg	Based on higher level of mortality, morbidity, hospitalisation, work-affected days, increased use of medication associated with increased concentrations of PM_{10}. The New Zealand Ministry for the Environment states there is no evidence of a threshold below which adverse health effects will not be observed.	NZ MfE (2002)
50	European Union Limit value 24 hour avg	The European limit value, not to be exceeded more than 35 times a calendar year, is based on the lowest reasonably practical value. The European review was unable to identify a threshold concentration below which ambient PM has no effect therefore the limit value was based on the lowest reasonably practical value.	EU (2004, 2008)
50	UK limit value 24 hour avg	The UK limit value is not to be exceeded more than 35 times a calendar year. No background documentation was found for the basis of this value, but it is likely the UK adopted the EU limit values (EU 2008).	UK Secretary of State (2010)
150	National air quality standard 24 hour avg	The national air quality standard of 150 µg/m^3 with no more than one expected exceedence per year on average over three years was first promulgated in 1979. The basis for the standard is not described in recent USEPA reviews of PM standards (US EPA, 2005, 2010) It is important to note the standard is currently under review. The USEPA is considering whether to revise the primary standard for coarse particulate matter (USEPA 2011).	US EPA (2004)
20	Ambient air quality guideline Annual avg	Based on the lowest levels of $PM_{2.5}$ at which total, cardiopulmonary and lung cancer mortality have been shown to increase with more than 95% confidence in response to $PM_{2.5}$ in the American Cancer Society study (Pope et al. 2002). A $PM_{2.5}$:PM_{10} ratio of 0.5 was used to derive the PM_{10} guideline value. This ratio is close to that observed typically in urban areas in developing countries and at the bottom of the range (0.5-0.8) found in urban areas in developed countries.	WHO (2006)
20	Ambient air quality guideline Annual avg	Based on higher level of mortality, morbidity, hospitalisation, work-affected days, increased use of medication associated with increased concentrations of PM_{10}. The New Zealand Ministry for the Environment states there is no evidence of a threshold below which adverse health effects will not be observed.	NZ MfE (2002)
40	European Union Limit value Annual avg	The European limit value is based on the lowest reasonably practical value. The Europeans reviewed the findings of the WHO and studies published since the WHO review and concluded that some studies suggest that long-term exposure to particulate matter is associated with possible effects below 20 µg/m^3 (as $PM_{2.5}$) or 30 µg/m^3 (as PM_{10}).	EU (2004, 2008)
40	UK limit value Annual avg	No background documentation was found for the basis of this value, but it is likely the UK adopted the EU limit values (EU 2008).	UK Secretary of State (2010)
Particulates ($PM_{2.5}$)			
25	Monitoring advisory standard 24 hour avg	An advisory reporting standard.	NEPC (2003)

Guideline µg/m^3		Derivation	Reference
25	Ambient air quality guideline 24 hour avg	Based on increases in short-term mortality and the relationship between 24-hour and annual PM levels.	WHO (2006)
35	National air quality standard 24 hour avg	Based on providing protection against health effects associated with short-term exposures (including premature mortality and increased hospital admissions and emergency room visits).	US EPA (2006)
8	Monitoring advisory standard Annual avg	An advisory reporting standard.	NEPC (2003)
10	Ambient air quality guideline Annual avg	Based on the lowest levels of $PM_{2.5}$ at which total, cardiopulmonary and lung cancer mortality have been shown to increase with more than 95% confidence in response to $PM_{2.5}$ in the American Cancer Society study (Pope et al. 2002).	WHO (2006)
25	UK limit value Annual avg	The basis of this value is not described.	UK Secretary of State (2010)
15	National air quality standard Annual avg	Based on providing protection against health effects associated with long-term exposure (premature mortality and development of chronic respiratory disease)	US EPA (2006)

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 5.3–27.4µg/m^3 with effects becoming more precise and consistently positive in locations with mean $PM_{2.5}$ concentrations of 15µg/m^3 and above (USEPA, 2009).

It is important to note that the WHO (2006) chronic guideline value for $PM_{2.5}$ is based on associations (small but important because of the size of the general population) for ambient fine particulate matter and cardiopulmonary and lung cancer mortality. The critical study used to derive the guideline is Pope (2002). This particular study was for the US population. Although annual average PM2.5 concentrations can vary spatially by a factor of 2 within a 50–100-km area, three-year average mass concentrations exceeding 15 µg/m^3 were measured at 50% of American urban sites. Twenty-four-hour $PM_{2.5}$ averages exceeded 65 µg/m^3 for 2% of the time at many Californian sites and this level was occasionally exceeded at all south-eastern sites. The mean $PM_{2.5}$ concentrations ranged between 21.1 µg/m^3 for the period 1973-1983 and 14.0 µg/m^3 for the period 1999-2000 (overall mean of 17.7 µg/m^3). Using a statistical analysis ('nonparametric smoothed'), the authors compared the relative risks with frequency plot of the mean (annual average) $PM_{2.5}$ concentration. No associations for all-cause mortality, cardiopulmonary mortality or lung cancer mortality were noted at 10 µg/m^3. Although it is not possible to conclude that this is a threshold for population-level effects to fine particulate matter at this concentration the effects are likely to be indistinguishable from background incidences for the health endpoints considered.

[a] The guideline for $PM_{2.5}$ is an advisory reporting standard expressed as an annual arithmetic average; the reference does not provide an annual average for PM_{10}, however a 24-hour average of 50 $\mu g/m^3$ is provided for PM_{10}.

[b] The guidelines are recommended as an annual arithmetic average. Additionally, 24-hour average guidelines of 25 and 50 $\mu g/m^3$ are provided for $PM_{2.5}$ and PM_{10} respectively; the recommendations act as an update to those made in WHO *Air Quality Guidelines for Europe* (2000).

[c] The guidelines are provided as annual average limit values. The limit value for $PM_{2.5}$ is to be met by January 2015; the reference also provides a 24-hour average guideline of 50 $\mu g/m^3$ for PM_{10} which is not to be exceeded more than 35 times in a calendar year.

[d] The guideline for PM_{10} is an annual arithmetic average; the reference does not provide an annual average for $PM_{2.5}$ however, the new Directive is introducing additional $PM_{2.5}$ objectives aimed at reducing ambient air $PM_{2.5}$ to 18 $\mu g/m^3$ by 2020.

[e] The guidelines are provided as 24-hour averages of 15 and 25 $\mu g/m^3$ for $PM_{2.5}$ and PM_{10} respectively; no annual average guideline value is provided.

[f] The guideline for $PM_{2.5}$ is recommended as an annual arithmetic average. To attain this standard, the 3-year average of the weighted annual mean $PM_{2.5}$ concentrations from single or multiple community-oriented monitors must not exceed 15.0 $\mu g/m^3$; the reference does not provide an annual average for PM_{10}, however 24-hour averages of 35 and 150 $\mu g/m^3$ are provided for $PM_{2.5}$ and PM_{10} respectively.

References for A1.2

Alessandrini, F., H. Schulz, S. Takenaka, B. Lentner, E. Karg, H. Behrendt and T. Jakob (2006). "Effects of ultrafine carbon particle inhalation on allergic inflammation of the lung." Journal of Allergy and Clinical Immunology 117(4): 824-830.

Alberg, T., F. R. Cassee, E.-C. Groeng, E. Dybing and M. LÃ¸vik (2009). "Fine Ambient Particles from Various Sites in Europe Exerted a Greater IgE Adjuvant Effect than Coarse Ambient Particles in a Mouse Model." Journal of Toxicology and Environmental Health, Part A 72(1): 1 - 13.

Alessandrini, F., I. Beck-Speier, D. Krappmann, I. Weichenmeier, S. Takenaka, E. Karg, B. Kloo, H. Schulz, T. Jakob, M. Mempel and H. Behrendt (2009). "Role of Oxidative Stress in Ultrafine Particle-induced Exacerbation of Allergic Lung Inflammation." Am. J. Respir. Crit. Care Med. 179(11): 984-991.

Archer, A. J., J. L. H. Cramton, J. C. Pfau, G. Colasurdo and A. Holian (2004). "Airway responsiveness after acute exposure to urban particulate matter 1648 in a DO11.10 murine model." American Journal of Physiology - Lung Cellular and Molecular Physiology 286(2): L337-L343.

De Haar, C., I. Hassing, M. Bol, R. Bleumink and R. Pieters (2006). "Ultrafine but not fine particulate matter causes airway inflammation and allergic airway sensitization to co-administered antigen in mice." Clinical & Experimental Allergy 36(11): 1469-1479.

Devlin, R. B., A. J. Ghio, H. Kehrl, G. Sanders and W. Cascio (2003). "Elderly humans exposed to concentrated air pollution particles have decreased heart rate variability." European Respiratory Journal 21(40 suppl): 76s-80s.

Diaz-Sanchez, D., M. Penichet-Garcia and A. Saxon (2000). "Diesel exhaust particles directly induce activated mast cells to degranulate and increase histamine levels and symptom severity." Journal of Allergy and Clinical Immunology 106(6): 1140-1146.

Dybing, E., T. Løvdal, R. B. Hetland, M. Løvik and P. E. Schwarze (2004). "Respiratory allergy adjuvant and inflammatory effects of urban ambient particles." Toxicology 198(1-3): 307-314.

EU (2004). Second position paper on particulate matter. CAFE Working group on particulate matter, European Commission. December 20th, 2004. http://ec.europa.eu/environment/archives/cafe/pdf/working_groups/2nd_position_paper_pm.pdf.

Geng, H., Z. Meng and Q. Zhang (2006). "In vitro responses of rat alveolar macrophages to particle suspensions and water-soluble components of dust storm PM2.5." Toxicology in Vitro 20(5): 575-584.

Granum, B., P. Gaarder, E. Groeng, R. Leikvold, E. Namork and M. Lovik (2001). "Fine particles of widely different compostion have an adjuvant effect n the production of allergen-specific antibodies." Toxicology Letters 118(3): 171-181.

He, M., T. Ichinose, S. Yoshida, M. Nishikawa, I. Mori, R. Yanagisawa, H. Takano, K.-i. Inoue, G. Sun and T. Shibamoto (2010). "Airborne Asian sand dust enhances murine lung eosinophilia." Inhalation Toxicology 22(12): 1012-1025.

Huang, Y.-C. and A. J. Ghio (2006). "Vascular Effects of Ambient Pollutant Particles and Metals." Current Vascular Pharmacology 4: 199-203.

Ichinose, T., S. Yoshida, K. Sadakane, H. Takano, R. Yanagisawa, K. Inoue, M. Nishikawa, I. Mori, H. Kawazato, A. Yasuda and T. Shibamoto (2008). "Effects of Asian Sand Dust, Arizona Sand Dust, Amorphous Silica and Aluminium Oxide on Allergic Inflammation in the Murine Lung." Inhalation Toxicology 20(7): 685-694.

Kang, X., N. Li, M. Wang, P. Boontheung, C. Sioutas, J. R. Harkema, L. A. Bramble, A. E. Nel and J. A. Loo (2010). "Adjuvant effects of ambient particulate matter monitored by proteomics of bronchoalveolar lavage fluid." PROTEOMICS 10(3): 520-531.

Li, N., M. Wang, L. A. Bramble, D. A. Schmitz, J. J. Schauer, C. Sioutas, J. R. Harkema and A. E. Nel (2009). "The Adjuvant Effect of Ambient Particulate Matter Is Closely Reflected by the Particulate Oxidant Potential." Environmental Health Perspectives 117(7): 1116-1123.

Liao, D., Y. Duan, E. A. Whitsel, Z.-j. Zheng, G. Heiss, V. M. Chinchilli and H.-M. Lin (2004). "Association of Higher Levels of Ambient Criteria Pollutants with Impaired Cardiac Autonomic Control: A Population-based Study." American Journal of Epidemiology 159(8): 768-777.

Liao, D., M. L. Shaffer, F. He, S. Rodriguez-Colon, R. Wu, E. A. Whitsel, E. O. Bixler and W. E. Cascio (2011). "Fine Particulate Air Pollution is Associated with Higher Vulnerability to Atrial Fibrillation-the APACR Study." Journal of Toxicology and Environmental Health, Part A: Current Issues 74(11): 693 - 705.

NEPC (1998). National Environment Protection (Ambient Air Quality) Measure - Revised impact statement. National Environment Protection Council, Canberra. http://www.ephc.gov.au/sites/default/files/AAQ_ImpStat__AAQ_NEPM_Revised_Impact_Statement_Final_199806.pdf.

NEPC (2003). National Environment Protection (Ambient Air Quality) Measure. National Environment Protection Council, Canberra. http://www.ephc.gov.au/sites/default/files/AAQ_NEPM__Ambient_Air_Quality_NEPM_Varied_scaleplus_Final_200305_1.pdf.

NEPC (2010). Review of the National Environment Protection (ambient air quality) measure. Discussion paper. Air quality standards. Canberra, Prepared for the National Environment Protection Council. http://www.ephc.gov.au/sites/default/files/AAQ_DiscPpr__Review_of_the_AAQ_NEPM_Discussion_Paper_AQ_Standards_Final_201007.pdf.

NSW DEC (2005). Air Pollution Economics Health Costs of Air Pollution in the Greater Sydney Metropolitan Region. NSW Department of Environment and Conservation.

Nurkiewicz, T. R., D. W. Porter, M. Barger, V. Castranova and M. A. Boegehold (2004). "Particulate Matter Exposure Impairs Systemic Microvascular Endothelium-Dependent Dilation." Environmental Health Perspectives 112(13): 1299-1306.

Nurkiewicz, T. W., D. W. Porter, M. Barger, L. Millecchia, K. M. K. Rao, P. J. Marvar, A. F. Hubbs, V. Castranova and M. A. Boegehold (2006). "Systemic Microvascular Dysfunction and Inflammation after Pulmonary Particulate Matter Exposure." Environmental Health Perspectives 114(3): 412-419.

Nurkiewicz, T. W., D. W. Porter, A. F. Hubbs, J. L. Cumpston, B. T. Chen, D. G. Frazer and V. Castranova (2008). "Nanoparticle inhalation augments particle-dependent systemic microvascular dysfunction." Particle and Fibre Toxicology 5: 1.

Nurkiewicz, T. R., D. W. Porter, A. F. Hubbs, S. Stone, B. T. Chen, D. G. Frazer, M. A. Boegehold and V. Castranova (2009). "Pulmonary Nanoparticle Exposure Disrupts Systemic Microvascular Nitric Oxide Signaling." Toxicological Sciences 110(1): 191-203.

NZ Ministry of for the Environment (2002). Ambient air quality guidelines. New Zealand Ministry for the Environment.

Pope III, C. A., Burnett, R. T., Thun, M. J., Calle, E. E., Krewski, D., Ito, K. and Thurston, G. D. (2002). Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution The Journal of American Medical Associates. 287 (9): 1132-1141.

RIVM (2001). Re-evaluation of human-toxicological maximum permissible risk levels. RIVM Report 711701 025. National Institute of Public Health and the Environment (RIVM), Bilthoven.
Streeton, J. A. (1997). A review of existing health data on six pollutants. National Environment Protection Council.

RIVM (2002). On health risks of ambient PM in the Netherlands. Netherlands Aerosol Programme, National Institute for Public Health and the Environment, Netherlands.

Streeton, J. A. (1997). A review of existing health data on six pollutants, National Environment Protection Council.

UK Secretary of State (2010). The Air Quality Standards Regulation 2010. Statutory Instrument 2010 No. 1001. http://www.legislation.gov.uk/uksi/2010/1001/contents/made.

US EPA (2004). Air Quality Criteria for Particulate Matter. National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency, Report no. EPA/600/P–99/002aF and EPA/600/ P–99/002bF. October 2004.

US EPA (2005). Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency. Report no. EPA EPA–452/ R–05–005a. December 2005.

US EPA (2006). National Ambient Air Quality Standards for Particulate Matter; Final Rule. 40 CFR Part 50. Federal Register, Tuesday, October 17, 2006. pp 61144 – 61233.

US EPA (2010). Integrated Science Assessment for Particulate Matter. United States Environmental Protection Agency. EPA/600/R-08/139F.
http://www.epa.gov/ncea/pdfs/partmatt/Dec2009/PM_ISA_full.pdf.

US EPA (2011). Policy assessment for the review of the particulate matter National Ambient Air Quality Standards. United States Environmental Protection Agency. EPA 452/R-11-003.

Whitsel, E. A., Quibrera, P. M., Christ, S. L., Liao, D., Prineas, R. J., Anderson, G. L. and Heiss, G. (2009). Heart Rate Variability, Ambient Particulate Matter Air Pollution, and Glucose Homeostasis: The Environmental Epidemiology of Arrhythmogenesis in the Women's Health Initiative. American Journal of Epidemiology. 169 (6): 693-703.

WHO (2006a). Air quality guidelines: Global update 2005. World Health Organization

WHO (2006b). Health risks of particulate matter from long-range transboundary air pollution. World Health Organization. E88189, Copenhagen, Denmark.

Appendix 2: Hazard quotient and hazard index calculations

Hazard Quotients and Hazard Indices have been calculated as discussed in section 5 and presented below in Tables A2.1 to Table A2.8. A brief summary of the calculations performed is provided below.

Hazard quotients (HQ) were calculated for acute and chronic exposures, using Equation A2.1:

$$HQ = \frac{GLC}{AGV}$$ **Equation A2.1**

The acute HQ were calculated using ground level concentration (GLC) at various percentiles (50th, 95th, 99th, 99.7th and 99.9th) and the maximum GLC. The chronic HQ were calculated using an average GLC.

The hazard index (HI) was calculated for both acute and chronic exposures assuming additive effects using Equation A2.2.

$HI_j = \sum HQ_{i \ldots j}$ **Equation A2.2**

Where HI_j is the sum of HQ's for all pollutants from i to j

The HI presented in this appendix and used in preparation of diagrams through the report were determined by summing the HQ calculated for $PM_{2.5}$ and NO_2. The HI does not include the HQ calculated for PM_{10} as the majority of the emissions from the site (90%) were considered to belong to the $PM_{2.5}$ fraction.

A2.1 Hazard Quotients for Scenario 1

Tables A2.1 to A2.4 below present the HQ and HI calculated for acute and chronic exposures due to incremental exposures; i.e.: scenario 1 or emissions only from the proposed IMT. The acute HQ for scenario 1 are provided below in Table A2.1 followed by Table A2.2 presenting the acute HI calculated from summing the HQ for $PM_{2.5}$ and NO_2 as discussed above and in section 5. The chronic HQ is presented in Table A2.3 and the chronic HI presented in Table A2.4.

Table A2.1: Scenario 1: Acute Hazard Quotients for particulate matter (PM_{10}, $PM_{2.5}$ and NO_2) at given various percentiles

Compound	Percentile	R1		R2		R3		R4		R5		R6		R7	
		µg/m³	HQ	µg/m³	HQ	µg/m³	HQ	µg/m³	HQ	µg/m³	HQ	µg/m³	HQ	µg/m³	HQ
PM_{10} (AGV = 50µg/m³)	50th Percentile	1.35	0.027	1.51	0.030	1.50	0.030	0.17	0.003	0.25	0.005	0.55	0.011	0.38	0.008
	95th Percentile	3.54	0.071	4.71	0.094	5.62	0.11	0.89	0.018	1.60	0.032	2.48	0.050	1.68	0.034
	99th Percentile	4.92	0.098	5.61	0.11	7.15	0.14	1.46	0.029	2.53	0.051	3.09	0.062	2.21	0.044
	99.7th Percentile	6.87	0.14	6.33	0.13	7.49	0.15	1.98	0.040	3.04	0.061	3.52	0.070	2.93	0.059
	99.9th Percentile	7.02	0.14	6.36	0.13	7.50	0.15	2.00	0.040	3.06	0.061	3.53	0.071	2.98	0.060
	Maximum	7.02	0.14	6.36	0.13	7.50	0.15	2.00	0.040	3.06	0.061	3.53	0.071	2.98	0.060
$PM_{2.5}$ (AGV = 25µg/m³)	50th Percentile	1.22	0.049	1.36	0.054	1.35	0.054	0.15	0.006	0.23	0.009	0.50	0.020	0.34	0.014
	95th Percentile	3.18	0.13	4.24	0.17	5.06	0.20	0.80	0.032	1.44	0.058	2.23	0.089	1.51	0.060
	99th Percentile	4.43	0.18	5.05	0.20	6.44	0.26	1.32	0.053	2.28	0.091	2.78	0.11	1.99	0.080
	99.7th Percentile	6.19	0.25	5.70	0.23	6.74	0.27	1.78	0.071	2.73	0.11	3.17	0.13	2.63	0.11
	99.9th Percentile	6.32	0.25	5.72	0.23	6.75	0.27	1.80	0.072	2.75	0.11	3.18	0.13	2.68	0.11
	Maximum	6.32	0.25	5.72	0.23	6.75	0.27	1.80	0.072	2.75	0.11	3.18	0.13	2.68	0.11
NO_2 (AGV = 246µg/m³)[1]	50th Percentile	0.36	0.001	0.65	0.003	0.18	0.001	0.00	0.000	0.00	0.000	0.00	0.000	0.00	0.000
	95th Percentile	24	0.10	25	0.10	23	0.09	10.8	0.04	19	0.08	28	0.12	11.5	0.05
	99th Percentile	42	0.17	41	0.17	38	0.16	32	0.13	41	0.17	47	0.19	33	0.14
	99.7th Percentile	50	0.20	51	0.21	47	0.19	40	0.16	48	0.20	55	0.22	46	0.19
	99.9th Percentile	55	0.22	63	0.26	58	0.23	48	0.20	54	0.22	62	0.25	56	0.23
	Maximum	72	0.29	74	0.30	67	0.27	58	0.23	85	0.35	84	0.34	79	0.32

[1] The AGV used for NO_2 in HQ calculations was based on STP conditions consistent with modelled data provided by PAE (2011).

Table A2.2: Scenario 1: Acute Hazard Indices (HI = HQ $PM_{2.5}$ + NO_2).

Percentile	R1	R2	R3	R4	R5	R6	R7
50th Percentile	0.05	0.06	0.05	0.01	0.01	0.02	0.01
95th Percentile	0.23	0.27	0.30	0.08	0.14	0.20	0.11
99th Percentile	0.35	0.37	0.41	0.18	0.26	0.30	0.22
99.7th Percentile	0.45	0.43	0.46	0.23	0.31	0.35	0.29
99.9th Percentile	0.48	0.48	0.50	0.27	0.33	0.38	0.33
Maximum	0.55	0.53	0.54	0.31	0.46	0.47	0.43

Table A2.3: Scenario 1: Chronic Hazard Quotients for particulate matter (PM_{10}, $PM_{2.5}$ and NO_2).

Receptor	PM_{10}		$PM_{2.5}$		NO_2	
	µg/m³	HQ	µg/m³	HQ	µg/m³	HQ
R1	1.48	0.07	1.34	0.13	5.04	0.081
R2	1.78	0.09	1.60	0.16	5.40	0.087
R3	2.02	0.10	1.82	0.18	5.11	0.082
R4	0.26	0.01	0.23	0.023	1.60	0.026
R5	0.48	0.02	0.43	0.043	2.78	0.045
R6	0.74	0.04	0.67	0.067	4.42	0.071
R7	0.53	0.03	0.48	0.048	1.92	0.031

Table A2.4: Scenario 1: Chronic Hazard Indices (HI = HQ $PM_{2.5}$ + NO_2).

Receptor	HI
R1	0.21
R2	0.25
R3	0.26
R4	0.05
R5	0.09
R6	0.14
R7	0.08

A2.2 Hazard Quotients for Scenario 2

Tables A2.5 to A2.8 below present the HQ and HI calculated for acute and chronic exposures due to cumulative exposures; i.e.: scenario 2 or emissions from the proposed IMT including background. The acute HQ for scenario 2 are provided below in Table A2.5 followed by Table A2.6 presenting the acute HI calculated from summing the HQ for $PM_{2.5}$ and NO_2 as discussed above and in section 5. The chronic HQ is presented in Table A2.7 and the chronic HI presented in Table A2.8.

Table A2.5: Scenario 2: Acute Hazard Quotients for particulate matter (PM_{10}, $PM_{2.5}$ and NO_2) at given various percentiles

Compound	Percentile	Background		R1[1]		R2[1]		R3[1]		R4[1]		R5[1]		R6[1]		R7[1]	
		µg/m³	HQ	µg/m³	HQ	µg/m³	HQ	µg/m³	HQ	µg/m³	HQ	µg/m³	HQ	µg/m³	HQ	µg/m³	HQ
PM_{10} (AGV = 20µg/m³)	50th Percentile	18.2	0.36	19.7	0.39	20.1	0.40	20.5	0.41	18.5	0.37	18.7	0.37	19.0	0.38	18.7	0.37
	95th Percentile	33.1	0.66	34.5	0.69	35.4	0.71	35.8	0.72	33.1	0.66	33.8	0.68	34.4	0.69	34.0	0.68
	99th Percentile	40.6	0.81	43.1	0.86	43.3	0.87	43.3	0.87	40.7	0.81	41.4	0.83	41.7	0.8	41.8	0.84
	99.7th Percentile	43.2	0.86	44.8	0.90	44.7	0.89	44.0	0.88	43.2	0.86	43.6	0.87	44.9	0.9	43.7	0.87
	99.9th Percentile	43.5	0.87	46.2	0.92	45.9	0.92	44.1	0.88	43.5	0.87	43.8	0.88	45.3	0.91	44.7	0.89
	Maximum	43.7	0.87	46.9	0.94	46.5	0.93	44.1	0.88	43.7	0.87	43.9	0.88	45.5	0.91	45.2	0.90
$PM_{2.5}$ (AGV = 10µg/m³)	50th Percentile	6.7	0.27	7.6	0.30	8.0	0.32	8.1	0.32	6.7	0.27	6.9	0.28	7.2	0.29	6.9	0.28
	95th Percentile	14.0	0.56	15.7	0.63	16.2	0.65	17.0	0.68	14.0	0.56	14.2	0.57	14.6	0.59	14.3	0.57
	99th Percentile	19.0	0.76	20.1	0.80	19.8	0.79	20.1	0.80	19.1	0.76	19.4	0.77	19.3	0.77	19.2	0.77
	99.7th Percentile	20.3	0.81	21.4	0.85	24.6	0.98	**26.1**	1.0	20.8	0.83	20.5	0.82	21.5	0.86	21.8	0.87
	99.9th Percentile	24.5	1.0	**25.2**	1.0	**28.5**	1.1	**28.5**	1.1	24.8	1.0	25.0	1.0	**25.4**	1.0	**26.2**	1.0
	Maximum	**26.7**	1.1	**27.2**	1.1	**30.5**	1.2	**29.6**	1.2	**26.9**	1.1	**27.5**	1.1	**27.4**	1.1	**28.5**	1.1
NO_2 (AGV = 62µg/m³)[2]	50th Percentile	18	0.08	23	0.094	23	0.092	23	0.092	21	0.083	21	0.084	23	0.092	21	0.083
	95th Percentile	47	0.19	56	0.23	57	0.23	55	0.22	51	0.21	53	0.22	57	0.23	52	0.21
	99th Percentile	59	0.24	70	0.28	73	0.30	68	0.28	65	0.27	71	0.29	72	0.29	68	0.27
	99.7th Percentile	72	0.29	83	0.34	86	0.35	78	0.32	76	0.31	88	0.36	88	0.36	80	0.33
	99.9th Percentile	88	0.36	99	0.40	96	0.39	91	0.37	90	0.37	101	0.41	105	0.43	90	0.37
	Maximum	109	0.44	124	0.50	111	0.45	109	0.44	109	0.44	145	0.59	144	0.58	109	0.44

[1] For scenario 2 the concentration at each percentile provided has been determined by summing the background and incremental data at each time point (1 hour for NO_2, 24 hour for PM) before percentile data was calculated.
[2] The AGV used for NO_2 in HQ calculations was based on STP conditions consistent with modelled data provided by PAE (2011).

Table A2.6: Scenario 2: Acute Hazard Indices (HI = HQ $PM_{2.5}$ + NO_2).

Percentile	Background	R1	R2	R3	R4	R5	R6	R7
50th Percentile	0.3	0.4	0.4	0.4	0.4	0.4	0.4	0.4
95th Percentile	0.8	0.9	0.9	0.9	0.8	0.8	0.8	0.8
99th Percentile	1.0	1.1	1.1	1.1	1.0	1.1	1.1	1.0
99.7th Percentile	1.1	1.2	1.3	1.4	1.1	1.2	1.2	1.2
99.9th Percentile	1.3	1.4	1.5	1.5	1.4	1.4	1.4	1.4
Maximum	1.5	1.6	1.7	1.6	1.5	1.7	1.7	1.6

Table A2.7: Scenario 2: Chronic Hazard Quotients for particulate matter (PM_{10}, $PM_{2.5}$ and NO_2).

Receptor	PM_{10}		$PM_{2.5}$		NO_2	
	µg/m³	HQ	µg/m³	HQ	µg/m³	HQ
Background	20	0.98	8.2	0.72	21	0.34
R1	**21**	1.04	8.5	0.82	26	0.42
R2	**22**	1.06	8.8	0.85	26	0.42
R3	**22**	1.07	7.2	0.88	26	0.42
R4	20	0.98	7.4	0.72	23	0.37
R5	20	0.99	7.6	0.74	24	0.39
R6	**21**	1.01	7.4	0.76	26	0.41
R7	20	1.0	8.2	0.74	23	0.37

Table A2.8: Scenario 2: Chronic Hazard Indices (HI = HQ $PM_{2.5}$ + NO_2).

Receptor	HI
Background	1.1
R1	1.2
R2	1.3
R3	1.3
R4	1.1
R5	1.1
R6	1.2
R7	1.1