SIMTA Intermodal Terminal Facility- Stage 1

Screening Health Impact Assessment

SIMTA

SYDNEY INTERMODAL TERMINAL ALLIANCE

Part 4, Division 4.1, State Significant Development

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Report

REPORT

SCREENING HEALTH IMPACT ASSESSMENT SIMTA STAGE 1 PROPOSAL

Westley Owers – Hyder Consulting

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1 EXECUTIVE SUMMARY

A Screening Health Impact Assessment (HIA) has been conducted to support the development application for approval of the first stage of the SIMTA Project, being the Stage 1Proposal. The HIA has considered the issues raised by the community through consultation for the Concept Plan Approval and has examined the potential impacts of operational phase of the Stage 1 Proposal on the local and regional community.

The Stage 1site is surrounded by the suburbs of Casula, Wattle Grove, Glenfield and Moorebank in south western Sydney. A review of the demographics of the population and the baseline health status has found that there are no significant differences between the indicators within these communities and the rest of Sydney and NSW. There are no underlying health issues that would make these communities more vulnerable to the effects of environmental factors, such as air pollution or noise from the site operations than the rest of Sydney.

A health risk assessment (HRA) has been conducted to assess the air quality and noise impacts on the health of the local community. For the air quality assessment a range of health outcomes were investigated including increases in mortality and morbidity such as hospital admissions as well as increases in cancer risk due to potential exposure to air toxics and diesel emissions. The HRA was conducted using predicted air pollution levels modelled for the worst case scenario without mitigation measures to reduce emissions from the Stage 1 operations being implemented. The results of the HRA found that the increase in risk due to air pollution from the operations of the Proposal are low and in most cases are negligible. The cancer risk form the air toxics are well below acceptable risk level set by international agencies. The implementation of best practice measures as outlined in the Air Quality Impact Assessment will lead to further reductions in air pollution levels and the associated health risks.

The noise HRA has investigated the impact of noise from the operations of the Proposal and rail noise on sleep disturbance and cognitive development in children using the WHO community noise guidelines. The noise modelling which forms the basis of the HRA was undertaken for the worst case scenario without actions being implemented to mitigate noise from the Stage 1 operations and associated rail movements. The noise from the operations at the site meets the WHO criteria. There are some small exceedances of the sleep disturbance criteria from the rail noise; however, with the implementation of the best practice measures outlined in the Noise and Vibration Impact Assessment, these exceedances will be minimised and the risk to health of the local community low.

2 BACKGROUND

The SIMTA Project involves the development of an intermodal facility, including warehouse and distribution facilities, freight village (ancillary site and operational services), stormwater, landscaping, servicing and associated works on the eastern side of Moorebank Avenue, Moorebank (the SIMTA site). The SIMTA Project also includes a Rail link, within an identified rail corridor (the Rail Corridor), which connects from the southern part of the SIMTA site to the Southern Sydney Freight Line (SSFL) (the entire area, SIMTA site and Rail Corridor referred to as the Project site). The SIMTA Project is to be developed in three key stages:

- Stage 1- Construction of the Intermodal Terminal Facility and Rail link
- Stage 2- Construction of warehouse and Distribution Facilities
- Stage 3- Extension of the Intermodal Terminal Facility and completion of Warehouse and Distribution Facilities.

A summary of the approvals undertaken to date for the SIMTA Project, include:

- **EPBC Approval** (No. 2011/6229) granted in March 2014 for the impact of the SIMTA Project on listed threatened species and communities (sections 18 and 18A of the *Environment Protection and Biodiversity Conservation Act 1999* (EPBC Act)) and Commonwealth land (sections 26 and 27A of the EPBC Act).
- **Concept Approval** (No. 10_0193) granted by the Planning Assessment Commission (PAC) on the 29 September 2014 for the 'Concept Approval' of the SIMTA Project under Part 3A of the EP&A Act.

Both of these approvals involved the preparation of design and environmental assessment documentation.

2.1 Report purpose

This report has been prepared for approval of the initial stage of the SIMTA Project, known as the Stage 1 Proposal. A summary of the works included in the Stage 1 Proposal is provided below. This report has been prepared to support a State Significant Development (SSD) Application for which approval is sought under Part 4, Division 4.1 of the *Environmental Planning and Assessment Act 1979* (EP&A Act).

This report has been prepared in accordance with the Secretary's Environmental Assessment Requirements (SEARs) (ref: SSD 14-6766 and dated December 2014). Table 1 provides a summary of the SEARs and the section where they have been addressed in this report.

Table 1: SEARs (SSD 14-6766) compliance table

Section/number	SEARs	Where addressed
2. Compliance with the Approved Concept Plan	Where relevant, the assessment of the key issues below, and any other significant issues identified in the risk assessment, must include: •adequate baseline data; •consideration of potential cumulative impacts due to other development in the vicinity; •measures to avoid, minimise and if necessary, offset the predicted impacts, including detailed contingency plans for managing any significant risks to the environment; •a health impact assessment of local and regional impacts associated with the development, including those health risks associated with relevant key issues; and •consideration of the cumulative impacts of this proposal with the adjacent Moorebank Intermodal Terminal proposal	Section 5 and Section 6

A Screening Health Impact Assessment (HIA) has been undertaken and the results are presented in this Technical Report. The HIA has examined the population profile and baseline health status of the local community and incorporates work previously done in the Social Impact Commentary done for the Concept Approval. The consultation outcomes for the Concept Plan Approval have been used to inform the key issues to be addressed as part of the HIA. A quantitative health risk assessment has been undertaken to address the potential health risks from exposure to air pollution and noise from the Stage 1Proposal operations.

2.2 Key terms

Table 2 provides a summary of the key terms which are included within this report. Figure 1 also provides an indication of the site areas discussed in this table.

Table 2- Key terms

	Description
Concept Plan Approval	Concept Plan Approval (MP 10_0193) granted on 29 September 2014 for the development of the SIMTA Moorebank Intermodal Terminal Facility at Moorebank. This reference includes the associated Conditions of Approval (CoA) and Statement of Commitments (SoC) which form the approval documentation for the Concept Plan Approval.
Approval	Approval (No. 2011/6229) granted under the EPBC Act on March 2014 by the Commonwealth Department of Environment for the development of the SIMTA Moorebank Intermodal Terminal Facility at Moorebank.
SIMTA Project	The SIMTA Moorebank Intermodal Terminal Facility at Moorebank as approved by the Concept Plan (MP_10_0913).
SIMTA site	Includes the former Defence National Storage and Distribution Centre (DNSDC) site, the land owned by SIMTA which is subject to the Concept Plan Approval (refer to Figure 1).
Rail Corridor	Area defined as the 'Rail Corridor' within the Concept Plan Approval. The Rail link is also included within this area (refer to Figure 1).
Project site	Includes the SIMTA site and the Rail Corridor, i.e. the entire site area which was approved under the Concept Plan Approval (refer to Figure 1).
Stage 1 site	The subject of this EIS, the western part of the SIMTA site which includes all areas to be disturbed by the Stage 1 Proposal (including the Operational area and Indicative Construction area) (refer to Figure 1). This area does not include the Rail Corridor.
Construction area	Extent of construction works, namely areas to be disturbed during construction of the Stage 1 Proposal (refer to Figure 1).
Operational area	Extent of operational activities for the operation of the Proposal (refer to Figure 1).
Proposal site	Includes the Stage 1 site and the Rail Corridor, i.e. the area for which approval (construction and operation) is sought within this EIS.
Rail link	The Rail link including the area on either side to be impacted by the construction works included in the Stage 1 Proposal.
Former DNSDC South	The land to the south of the operational footprint of the Intermodal Terminal, to the boundary fence of the former DNSDC.

	Description
Southern Boot Land	Commonwealth owned land to the south of Former DNSDC South, and to the north of the RailCorp Land (part of the Boot Land in the MIC proposal).
RailCorp Land	Lot 1 DP 825352 (part of the Rail Corridor) and owned by RailCorp.
The Proposal	Stage 1 of the SIMTA Moorebank Intermodal Terminal Facility including construction and operation of the intermodal terminal facility and Rail link, i.e. all works and built form for which approval is sought in this EIS/Technical Report.
MIC Proposal	The development of an intermodal facility, associated commercial infrastructure (warehousing) and a rail link (3 options have been provided) to be located on the MIC site, for which an approval, under Part 4, Division 4.1 of the *Environmental Planning and Assessment Act 1979*. This proposal is currently under assessment by the Department of Planning and Environment.
MIC site	The former School of Military Engineering site to the immediate west of the SIMTA site, across Moorebank Avenue.
Study area	The suburbs in close proximity to the SIMTA Project, including Moorebank, Glenfield, Casula and Wattle Grove.

Figure 1- Key terms and proposal locations

Figure 1: SIMTA Stage 1 Location Plan and key areas

2.3 Proposal overview

The Proposal involves the construction and operation of the necessary infrastructure to support a container freight road volume of 250,000 TEU (twenty-foot equivalent units) per annum. Specifically, Stage 1 includes the following key components, which together comprise the intermodal terminal facility:

- Truck processing, holding and loading areas- entrance and exit from Moorebank Avenue.
- Rail loading and container storage areas – installation of four rail sidings with adjacent container storage area serviced by manual handling equipment initially and overhead gantry cranes progressively. .
- Administration facility and associated car parking- light vehicle access from Moorebank Avenue.
- The Rail link – located within the Rail Corridor, including a connection to the intermodal terminal facility, traversing of Moorebank Avenue, Anzac Creek and Georges River and connection to the SSFL.
- Ancillary works- vegetation clearing, remediation, earth works, utilities installation/connection, signage and landscaping.

2.4 Site Description

The SIMTA Project, including the Stage 1 site, is located approximately 27 kilometres south-west of the Sydney Central Business District (CBD) and approximately 26 kilometres west of Port Botany. The SIMTA Project is situated within the Liverpool Local Government Area (LGA), in Sydney's South West Sub-Region, approximately 2.5 kilometres from the Liverpool City Centre.

The SIMTA site is located approximately 800 metres south of the intersection of Moorebank Avenue and the M5 Motorway. The M5 Motorway provides the main road link between the SIMTA site and the key employment and industrial areas within the West and South Western Sydney Sub-Regions. The M5 Motorway connects with the M7 Motorway to the west, providing access to the Greater Sydney Metropolitan Region and NSW road network. Similarly the M5 Motorway is the principal connection to Sydney's north and north-east via the Hume Highway.

The Southern Sydney Freight Line (SSFL) is located one kilometre to the west of the proposed SIMTA site. The SSFL is a 36 kilometre dedicated freight line between Macarthur and Chullora.

The SIMTA site was recently operating as the Defence National Storage and Distribution Centre (DNSDC) however Defence has recently relocated this operation and vacated the SIMTA site. The majority of land immediately surrounding the SIMTA site is owned and operated by the Commonwealth and comprises:

- School of Military Engineering (SME), on the western side of Moorebank Avenue directly adjacent to the SIMTA site.
- Holsworthy Military Reserve, to the south of the site on the southern side of the East Hills Passenger Railway Line.
- Commonwealth Residual Land, to the east between the SIMTA site and the Wattle Grove residential area.
- Defence National Storage and Distribution Centre (DNSDC), to the north and north east of the SIMTA site.

The site immediately west of the SIMTA site which currently includes the SME is the subject of a Development Application (DA) (SSD-5066), under Part 4, Division 4.1 of the EP&A Act, for the development of an intermodal facility known as the Moorebank Intermodal Terminal Project (MIC Proposal). The EIS for the MIC Proposal has recently been prepared and publically exhibited on 8

October 2014 to 8 December 2014. A Preferred Project Report (PPR) is currently under preparation to respond to submissions received during public exhibition. The MIC Proposal has yet to be determined by the Department of Planning and Environment (DP&E).

A number of residential suburbs are located in proximity to the Stage 1 site, including:

- Wattle Grove, located approximately 600 metres from the Stage 1 site and 750 metres from the Rail link to the east.
- Moorebank, located approximately 1,700 metres from the Stage 1 site and more than 2,700 metres from the Rail link to the north.
- Casula, located approximately 1,100 metres from the Stage 1 site and 250 metres from the Rail link to the west.
- Glenfield, located over 1,700 metres from the Stage 1 site and 750 metres from the Rail link to the south-west.

3 METHODOLOGY

The Environmental Health Standing Committee (EnHealth), a standing committee of the Australian Health Protection Principal Committee[a], has developed a framework to conduct HIAs in Australia - *Health Impact Assessment Guidelines 2001* (HIA Framework). Under this framework a HIA needs to consider the issues that are important to the community in terms of potential impacts on the health of the affected population which go beyond direct impacts on health and include the broader social determinants that can affect people's health and wellbeing. The Centre for Health Equity Training, Research and Evaluation[b] (CHETRE) has developed documentation (*Health Impact Assessment – A Practical Guide, 2007*) (CHETRE Guidelines) that provides guidance in how to apply the enHealth HIA framework in practice. These two documents guide the conduct of HIAs in Australia and are adopted by regulatory agencies for this purpose.

As part of a HIA, a health risk assessment (HRA) is usually conducted. The HRA aims to quantify the impacts of hazards, in this case environmental hazards, on human health. The Australian guidance for conducting HRAs is set out in the enHealth *Guidelines for Health Risk Assessment*, 2012 (HRA Guidelines). For the assessment of health risks from air pollution the National Health and Medical Research Council (NHMRC) *Approach to Hazard Assessment for Air Quality,* 2006 and the National Environment Protection Council (NEPC) *Methodology for Setting Air Quality Standards in Australia,* 2011 provide detailed frameworks to assess health risks primarily via the inhalation pathway.

The key steps in the HIA process are:

- Conduct screening assessment to determine if HIA is required
- Community consultation to scope HIA
- Identify Key issues
- Develop scope of HIA
- Identification of potential health impacts
- Assessment
- Decision making and recommendations

[a] The Committee has the responsibility of providing agreed environmental health policy advice and implementing the National Environmental Health Strategy 2012-2015.

[b] Part of the University of New South Wales' Centre for Primary Health Care and Equity and also NSW Population Health.

- Evaluation and follow-up

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

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

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

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

Risk assessments are often conducted by considering possible or theoretical community exposures predicted from air dispersion modelling or using environmental concentrations that have been measured in the potentially affected population. Conservative safety margins are built into a risk assessment analysis to ensure protection of the public. During the risk assessment analysis the most vulnerable people (e.g. children, the sick and elderly) are carefully considered to make sure that all members of the public will be protected.

The risk assessment helps answer common questions for people who might be exposed to hazardous pollutants in the environment. The HRA is a useful tool for estimating the likelihood and severity of risks to human health, safety and the environment and for informing decisions about how to manage those risks.

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

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

1. **Issue Identification** – Identifies issues that can be assessed through a risk assessment and assists in establishing a context for the risk assessment.
2. **Hazard Assessment** – Identifies hazards and health endpoints associated with exposure to hazardous agents and provides a review of the current understanding of the toxicity and risk relationship of the exposure of humans to the hazards.

3. **Exposure Assessment** – This task identifies the groups of people who may be exposed to hazardous agents and quantifies the exposure concentrations.
4. **Risk Characterisation** – This task provides the qualitative evaluation of potential risks to human health. The characterisation of risk is based on the review of concentration response relationship and the assessment of the magnitude of exposure.
5. **Uncertainty Assessment** – identifies potential sources of uncertainty and qualitative discussion of the magnitude of uncertainty and expected effects on risk estimates.

For the Stage 1 Proposal a screening HIA has been conducted which incorporates a HRA for air quality and noise. The screening HIA builds on the consultation conducted as part of the Concept Plan Approval (Elton Consulting, 2015) as community consultation for the Stage 1 Proposal is expected to be conducted during public exhibition of the Stage 1 Proposal EIS. The potential social impacts of the Proposal have been drawn from the Social Commentary conducted for the Concept Plan Approval (Urbis, 2012).

4 RESULTS

4.1 Issues Raised through Consultation

A consultative process was conducted as part of the approvals process for the Concept Plan Approval of the SIMTA Project and the outcomes have been summarised (Elton Consulting, 2015). This report provides a high level summary of the issues raised through the consultative process. As part of the screening HIA a review of the submissions received during the public exhibition phase for the Concept Approval has been undertaken. These submissions are available on the NSW Planning Major Projects website (http://majorprojects.planning.nsw.gov.au/page/development-categories/transport--communications--energy---water/rail---related-facilities/?action=view_job&job_id=4400).

The key issues raised through the consultative process in relation to health included:

1. The health effects associated with exposure to PM_{10}, $PM_{2.5}$, SO_2 and NO_2 associated with the activities at the site
2. Health effects of diesel pollution and reduced air quality
3. Impact of trucks queuing and idling
4. Cumulative impacts on air quality, noise and traffic from the two adjacent intermodal sites
5. Impacts of noise including the noise generated by the SSFL
6. The impacts of the proposal on the residents of Casula and Wattle Grove as well as Moorebank
7. The impact of the SIMTA site on regional air quality
8. Health effects of air toxics emitted from the site
9. Growth in heavy vehicle traffic in the area and the impact on air quality
10. Dust emissions from the construction activities on the site
11. The impact of noise on sleep disturbance
12. Existing air pollution
13. Effects of air pollutants on people with asthma
14. Cancer risk from diesel and other air toxics
15. Effect of rail noise on sleep disturbance for people within 400m of the Rail link

The screening HIA and incorporated HRA have drawn on information presented in the air quality (Environ, 2015) and noise and vibration assessments (Wilkinson-Murray, 2015) conducted for the Stage

1 Proposal. Based on information from these reports the issues raised through the consultative process have been addressed where possible.

4.2 Health and Population Profile

The Proposal site is located within the Liverpool LGA and is surrounded by the suburbs of Casula, Moorebank, Glenfield and Wattle Grove. The Liverpool LGA is located in the south west of Sydney and encompasses a total land area of 305 square kilometres. Its boundaries include Fairfield and Penrith cities in the north, Camden Council and Campbelltown City areas in the south, the Wollondilly Shire in the south and Bankstown City in the east.

According the Liverpool Community Health Profile (SWSLHD, 2014) the population in the Liverpool LGA is predicted to increase significantly from 188,088 people in 2011 to 288,959 in 2031. The predicted population growth in various age groups is shown in Figure 2 (taken from Liverpool Community Health Profile, SWSLHD, 2014).

Figure 2: Predicted Population Growth Liverpool LGA

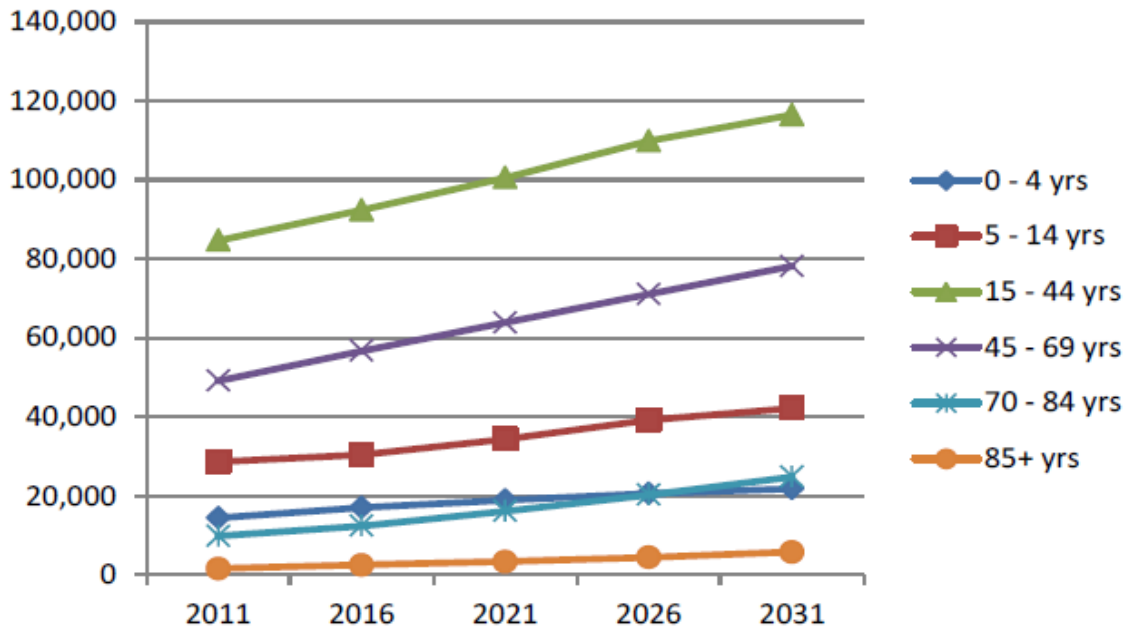


Figure 2 shows that the most significant population growth is predicted for people less than 69 years of age.

Population statistics for 2011 have been obtained from ABS for the each of the suburbs in close proximity to the SIMTA Project and which have been considered in the health risk assessment. The population at the 2011 Census in the study area was 30,164 people. Of this 8% were less than 5 years of age and 10% were 65 years or older. The median age of the population is 33 years of age. This is younger than the NSW average of 38 years of age. The population statistics for Casula, Wattle Grove, Moorebank and Glenfield are shown in Table 3.

Table 3: Population Profile for Project Area

	Casula		Wattle Grove		Moorebank		Glenfield	
	Number of people	% of population	Number of people	% of population	Number of people	% of population	Number of People	% of population
< 5 years	1168	7.9	714	8.7	641	8.4	500	6.6
<15 years	3341	22.7	2154	26.3	1641	21.6	1424	18.8
15 to 64 years	9835	66.9	5610	68.5	4982	65.6	6008	79.5
65+	1520	10.3	428	5.2	972	12.8	1049	13.8

People who are of low socioeconomic status (SES) have been identified as a vulnerable group for the effects of air pollution. There are several indices of social deprivation used to assess SES status in Australia. One commonly used is the SEIFA index. The Socio-Economic Indexes for Areas (SEIFA) index is a measure of relative social disadvantage and takes into account 20 variables to assess relative social disadvantage. The lower the SEIFA index the greater the level of disadvantage.

The SEIFA Index of Relative Socio-Economic Advantage/Disadvantage is derived from attributes such as low income, low educational attainment, high unemployment, jobs in relatively unskilled occupations and variables that broadly reflect disadvantage rather than measure specific aspects of disadvantage (e.g. Indigenous and Separated/Divorced). At the advantage end of the scale, households with high incomes, high education levels, large dwellings, high numbers of motor vehicles, spare bedrooms and professional occupations contribute to a higher score.

High scores on the Index of Relative Socio-Economic Advantage/Disadvantage occur when the area has many families with large houses, high incomes, low unemployment rates and professional occupations. Low scores on the index occur when the area has many low income families and people with little training and in unskilled occupations, as well as children in households with jobless parents. Unlike the index of disadvantage, a high score on this index indicates both a lack of disadvantage and a high level of advantage, based on these attributes. The index is relative to a score of 1000 which is considered as the Australian average.

The SEIFA index for Relative Advantage and Disadvantage for the Casula, Moorebank, Wattle Grove and Glenfield are shown in Table 4 compared to Liverpool City, Sydney, NSW and Australia.

Table 4: SEIFA Index for Relative Advantage and Disadvantage

	Australia	NSW	Greater Sydney	Liverpool City	Casula	Moorebank	Wattle Grove	Glenfield
SEIFA INDEX	1002	1001	1025	968	998	1022	1089	1001

The data in Table 4 show that both Moorebank and Wattle Grove have a higher level of socio-economic advantage than the NSW and Australian average as well as the average for Liverpool City and Casula. Casula has a slightly higher level of disadvantage that the NSW average. Glenfield is consistent with the NSW average.

Casula, Moorebank and Wattle Grove have high levels of employment with 93%, 95.7% and 96.9% of the population in full or part time employment. This level of employment is similar to the Sydney average of 94.3%. No information was available for Glenfield. The three top professions are:

1. Clerical and administration
2. Professional
3. Technical and trade services

The average life expectancy in Liverpool City is 79.5 years for males and 83.4 years for females. This compares to the NSW life expectancy of 79.6 years for males and 84.4 years for females.

The Liverpool Community Health Profile (SWSLHD, 2014) presents the baseline health statistics for the local population. These are shown in Table 5 (taken from SWSLHD, 2014)

Table 5: Baseline Health Status Liverpool LGA

Indicator	Liverpool	NSW
Hospitalisations		
Hospitalisations 2009-10 to 2010-11, smoothed number of separations per year (smoothed estimate of Standardised Separation Ratio – seSSR)[10]	58,010 (99.9)	2,645,561 (100)
Potentially preventable hospitalisations 2010-11 2011-12 smoothed number of hospitalisations per year (seSSR)	3,850 (95.4)	183,951 (100)
Alcohol attributable hospitalisations, 2010-11 to 2011-12, smoothed number of hospitalisations per year, (seSSR)	934 (81.8)	49,410 (100)
Smoking attributable hospitalisations, 2010-11 to 2011-12, smoothed number of hospitalisations per year (seSSR)	905 (100.5)	45,069 (100)
High body mass index attributable hospitalisations, smoothed number of separations per year 2010-11 to 2011-12 (seSSR)	719 (101)	35,952 (100)
Coronary heart disease hospitalisations 2009-10 to 2010-11 smoothed number of hospitalisations per year (seSSR)	821 (91.2)	49,935 (100)
COPD Hospitalisations, persons aged over 65, 2009-10 to 2010-11, smoothed number of separations per year (seSSR)	262 (112.9)	15,037 (100)
Diabetes hospitalisations, 2009-10 to 2010-11, smoothed number of separations per year (seSSR)	515 (132.1)	19,193 (100)
Fall-related injury overnight hospitalisations, persons aged 65 years and over, 2010-2011 to 2011-2012 combined, smoothed number of hospitalisations per year (seSSR)	572 (116.9)	34,766 (100)
Stroke hospitalisations 2010-11 to 2011-12, smoothed number of hospitalisations per year (seSSR)	196 (97.6)	11,853 (100)
Deaths		
Potentially avoidable deaths, persons aged under 75 years, 2006 to 2007 combined, smoothed number of deaths per year, smoothed Standardised Mortality Ratio (sSMR) [11]	211 (99.5)	10,356 (100)
Potentially avoidable deaths from preventable causes, persons aged under 75 years, 2006 to 2007 combined (sSMR)	122 (96.6)	6,133 (100)
Potentially avoidable deaths from causes amenable to health care, persons aged under 75 years, 2006 to 2007 combined, smoothed number of deaths per year (sSMR)	84 (97.8)	4,235 (100)
High body mass index attributable deaths by LGA 2006 2007, (sSMR)	46 (91.1)	2.926 (100)
Alcohol attributable deaths, smoothed estimate of standardised mortality ratios per year, 2006 – 2007 (sSMR)	23 (94.6)	1,238(100)
Smoking attributable deaths, smoothed estimate of standardised mortality ratios per year, 2006 – 2007 (sSMR)	79 (99.2)	5,195 (100)

Source: Centre for Epidemiology and Evidence. Health Statistics New South Wales. NSW Ministry of Health http://www.healthstats.nsw.gov.au/

The data shown in Table 5 indicate that the baseline health status of the local population in the study area, which includes Casula, Moorebank, Wattle Grove and Glenfield, does not differ significantly from the data for NSW as a whole.

According to the Liverpool Community Health Profile (SWSLHD) the asthma prevalence rate in people over 16 years of age in the area is 6.3%. This is lower than the NSW average for the same age group. Throughout the consultation conducted for the Concept Plan Approval, the issue of higher asthma rates in the Liverpool area was raised. The key concern was that the community was already more vulnerable to the effects of air pollution due to existing asthma prevalence. The information provided in the Liverpool Community Health Profile indicates that the asthma prevalence is no different to the rest of NSW.

The Social Impact Commentary prepared for the Concept Plan Approval (Urbis, 2012) found that:

- The local environment is characterised by a high level of labour market regionalisation with a low job to resident ratio exacerbated by rapid growth of the local labour market, which has grown faster than the overall population.

- There is significant population and employment growth anticipated in the LGA. Growth will occur across a number of areas and industries. Liverpool is assigned the highest employment growth target for the region. Residential growth is anticipated to occur in Moorebank in the form of infill development.

- There has been a declining local employment base with a fall in the ratio of local jobs to local workers. This is forecast to change over the next 10 years facilitated by the North West and West Central Sydney Employment Strategies.

- There is an existing strong employment base in logistics, with Western Sydney well represented in the manufacturing, construction, retail, transport and warehousing. Many employment opportunities at the fringe have tended to occur in low skilled areas, including manufacturing, wholesaling, transport and construction, with fewer opportunities in more skilled employment sectors.

Local planning documents identified a number of priorities that were relevant to SIMTA Project including:

- Reduce unemployment particularly for young people and those experiencing long term unemployment.

- Support initiatives that improve employment outcomes for Aboriginal people.

- Support economic development and access to local employment opportunities.

- Support development of Liverpool City Centre into a regional City with accessible services and employment opportunities.

- Support affordable goods and services.

The Social Impact Commentary concluded that there are significant local employment opportunities that will be associated with both the construction and operation of the Stage 1 Proposal and that this had the potential to provide a significant social benefit to the region considering the regional demographic and policy context including.

- Reduced travel distance and commuting time for local potential employees.

- New jobs created in construction, operation, maintenance, logistics and transport.

- The types of employment that will include a range of skilled and unskilled labour needs.

- Potential opportunities for some of the youth or long term unemployed people in the region.

- Potential opportunities to support and improve employment outcomes for Aboriginal people.

The report presented a number of recommendations for consideration, including:

- Further consideration of landscaping design in relation to minimising visual impact and light spill and enhancing the local environment.

- Consideration of the development of a vehicle efficiency and emissions reduction program for the facility to encourage good maintenance and efficient vehicle selection.

- Ensure effective noise management and complaints reporting procedure is set up for the construction and operational phases of the development.

The Social Impact Commentary noted that concerns had been raised about the potential health effects of air pollution, in particular diesel emissions, noise impacts and traffic issues however the authors believed that the mitigation and management measures proposed in the Specialist Technical Documents, accompanying the Concept Plan Approval, would adequately manage any potential impacts.

4.3 Summary

A review of the demographics and health status of the community in proximity to the Proposal site has shown that the overall health status of the population does not differ from that of the rest of NSW. The population has a high level of employment and generally has a higher level of social advantage (as shown by the SEIFA index) than the NSW average. The findings of the Social Impact Commentary conducted for the Concept Plan Approval concluded that the development of the SIMTA Project would provide employment opportunities for the local community. The Social Impact Commentary also concluded that development of the Proposal would provide opportunities to improve community services and economic benefits in the form of employment and local and regional productivity.

4.4 Health Risk Assessment – Air Quality

The air quality health risk assessment for the Stage 1 Proposal has been conducted to address the issues raised through the consultative process. The health effects attributable to PM_{10}, $PM_{2.5}$, NO_2, SO_2 and CO have been assessed for increases in mortality, hospital admissions for respiratory and cardiovascular disease, and emergency department visits for asthma in children. Baseline health statistics for Sydney have been used in the assessment and the risk has been assessed for Casula, Wattle Grove, Glenfield and Moorebank. The cancer risk due to diesel emissions, benzene, 1,3-butadiene and PAHs has also been calculated for the local area. No quantitative assessment has been undertaken for regional changes to air quality as the data was not available from the Air Quality Assessment (Environ, 2015). Environ concluded in this report that any changes to regional air quality would be negligible.

The air quality data used in the HRA has been generated as part of the Air Quality Assessment (Environ, 2015) conducted for the Stage 1 Proposal. The modelling results that have been used in the HRA represent the worst case emission scenarios for the Stage 1 Proposal operations. The modelling has included the use of reach stackers for container handling which are operated by diesel engines. One option identified through the best practice review for air quality is to move the container handling to electrified gantry crane systems which would eliminate the majority of the diesel emissions from the container handling equipment (Environ, 2015). This would lead to a significant reduction in PM_{10}, $PM_{2.5}$ and NO_2 arising from operations of the Stage 1 site.

The risk assessment for each of the pollutants is presented in the following sections and is presented following the enHealth guidelines for conducting HRAs.

4.4.1 PM_{10} and $PM_{2.5}$

According to the Air Quality Assessment (Environ, 2015) conducted for the Stage 1 Proposal the main sources of PM_{10} and $PM_{2.5}$ are locomotives (travelling and idling), trucks (travelling and idling) and container handling, when using mobile container handlers.

4.4.1.1 Literature Review of the Health Effects of PM_{10} and $PM_{2.5}$

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

Several studies conducted in Australia also show adverse effects of both PM_{10} and $PM_{2.5}$ on mortality and morbidity outcomes (Simpson et al., 2005a, b; Barnett et al., 2005; 2006) similar to those observed in overseas studies. The effects observed in the Australian studies appear to be higher than those observed in the US and Europe but comparable to the results of Canadian studies.

A recent review conducted by the World Health Organization (WHO), (2013) concluded that both PM_{10} and $PM_{2.5}$ are related to increases in mortality from respiratory and cardiovascular causes, hospital admissions and emergency department attendances for respiratory and cardiovascular causes including asthma, exacerbation of asthma and increases in respiratory symptoms. In recent years studies have provided much stronger evidence for the cardiovascular effects of particles, in particular $PM_{2.5}$. There has also been increase in evidence to support a biological mechanism the cardiovascular effects

which includes interference with electrical process within the heart, systemic inflammation and oxidative stress. The WHO concluded that long-term exposure to $PM_{2.5}$ is the cause of both cardiovascular mortality and morbidity. The USEPA (2012) concluded that there was new evidence regarding cardiovascular mortality showing strong effects between $PM_{2.5}$ exposure and cardiovascular mortality especially in women. In addition there is evidence for long-term exposure to $PM_{2.5}$ and respiratory effects including the incidence of lung cancer. Studies of cardiovascular effects provided evidence of an association between long-term exposure $PM_{2.5}$ and myocardial infarction (heart attack), hypertension, diabetes and stroke especially among women.

A number of new studies linking long-term exposures have examined additional health outcomes apart from the respiratory and cardiovascular outcomes. These outcomes include atherosclerosis, adverse birth outcomes and childhood respiratory disease. Studies have also shown possible links between long-term exposure to $PM_{2.5}$ and neurodevelopment and cognitive function as well as other chronic conditions such as diabetes. In recent years the evidence for a link between exposure to particles and diabetes has been strengthened.

Birth cohort studies from Europe and elsewhere have found associations between $PM_{2.5}$ and respiratory infections and asthma in young children. Reduced lung function is also linked to $PM_{2.5}$ exposure. Findings of cohort study conducted in the Netherlands supports the findings of previous studies conducted in the US and Europe linking exposure to particles and these health outcomes. Associations with birth outcomes such as low-birth-weight, preterm birth and small gestation age at birth have also been found with long-term exposure to $PM_{2.5}$. These outcomes may affect a child's development later in life. The USEPA (2012) also identified several recent studies that showed associations between long-term exposure to $PM_{2.5}$ and respiratory morbidity including hospital admissions and respiratory symptoms as well is the incidence of asthma. Studies of reproductive and developmental effects also provided evidence for long-term exposure to $PM_{2.5}$ and reduced birth weight.

With respect to short-term effects the USEPA (2012) found that were significant new studies that increase the evidence for an association between $PM_{2.5}$ and mortality and morbidity outcomes and strengthen the previous US EPA conclusion that there is a causal association between short-term exposure to $PM_{2.5}$ and these outcomes. Associations were found for hospital admissions and emergency department attendances for all cardiovascular and respiratory causes as well is cause specific outcomes, in particular asthma.

The Australian Child Health and Air Pollution Study (ACHAPS) study, which used a similar study design as that used in the Southern Californian Children's Health Study, was conducted to inform the review of the particle standards in the Ambient Air Quality NEPM (SCEW, 2011). The results of a cross-sectional study of approximately 4,000 Australian school children aged 7-11 years showed varied results for the particulate matter exposures used in ACHAPS. PM_{10} was associated with decline in lung function (FEV_1) post-bronchodilator use and increase in exhaled NO (an indicator of airway inflammation), but no overall increase in current respiratory symptoms. $PM_{2.5}$ was associated with an adverse effect on lung function (measured as Forced Vital capacity, FVC) post-bronchodilator use and on exhaled NO, with no overall effects on current symptoms, but showed increased risk of lifetime wheezing, asthma, and asthma medication use, and current asthma, use of beta-agonists and itchy rash in non-atopic children. Females had an increase in FEV_1/FVC ratio pre-bronchodilator for recent $PM_{2.5}$, and recent PM_{10} exposures, with non-significant effects in males. Despite the absence of effect on current symptoms, a reduction in lung volume at this age may have longer-term adverse consequences if it persists into later life (SCEW, 2011).

No studies investigating the long term effects of exposure to PM_{10} on health have been conducted in Australia, however there have been several international studies that have shown strong associations between long-term exposure to PM_{10} and increases in mortality.

4.4.1.2 Exposure Assessment

Annual average and 24-hour PM_{10} and $PM_{2.5}$ have been modelled as part of the air quality assessment (Environ, 2015) for the Proposal. Figures 3 and 4 show the 24 hour averages for the most affected receptors in the surrounding suburbs area. The air quality standards for PM_{10} and $PM_{2.5}$ contained in the Ambient Air Quality NEPM are 50µg/m^3 and 25µg/m^3 respectively. The data shown in Figures 3 and 4 are well below these standards.

The PM_{10} and $PM_{2.5}$ data generated in the Air Quality Assessment (Environ, 2015) has been used to calculate the risk of adverse health outcomes associated with exposure to PM_{10} and $PM_{2.5}$ from the operation of the Stage 1 Proposal.

Figure 3: Daily average PM_{10} from Stage 1 operations

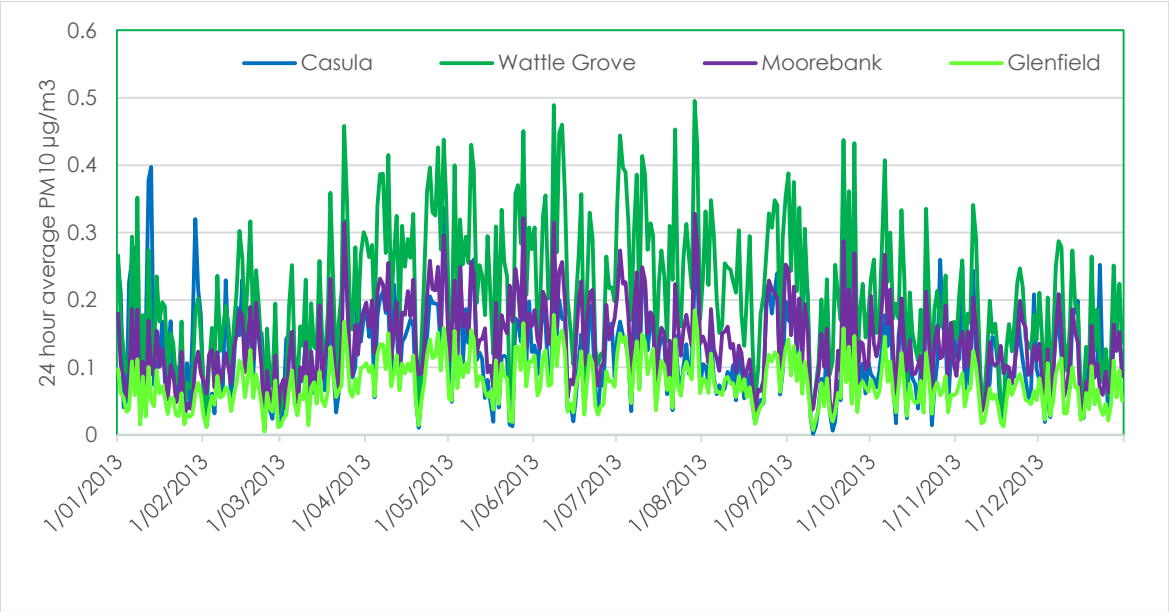
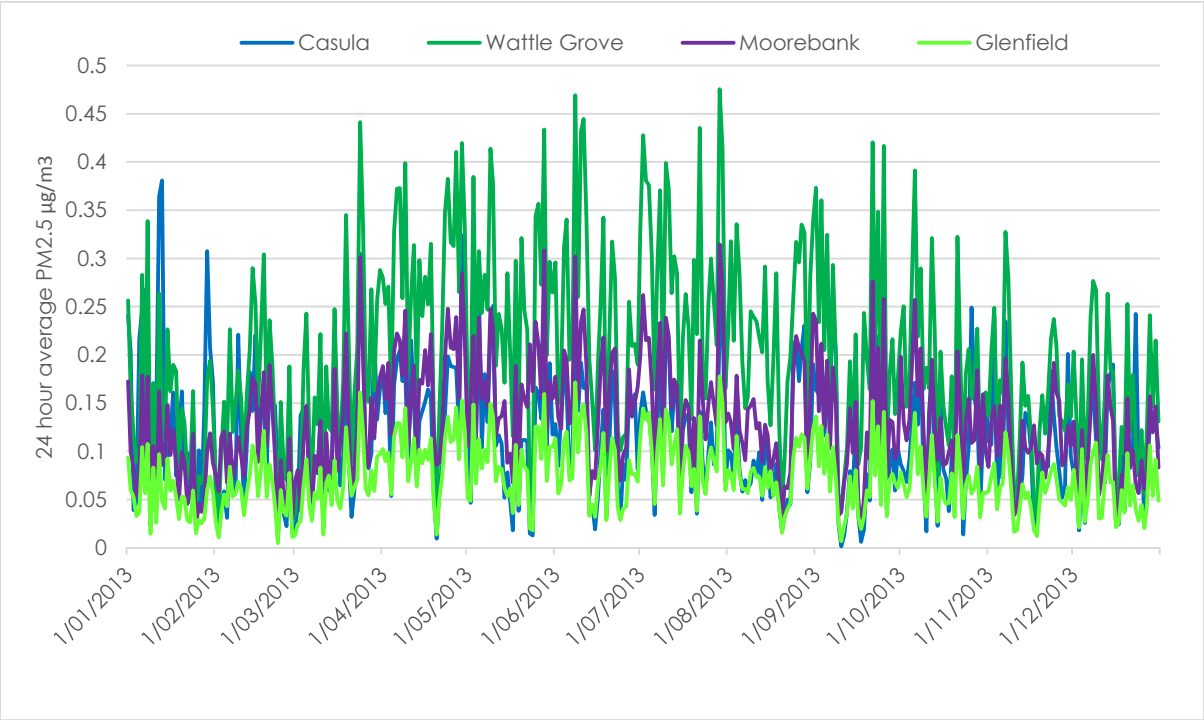


Figure 4: Daily average $PM_{2.5}$ from Stage 1operations



The data shown in Figures 3 and 4 shows that the predicted levels are similar at Casula and Moorebank but slightly higher at the Wattle Grove site. The predicted values at Glenfield are lower than at the other sites. All predicted concentrations are well below the current Air Quality NEPM standards.

4.4.1.3 Risk Characterisation

The results of epidemiological studies have shown that a wide range of health effects are associated with exposure to PM_{10}. Australian studies (NEPC, 2012; EPHC 2006) have found associations between PM_{10} levels currently experienced in Australian cities and the following health outcomes:

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

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

Although no studies investigating the long term effects of exposure to PM_{10} on health have been conducted in Australia, there have been several international studies that have shown strong associations between long-term exposure to PM_{10} and increases in mortality. On the basis of the findings of these studies of long-term mortality has also been assessed.

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

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

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

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

To calculate the number of people that might be impacted by air pollution exposure-response functions for each outcome being assessed are required. These functions are a measure of the change in the health outcome within the population for a given change in PM_{10} or $PM_{2.5}$ concentration.

The exposure-response functions in

Table 6 and Table 7 have been taken from Australian studies and in particular two multicity meta-analyses (Simpson et al., 2005; EPHC, 2011). The use of Australian meta-analyses is consistent with the NHMRC (2006) and NEPC (2011) recommendations for selecting exposure response functions for risk assessments for air pollution.

The exposure-response functions for long-term exposure to PM_{10} and $PM_{2.5}$ have been taken from the American Cancer Society study (HEI, 2009). This study is considered by the WHO as the most reliable study to assess long-term effects of air pollution. The use of this value is also consistent with the recommendations made by NHMRC (2006) and NEPC (2011).

Table 6: Exposure Response Functions for PM_{10} Selected Health Outcomes (Taken from EPHC, 2011; HEI, 2009)

Outcome	Averaging Period	Exposure Response Function per 1 µg/m³ increase in PM_{10}
Annual all-cause mortality (non-accidental) 30+ years	Annual Average	0.004
Daily all-cause mortality(non-accidental) all ages	24 hours	0.002
Daily mortality cardiovascular disease - all ages	24 hours	0.002
Hospital Admissions respiratory disease 65+ years	24 hours	0.003
Hospital Admissions cardiac disease 65+ years	24 hours	0.002
Hospital Admissions pneumonia and bronchitis 65+ years	24 hours	0.0013
Hospital Admissions respiratory disease 15-64 years	24 hours	0.003
ED Visits Asthma 1-14 years	24 hours	0.015

Table 7 shows the exposure response functions used for $PM_{2.5}$.

Table 7: Exposure Response Functions for $PM_{2.5}$ Selected Health Outcomes Taken from EPHC, 2011; HEI, 2009)

Outcome	Averaging Period	Exposure Response Function per 1 µg/m³ increase in $PM_{2.5}$
Annual all-cause mortality (non-accidental) 30+ years	Annual Average	0.006
Annual cardiopulmonary mortality 30+	Annual average	0.014
Annual mortality ischemic heart disease 30+ years	Annual average	0.024
Annual mortality lung cancer 30+ years	Annual average	0.014
Daily all-cause mortality(non-accidental) all ages	24 hours	0.0023
Daily mortality cardiovascular disease - all ages	24 hours	0.0013
Hospital Admissions respiratory disease 65+ years	24 hours	0.004
Hospital Admissions cardiac disease 65+ years	24 hours	0.005
Hospital Admissions cardiovascular disease 65+ years	24 hours	0.003
Hospital Admissions ischemic heart disease 65+ years	24 hours	0.004

Hospital Admissions COPD 65+ years	24 hours	0.004
Hospital Admissions pneumonia and bronchitis 65+ years	24 hours	0.005
Hospital Admissions respiratory disease 15-64 years	24 hours	0.003
ED Visits Asthma 1-14 years	24 hours	0.0015

Using the predicted annual average and 24 hour average PM_{10} and $PM_{2.5}$ concentrations for the most affected receptors from the air dispersion modelling done as part of the air quality assessment (Environ, 2015)and the exposure response function in Tables 6 and 7, the health effects attributable to PM_{10} and $PM_{2.5}$ have been calculated using the following equation:

Number of attributable cases = exposure response function (Change in health outcome) per 1µg/m^3 increase in PM x PM concentration x baseline health incidence rate/ 100,000 population x actual population

The baseline health statistics for Sydney were used in this assessment. The number of attributable cases are shown in Table 8 (PM_{10}) and Table 9 ($PM_{2.5}$). The number of attributable cases is the increase in the number, for example hospital admissions for respiratory disease that may arise from exposure to PM from the SIMTA operations. If the same example is used, the results in Table 8 show that for Wattle Grove, based on the predicted increase in daily PM_{10} concentrations, that there would be an additional 0.01 hospital admissions per year which is equivalent to 1 additional hospital admission per 100 years, which may be attributed to emissions of PM_{10} from the SIMTA Stage 1 operations. This equates with an increase in risk of approximately 2 in 100,000 based on a worst case scenario that no mitigation measures have been implemented. The move from diesel powered reach stackers for container handling to electrified gantry cranes will reduce the PM emissions by approximately 45% with further reductions obtained by implementation of other best practice measures identified in the Air Quality Assessment (Environ, 2015). The resulting risk is within internationally acceptable limits of between 1 in a million and 1 in 100,000.

Table 8: Attributable health outcomes due to PM_{10} from the Stage 1 Proposal

Health Outcome	Casula	Wattle Grove	Moorebank	Glenfield
Annual Mortality 30+ years	0.04	0.03	0.02	0.02
Daily Mortality all causes all ages	0.02	0.02	0.01	0.007
Daily Mortality Cardiovascular Disease all ages	0.007	0.008	0.004	0.002
Hospital Admissions Respiratory Disease 65+	0.02	0.01	0.02	0.009

Hospital Admissions Cardiac Disease 65+	0.02	0.01	0.02	0.01
Hospital Admissions Pneumonia and Bronchitis 65+	0.002	0.001	0.002	0.001
Hospital Admissions Respiratory Disease 15-64 years	0.03	0.03	0.02	0.01
Emergency Department Visits 0-14 years	0.007	0.008	0.004	0.002

As can be seen from Table 8 the number of attributable cases from PM_{10} from the Stage 1site are low. The highest risk is for all-cause mortality from long-term exposures with between 2 and 4 additional deaths per 100 years that are attributable to PM_{10} from the Proposal. All other risks are lower than that predicted for long-term mortality. All risks are very low and the increase is unlikely to be observed within the community. . These risk levels are within the acceptable risk levels established by international agencies.

These calculations are also based on the assumption that the whole population of each suburb is exposed to the highest predicted concentration which is not the case. The predicted concentrations decrease with distance from the Proposal. The assumption that the whole population is exposed to the highest concentration will overestimate the risk. The PM_{10} data that was provided for the HRA did not include implementation of the best practice measures outlined in the Air Quality Assessment Report (Environ, 2015). According to the best practice review undertaken for the Air Quality Assessment, a 45% reduction in PM_{10} and $PM_{2.5}$ levels would be achieved by implementation of the measures such as moving from diesel powered stack reachers for container handling to electrified gantries. This reduction in PM levels would translate to a similar reduction in health risk.

The results for $PM_{2.5}$ are shown in Table 9:

Table 9: Attributable health outcomes due to $PM_{2.5}$ from the Stage 1 Proposal

OUTCOME	Casula	Wattle Grove	Moorebank	Glenfield
All-cause mortality 30+years (long-term)	0.05	0.05	0.03	0.02
Cardiopulmonary mortality 30+years (long-term)	0.05	0.05	0.03	0.02
Ischemic Heart Disease 30+ years (long-term)	0.03	0.03	0.02	0.01
Lung cancer mortality 30+ years (long-term)	0.008	0.007	0.005	0.003
Daily mortality all causes all ages	0.02	0.02	0.01	0.007
Daily mortality cardiovascular disease all ages	0.004	0.004	0.002	0.001
Hospital admissions respiratory disease 65+ years	0.03	0.01	0.02	0.01
Hospital admissions cardiac disease 65+ years	0.05	0.03	0.04	0.02
Hospital admissions pneumonia and bronchitis 65+ years	0.009	0.005	0.007	0.004
Hospital admissions cardiovascular disease 65+ years	0.05	0.02	0.03	0.02
Hospital admissions respiratory disease 15-64 years	0.03	0.03	0.02	0.01
Emergency Department visits asthma 1-14 years	0.007	0.007	0.004	0.002

As with PM_{10} the number of attributable cases from $PM_{2.5}$ from the Stage 1 Proposal are low. The highest risk is for all-cause mortality and cardiopulmonary mortality from long-term exposures with 5 additional deaths per 100 years that are attributable to $PM_{2.5}$ from the Proposal. There are 5 additional hospital admissions for cardiac and cardiovascular disease in people over 65 years of age per 100 years predicted. All other risks are lower than that predicted for these outcomes. All risks are very low and the increase is unlikely to be observed within the community. The risks translate to between a 1 in a million and 1 in 100,000 increase in health outcome which is within the generally accepted risk levels internationally.

These calculations, as with PM_{10}, are based on the assumption that the whole population of each suburb is exposed to the highest predicted concentration which is not the case and that the emissions are

unmitigated. The predicted concentrations decrease with distance from the Proposal. The assumption that the whole population is exposed to the highest concentration will overestimate the risk. As with PM_{10} implementation of best practice measures will lead to a reduction in risk.

The impact of PM_{10} and $PM_{2.5}$ emissions on regional air quality were not assessed quantitatively in the Air Quality Assessment (Environ, 2015). Therefore a quantitative assessment of risk cannot be undertaken. The Air Quality Assessment of regional impacts was based on previous work done on the emissions from road vehicles and railway locomotives associated with the SIMTA proposal on the Port Botany-Moorebank Corridor (Pacific Environment, 2013). This report used changes in total emissions to estimate the impact on regional air quality and found that the changes in emissions would be negligible when considered at a regional level. On this basis Environ (2015), concluded that the impacts on regional air quality would also be negligible. If there is no increase in emissions that would subsequently lead to an increase in regional air pollution associated with the SIMTA proposal, then it can be concluded that there will be no associated increase in health risk.

4.4.2 Nitrogen Dioxide (NO_2)

According to the Air Quality Assessment (Environ, 2015) conducted for the Stage 1 Proposal the main sources of NO_2 are locomotives (travelling and idling), trucks (travelling and idling) and container handling. The Air Quality Assessment has assessed NO_2 as being 100% of the NO_x emissions which a very conservative assumption. The modelling has also been undertaken without considering the reduction in emissions that would be achieved by implementing the Best Practice Measures in the Environ (2015) report.

4.4.2.1 Literature Review of the Health Effects of NO_2

In recent years there has been an increased interest in the health effects of nitrogen dioxide. The REVIHHAP study (WHO, 2013) investigated the new studies of both long-term and short-term exposure to NO_2 and associations with mortality, hospital admissions and respiratory symptoms and concluded that these new studies show that short-term exposure to NO_2 is associated in increases in these outcomes. Studies of the long-term effects of exposure to NO_2 have shown associations with both mortality and morbidity outcomes. The effects that have been observed for both long-term and short-term exposure are occurring below current WHO air quality guidelines for NO_2 which are lower than the current NEPM standards. Controlled human exposure and toxicological studies provide support for biological mechanisms for the effects that are observed in epidemiological studies and provide evidence for a causal relationship between exposure to NO_2 and these outcomes. The most recent studies have provided evidence that NO_2 has an independent effect from other pollutants. Epidemiological studies of long-term effects of NO_2 exposure on mortality (both respiratory and cardiovascular causes) and with children's respiratory symptoms and lung function also support the conclusion that NO_2 has an independent effect on health.

Controlled human exposure studies show increased inflammation of the airways and airway hyperresponsiveness at nitrogen dioxide levels down to 0.2 ppm in healthy individuals. As previously mentioned, the general population includes sensitive populations and effects are likely to occur at lower levels of NO_2 than those where adverse effects have been observed in controlled human exposure studies.

Short-term exposure to NO_2 has been linked to increases in all cause, cardiovascular and respiratory mortality. The effects were greater in people 65 years of age and older and for respiratory mortality (WHO, 2013). Epidemiological studies provide no evidence of a threshold for the effect. Recent studies have provided evidence that has strengthened the association with hospital admissions and emergency

department visits for respiratory disease including all respiratory causes, asthma and chronic obstructive pulmonary disease (COPD) (WHO, 2013). Strong associations have been observed for all respiratory causes in people 65 years and older and for children with asthma. The effects are not as strong for cardiovascular causes and in some cases there is no consistent effect observed with cardiovascular effects. There is some evidence for an association with cardiac hospital admissions but these findings are not consistent across studies.

Panel studies of children with asthma show associations between nitrogen dioxide and reductions in lung function, increases in cough, night-time asthma and school absenteeism. There is also an increase in symptoms in asthmatic children and changes in lung function observed, as well as increases in airway inflammation and hyperresponsiveness. Controlled human exposure and animal toxicological studies support the findings of the epidemiological and panel studies.

Long-term exposure to NO_2 has been linked to deficits in lung function growth. These findings have been found in studies in California, Mexico and Sweden. In these studies the effects on nitrogen dioxide were greater than those observed for other pollutants including $PM_{2.5}$. There is also strong evidence of an association between long-term exposure to NO_2 and the incidence of asthma and wheeze. This new evidence suggests that NO_2 exposure may actually cause asthma rather than just exacerbate existing asthma (WHO, 2013; USEPA, 2014). There have also been studies have shown increases in mortality with long-term exposure NO_2 including all-cause, cardiovascular (especially ischaemic heart disease) lung cancer and respiratory mortality. These effects are similar to those observed for $PM_{2.5}$ if not larger and are independent of $PM_{2.5}$.

Australian studies have reported similar associations between hospitalization for respiratory effects, including asthma, and daily NO_2 as overseas studies (Morgan et al. 1998a; Barnett et al. 2005; Erbas et al., 2005; Jalaludin et al. 2004; Rodriguez et al., 2007). In a meta-analysis of results from 5 Australian and 2 New Zealand cities Barnett et al. (2005) analysed hospital admissions for 3 age groups of children. Significant increases in hospital admissions for respiratory disease (1–4, 5–14 years) and asthma (5–14 years) were associated with interquartile range increases in either 1-hr or 24-hr NO_2. The largest association reported was a 6.0% increase in asthma admissions with a 5.1 part per billion (ppb) increase in 24- 30 hr NO_2 and the effect was not reduced by inclusion of PM_{10} in the analysis. A meta-analysis of the associations between pollutants and cardiovascular hospital admissions in the elderly in Brisbane, Canberra, Melbourne, Perth, Sydney, Auckland and Christchurch found significant associations between CO, NO_2, and particles and five categories of cardiovascular disease admissions. The two largest statistically significant increases were for cardiac failure, with a 6.9% increase for a 5.1-ppb unit increase in NO_2 and a 6.0% increase for a 0.9-ppm increase in CO (Barnett et al, 2006).

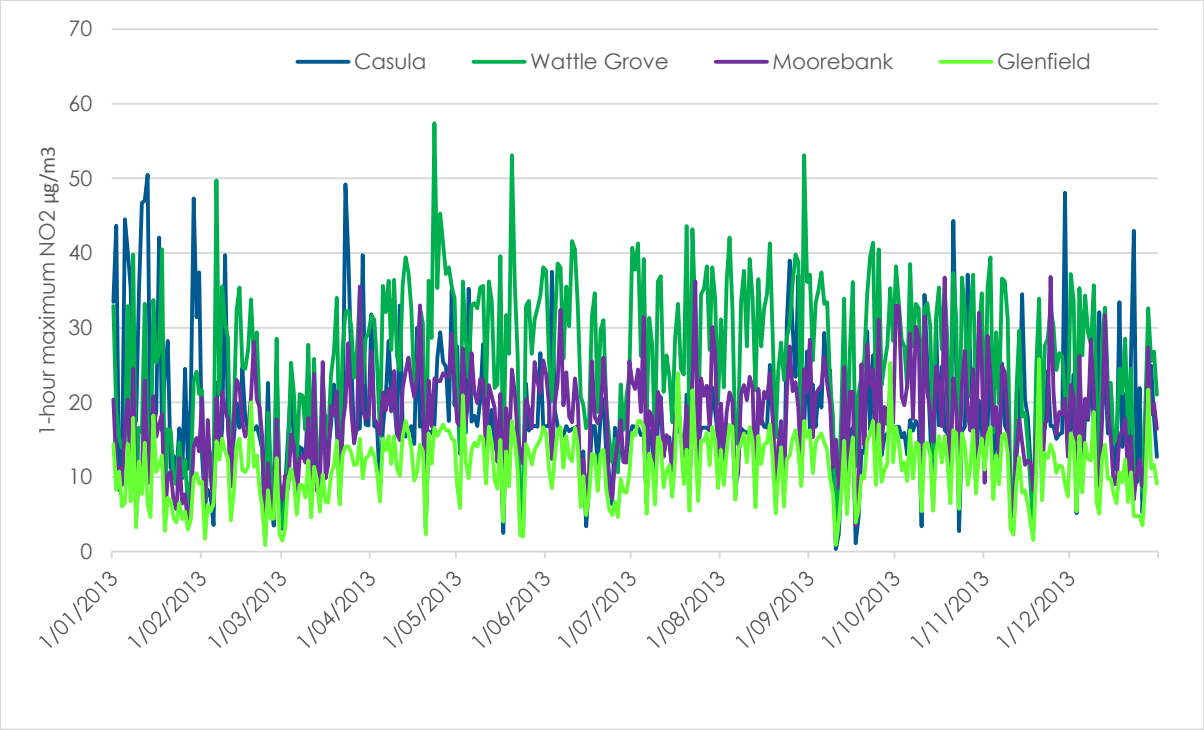
In the ACHAPS panel study (SCEW, 2012) the most consistent adverse effect was that increased NO_2 exposure was associated with an increased risk of cough and wheezing during the day and night, and increased use of bronchodilators for symptom relief. Relationships between NO_2 and night symptoms and effects were greater for NO_2 24-hr than for NO_2 1-hr and were more consistent. For lag 2 NO_2 1hr the odds ratio (OR) (95% CI) was 1.03 (1.01-1.05) per ppb for the association with night cough. For lag 2 NO_2 24-hr, ORs were 1.06 (1.03-1.09) per ppb for the association with night cough, 1.05 (1.011.10) per ppb for the association with night wheeze, and 1.05 (0.99-1.12) per ppb for the association with night shortness of breath. Effects upon symptoms occurring during the day were strongest at lag 0. For lag 0 NO_2 1-hr, the ORs (95% CI) were 1.02 (1.0-1.03), 1.04 (1.011.06), and 1.02 (0.99-1.05) per ppb for associations with day cough, wheeze and shortness of breath respectively. For lag 2 NO_2 24-hr, ORs were 1.05 (1.02-1.09), (1.11 (1.07-1.16), and 1.06 (1.01-1.11) per ppb for the association with day cough, wheeze and shortness of breath respectively.

The ACHAPS cross-sectional study shows consistent evidence of respiratory adverse effects of NO_2 for both recent and life-time exposure (SCEW, 2012). These adverse effects are manifested as increased risk of asthma-like symptoms (in particular, wheeze), increased airway inflammation and reduced lung volumes. For current asthma and per ppb recent exposure NO_2, the odds ratio (OR) was 1.06 (1.02, 1.10), with OR per interquartile range (IQR) NO2 1.26 (1.08, 1.48). For recent wheeze after exercise, the OR was 1.07 (1.03, 1.120) per ppb and 1.32 (1.12, 1.57) per IQR. Airways inflammation as measured by exhaled nitric oxide (NO) increased by 3% (1%-5%) and lung volume as measured by pre-bronchodilator forced expiratory volume (FEV1) and forced vital capacity (FVC) decreased by 7.1 ml (2.8-11.4) and 6.8 ml (2.7-10.9) per ppb respectively. Effect estimates were slightly smaller for lifetime exposure. Per IQR decreases in lung function measured by FEV1 and FVC pre- and post-bronchodilator ranged from 27.5 to 29 ml.

4.4.2.2 Exposure Assessment

Air dispersion modelling conducted as part of the air quality assessment has predicted maximum 1-hour, 24-hour average and annual average NO_x concentrations for a range of receptors within the suburbs surrounding the SIMTA site. The air quality assessment has assumed that all NO_x is NO_2 which is a conservative assumption. The data provided for the HRA also assumes that all NO_x is NO_2. The daily 24-hour NO_2 concentrations at the most affected receptors used in the HRA are shown in Figure 5:

Figure 5: Daily 1-hour maximum NO_2 concentrations for the SIMTA Stage 1 proposal



The data shown in Figure 5 show that all levels are all low. Assessment conducted as part of the Air Quality Assessment (Environ, 2015) shows that for all relevant averaging periods the NO_2 levels due to the SIMTA proposal are well below the current NEPM air quality standards. For the 1-hour maximum values the relevant NEPM standard is 246 µg/m^3. The levels predicted at Casula and Moorebank are similar but are slightly higher at the Wattle Grove location. The predicted values at Glenfield are lower than those at the other sites.

The NO_2 data generated in the Air Quality Assessment (Environ, 2015) has been used to calculate the risk of adverse health outcomes associated with exposure to NO_2 from the operation of the Stage 1 Proposal.

4.4.2.3 Risk Characterisation

The results of epidemiological studies have shown that a wide range of health effects are associated with exposure to NO_2. Australian studies (NEPC, 2012; EPHC 2006) have found associations between NO_2 levels currently experienced in Australian cities and the following health outcomes:

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

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

Although no studies investigating the long term effects of exposure to NO_2 on health have been conducted in Australia, there have been several international studies that have shown strong associations between long-term exposure to NO_2 and increases in mortality. On the basis of the findings of these studies long-term mortality has also been assessed.

The groups that were identified as being susceptible to the effects of NO_2 are:

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

The exposure-response functions in

Table 10 have been taken from Australian studies and in particular two multicity meta-analyses (Simpson et al., 2005; EPHC, 2006). Exposure response functions are the increase in a health outcomes observed per 1 µg/m^3 increase in pollutant concentration. The use of Australian meta-analyses is consistent with the NHMRC (2006) and NEPC (2011) recommendations for selecting exposure response functions for risk assessments for air pollution.

The exposure-response functions for long-term exposure to NO_2 have been taken from the results of a cohort of more than a million adults in Rome (Cesaroni et al., 2013). This study has been reviewed by the WHO as part of the REVIHHAP review (WHO, 2013). The use of this value is also consistent with the recommendations made by NHMRC (2006) and NEPC (2011).

Table 10: Exposure Response Functions for NO_2 Selected Health Outcomes (EPHC, 2005; Cesaroni et al. 2013)

Outcome	Averaging Period	Exposure Response Function per 1 µg/m³ increase in NO_2
Annual all-cause mortality (non-accidental) 30+ years	Annual Average	0.0028
Annual cardiovascular mortality 30+ years	Annual Average	0.0028
Annual respiratory mortality 30+ years	Annual Average	0.0028
Daily all-cause mortality(non-accidental) all ages	24 –hour average	0.001
Daily mortality respiratory disease - all ages	24 –hour average	0.0023
Daily mortality cardiovascular disease - all ages	24 –hour average	0.001
Hospital Admissions respiratory disease 65+ years	24 –hour average	0.003
Hospital Admissions cardiovascular disease 65+ years	24 –hour average	0.0014
Hospital Admissions respiratory disease 15-64 years	24 –hour average	0.001
ED Visits Asthma 1-14 years	24 –hour average	0.0006

The data shown in Figure 5 has been combined with the exposure response functions in Table 10 and baseline health statistics for Sydney to calculate the number of attributable cases attributable to NO_2 from the Stage 1 site operations. The number of attributable cases has been calculated using the following equation:

Number of attributable cases = exposure response function (increase in health outcome) per 1µg/m³ increase in NO_2 x NO_2 concentration x baseline health incidence rate/ 100,000 population x actual population

The number of attributable cases are summarised in Table 11.

Table 11: Attributable health outcomes due to NO_2 from the Stage 1 Proposal

OUTCOME	Casula	Wattle Grove	Moorebank	Glenfield
All-cause mortality 30+years (long-term)	0.7	0.6	0.4	0.3
Cardiovascular mortality 30+years (long-term)	0.2	0.2	0.14	0.1
Respiratory Mortality 30+ years (long-term)	0.06	0.05	0.04	0.03
Lung cancer mortality 30+ years (long-term)	0.05	0.05	0.03	0.02
Daily mortality all causes all ages	0.3	0.2	0.15	0.09
Daily mortality cardiovascular disease all ages	0.09	0.08	0.05	0.03
Daily mortality respiratory disease all ages	0.05	0.05	0.03	0.03
Hospital admissions respiratory disease 65+ years	0.6	0.3	0.4	0.02
Hospital admissions cardiovascular disease 65+ years	0.6	0.3	0.4	0.3
Hospital admissions respiratory disease 15-64 years	0.3	0.2	0.14	0.1
Emergency Department visits asthma 1-14 years	0.07	0.07	0.04	0.02

The results shown in Table 11 show that the risk from exposure to NO_2 from the Stage 1 Proposal operations is higher than that predicted for PM_{10} and $PM_{2.5}$. Based on the modelling data provided for the HRA, the highest risk is for long-term mortality in people over 30 years of age with approximately 7 additional deaths every 10 years predicted. A similar risk is predicted for hospital admissions for respiratory and cardiovascular disease in the 65+ years age group. This translates to a risk of between 1 in a million and 8 in 100,000. These calculations are based on a number of conservative assumptions that overestimate

the potential risk. Firstly, the worst case concentrations for each suburb would apply to the whole population of that suburb and secondly that all NO_x is NO_2. Based on monitoring from the Liverpool Air Monitoring station the ratio of NO_2 and NO_x is 0.7, ie, NO_2 is 70% of the monitored NO_x levels. If this is applied to the emissions from the SIMTA Stage 1 operations the risk from NO_2 is reduced by 30%. A number of actions that would reduce NO_2 emissions for the SIMTA Stage 1 operations are discussed in the Best Practice Review conducted as part of the Air Quality Assessment (Environ, 2015) and include the use of electric gantry cranes to replace diesel equipment for container handling and avoiding unnecessary idling of trucks and locomotives. The move to gantry cranes is predicted to reduce emissions of NO_2 by approximately 30%. Applying the 30% reduction for the ratio of NO_2 to NO_x from the data from the Liverpool Air Monitoring Station and the 30% reduction in NO_2 emissions by moving from diesel powered stack reachers to electrified gantry cranes for container handling reduces the risk estimates to 1 in a million to 3 in 100,000. Further reductions in NO_2 emissions will be achieved by the implementation of other measures outlined in the Best Practice Review for Air Quality which forms part of the Air Quality Assessment (Environ, 2015). The predicted increase in risk, based on the implementation of some of the best practice measures outlined in the Air Quality Assessment (Environ, 2015) is low. Further reductions in risk will be achieved through the implementation of further best practice measures such as reduction in idling times of locomotives and trucks.

4.4.3 Sulfur Dioxide (SO_2)

According to the Air Quality Assessment (Environ, 2015) conducted for the Stage 1 Proposal the main sources of SO_2 are locomotives (travelling and idling) and trucks (travelling and idling). The modelling has also been undertaken without considering the reduction in emissions that would be achieved by implementing the best practice measures in the Environ (2015) report

4.4.3.1 Literature Review of the Health Effects of SO_2

The health effects of sulfur dioxide (SO_2) linked to ambient air exposures have been well studied and reviewed by international agencies such as NEPC (2010), USEPA (2008), WHO (2005) and California EPA (OEHHA, 2011, 2000).

A large number of population-based epidemiological studies have reported a link between short term SO_2 exposure and daily mortality and respiratory and cardiovascular effects. The associations persist when other pollutants, such as particles, are controlled for. The epidemiological evidence is supported by controlled human exposure studies and animal toxicology studies. The strongest evidence comes from controlled human exposure studies examining short term exposure to SO_2 and respiratory effects. These studies have exposed volunteers to SO_2 for periods ranging from 5–10 min up to one hour. Adverse effects, such as sneezing or shortness of breath, occur within the first few minutes after inhalation and are not changed by further exposure. The effects are greater when the person is exercising, and are most pronounced in people with asthma and other respiratory conditions such as COPD, and particularly in exercising asthmatics.

A large number of epidemiological studies in cities in various parts of the world, including the United States, Canada and Europe, have reported associations between exposure to ambient levels of sulfur dioxide and increases in all-cause (non-accidental) and respiratory and cardiovascular mortality, often at mean 24-h average levels of <10ppb (Biggeri et al. 2005; Samet et al., 2000a; Dominici et al., 2003; Burnett et al., 1998a, 2000, 2004; Katsouyanni et al. 1997, 2006; Samoli et al., 2001, 2003; US EPA, 2008; Stieb et al. 2002, 2003). The mortality effect estimates for cardiovascular and respiratory causes are generally larger than for all-cause mortality (Zmirou et al., 1998), and the effect estimates for respiratory mortality are larger than the cardiovascular mortality, suggesting a stronger association of SO_2 with respiratory mortality compared to cardiovascular mortality. The mortality effect estimates from the multipollutant

models in the multicity studies suggest some extent of confounding between SO_2 and particles and/or NO_2 (USEPA, 2008).

The epidemiological evidence, supported by controlled human exposure studies and a limited number of animal toxicological studies conducted at near ambient concentrations, indicate an association between short-term exposure to SO_2 and several measures of respiratory health, including respiratory symptoms, inflammation, and airway hyper-responsiveness.

The epidemiological evidence further indicates that the SO_2-related respiratory effects (≥ 1-hour, generally 24-h average) are more pronounced in asthmatic children and older adults (65+ years). In the limited number of studies that examined potential confounding by copollutants through multipollutant models, the SO_2 effect was generally found to be robust after adjusting for particles and other co-pollutants (USEPA, 2008).

The strongest evidence for a causal relationship between respiratory morbidity and short term exposure to SO_2 comes from human clinical studies reporting respiratory symptoms and decreased lung function following peak exposures of 5–10min duration to SO_2. The exact duration is not critical, however, because responses occur very rapidly, within the first few minutes from commencement of inhalation; continuing the exposure further does not increase the effects. These effects have been observed consistently across studies involving mild to moderate asthmatics during exercise. Statistically significant decrements in lung function accompanied by respiratory symptoms including wheeze, chest tightness and shortness of breath have been clearly demonstrated following exposure to 0.4–0.6ppm SO_2.

Several studies have observed positive associations between ambient SO_2 concentrations and emergency department visits or hospital admissions for cardiovascular diseases (e.g., all cardiovascular diseases, cardiac diseases, cerebrovascular diseases) particularly among individuals 65+ years of age, but results are not consistent across studies. The strongest evidence comes from a large multicity study conducted in Spain (Ballester et al. 2006) that observed statistically significant positive associations between ambient SO_2 and cardiovascular disease admissions.

A large body of epidemiological studies generally report consistent and robust associations between ambient SO_2 concentrations and emergency department visits and hospitalizations for all respiratory causes, particularly among children and older adults (65+ years), and for asthma and chronic obstructive pulmonary disease (COPD) (USEPA, 2008).

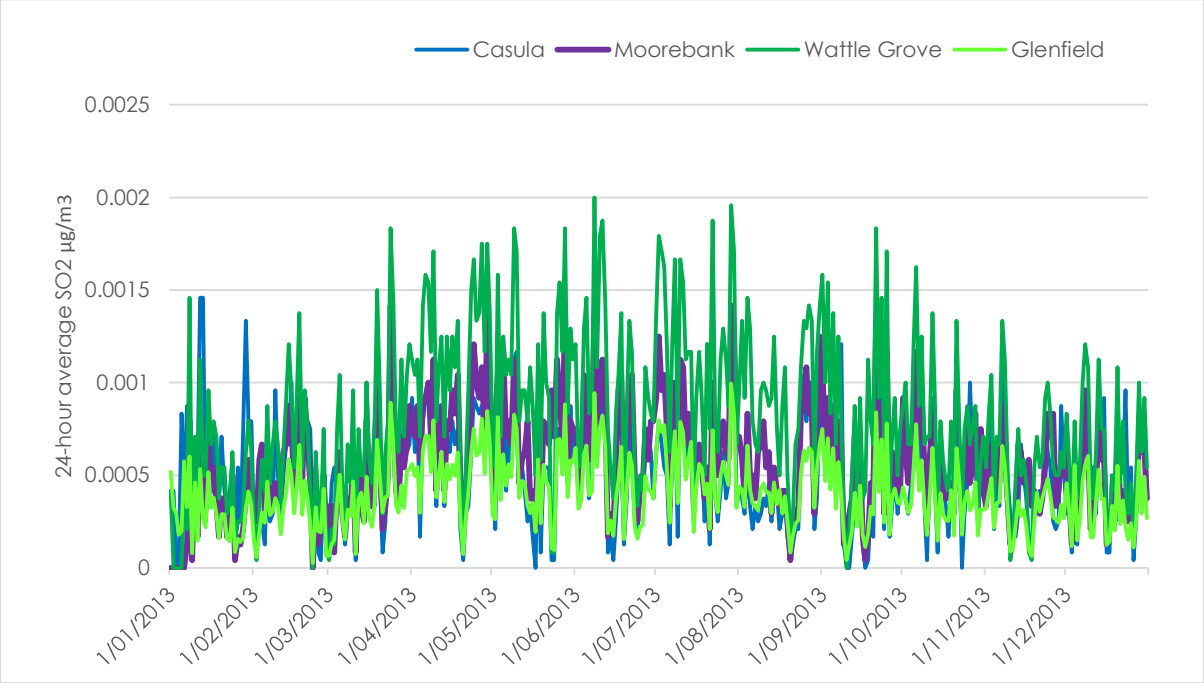
In a case-crossover study of air pollution and child respiratory health undertaken in five Australian and two New Zealand cities, Barnett et al. (2005) found a statistically significant increase in hospital admissions and SO_2 with an interquartile range of 5.4ppb for 1-hour SO_2. The ambient levels recorded during the study included: SO_2 1 hour mean (3 cities) 7.1ppb, range of means 3.7 to 10.1ppb; 24 hour mean (4 cities) 4.5ppb range of means 0.9 to 4.3ppb. In the 1–4 year age group there was evidence of seasonal impacts on pneumonia and acute bronchitis admissions for SO_2 (May to October 4.9% increase 95% CI, 0.6–10.8%, November to April 10.4% increase 95% CI, 2.1–19.4%) (Barnett et al. 2005).

A study of 123,840 singleton births of over 20 weeks' gestation in Sydney, between 1998 and 2000, found that 4.9% of babies were born at less than 37 weeks gestation. The mean of the one hour maximum SO_2 levels was 3.6ppb. SO_2 level in early pregnancy had a large adverse impact on gestational age in those infants conceived in autumn and winter for a 1ppb increase in SO_2. The authors noted that SO_2 appears to be an important pollutant, despite SO_2 levels in Sydney being well below the national standard, with vehicular traffic being the primary source and it is conceivable that SO_2 is a marker for traffic related air pollutants in the study (Jalaludin et al 2007).

4.4.3.2 Exposure Assessment

Air dispersion modelling conducted as part of the Air Quality Assessment (Environ, 2015) has predicted maximum 1-hour, 24-hour average and annual average SO_2 concentrations for a range of receptors within the surrounding suburbs. The daily 24-hour SO_2 concentrations at the most affected receptors used in the HRA are shown in Figure 6:

Figure 6: Daily 24-hour Average SO_2



The data shown in Figure 6 show that all levels are below the current Air Quality NEPM air quality standards of 228 µg/m³. The levels are similar across all locations but slightly higher at the Wattle Grove location. The SO_2 data generated in the Air Quality Assessment (Environ, 2015) has been used to calculate the risk of adverse health outcomes associated with exposure to SO_2 from the operation of the Stage 1 Proposal.

4.4.3.3 Risk Characterisation

The results of epidemiological studies have shown that a wide range of health effects are associated with exposure to SO_2. Australian studies (NEPC, 2012; EPHC 2006) have found associations between SO_2 levels currently experienced in Australian cities and the following health outcomes:

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

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

No studies investigating the long term effects of exposure to SO_2 on health were identified.

The groups that were identified as being susceptible to the effects of SO_2 are:

- Elderly
- People with existing cardiovascular and respiratory disease
- People with asthma
- Children

The exposure-response functions in Table 12 for mortality outcomes have taken from the results of the APHEA2 study in Europe (Katsouyanni et al., 2006). This study was a large meta-analysis across a number of cities and provides a robust exposure-response function in the absence of Australian data. The exposure-response function for hospital admission for respiratory disease has been taken from an Australian multicity meta-analysis (Simpson et al., 2005). A study by Jalaudin et al., (2008) examining the association between emergency department attendances for asthma in children conducted in Sydney has been used as the basis for the exposure-response function for this outcome.

Table 12: Exposure Response Functions for SO_2

Outcome	Averaging Period	Exposure Response Function per 1 µg/m³ increase in SO_2
Daily all-cause mortality(non-accidental) all ages	24 –hour average	0.0006
Daily mortality respiratory disease - all ages	24 –hour average	0.0013
Daily mortality cardiovascular disease - all ages	24 –hour average	0.0008
Hospital Admissions respiratory disease 65+ years	1 –hour maximum	0.002
ED Visits Asthma 1-14 years	24 –hour average	0.008

The data shown in Figure 6 has been combined with the exposure response functions in Table 12 and baseline health statistics for Sydney to calculate the number of attributable cases attributable to NO_2 from the Stage 1 Proposal operations. The number of attributable cases have been calculated using the following equation:

Number of attributable cases = exposure response function (increase in health outcome) per 1µg/m³ increase in SO_2 x SO_2 concentration x baseline health incidence rate/ 100,000 population x actual population

The number of attributable cases are summarised in Table 13:

Table 13: Attributable health outcomes due to SO_2 from the Stage 1 Proposal

HEALTH OUTCOME	Casula	Wattle Grove	Moorebank	Glenfield
Daily mortality all causes all ages	$3x10^{-5}$	$2x10^{-5}$	$2x10^{-5}$	$1.1x10^{-5}$
Daily mortality cardiovascular disease all ages	$1x10^{-5}$	$1x10^{-5}$	$7x10^{-6}$	$4.7x10^{-6}$
Daily mortality respiratory disease all ages	$2x10^{-3}$	$2x10^{-3}$	0.001	$7x10^{-4}$
Hospital admissions respiratory disease 65+ years	$4x10^{-4}$	$2x10^{-4}$	$2x10^{-4}$	$2x10^{-4}$
Emergency Department visits asthma 1-14 years	$2x10^{-4}$	$2x10^{-4}$	$9x10^{-5}$	$2x10^{-5}$

The results shown in Table 13 show that the risk from exposure to SO_2 from the Stage 1 Proposal operations is negligible. Based on the modelling data provided for the HRA, the highest risk is for daily mortality from respiratory causes with approximately 2 additional deaths per thousand years. All other risks are lower than this. The risks calculated are between 0.04 and 0.12 in a million at least an order of magnitude lower than the acceptable risk criteria adopted by international agencies.

4.4.4 Carbon Monoxide (CO)

According to the Air Quality Assessment (Environ, 2015) conducted for the Stage 1 Proposal the main sources of CO are locomotives (travelling and idling), container handling and trucks (travelling and idling). The modelling has also been undertaken without considering the reduction in emissions that would be achieved by implementing the best practice measures in the Environ (2015) report

4.4.4.1 Literature Review of the Health Effects of CO

The health effects of carbon monoxide are based on the ability of carbon monoxide to remove haemoglobin from your blood forming carboxyhaemoglobin (COHb). The clearest evidence of the health effects associated with short-term exposure to CO is provided by studies of cardiovascular morbidity. The combined health effects evidence supports a likely causal relationship for this outcome. Controlled human exposure studies provide strong evidence of independent effects of CO on cardiac function with effects being observed in patients with chronic airways disease following short-term CO exposures resulting in 2 to 2.4% the COHb. Epidemiological studies of emergency department visits and hospital admissions for ischaemic heart disease report consistent positive associations with additional preliminary evidence for an increase in cardiovascular related mortality provided by multi-city studies. This epidemiological evidence is coherent with ischemia related effects observed in control human exposure studies. New toxicological evidence suggests that other mechanisms involving altered cellular signalling may play a role in cardiovascular disease outcomes following carbon monoxide exposure.

Consistent decreases in time to onset of exercise induced angina along with changes in the heart that are indicative of myocardial ischaemia were observed in individuals with coronary artery disease (CAD)

following controlled CO exposures resulting in COHb concentrations of 2 to 6% with no evidence of a threshold at the lowest levels tested. Volunteers who participated in controlled exposure studies were diagnosed with moderate to severe CAD may not be representative of the most sensitive individuals in the population. Variability in activity pattern and severity of disease combined with daily fluctuations in baseline COHb levels may influence a critical level of increased COHb which can lead to adverse cardiovascular effects in a particular individual.

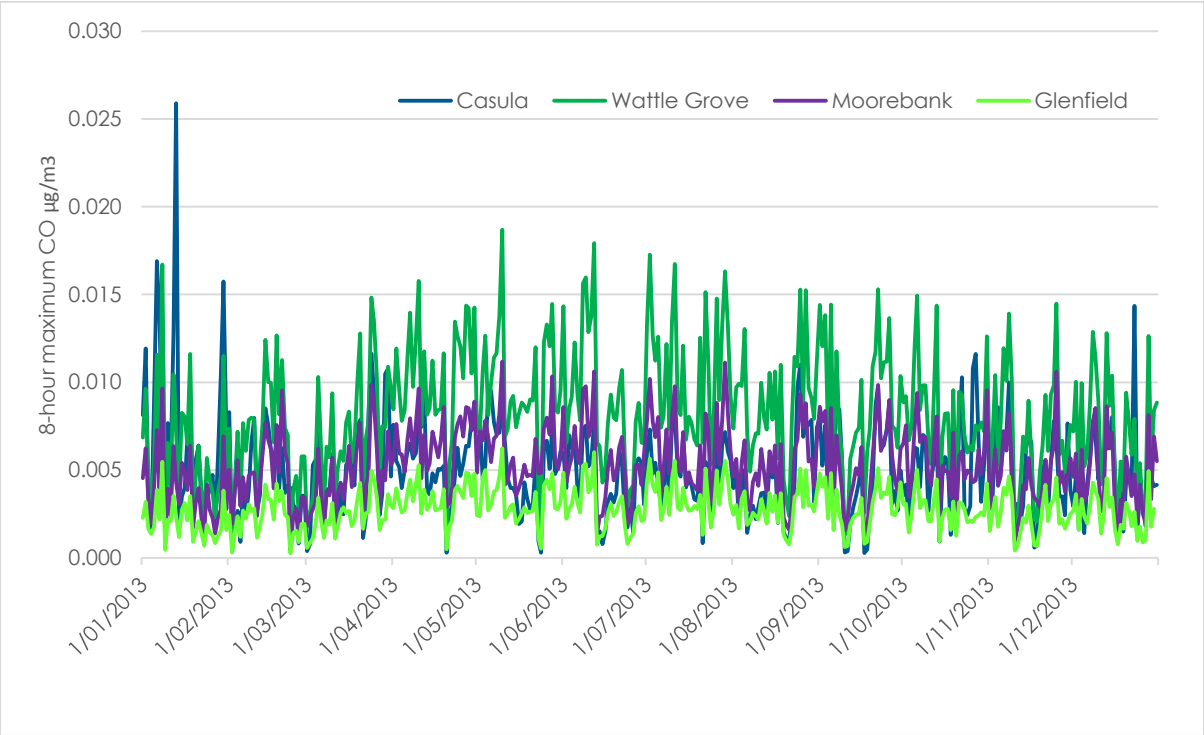
Controlled human exposure studies reviewed by the USEPA showed definitive evidence of cardiovascular effects among individuals with CAD following short-term CO exposure resulting in COHb concentrations as low as 2 to 2.4%. Evidence from control human exposure studies provide evidence of a causal relationship and reduce the uncertainties of previous assessments on the health effects of carbon monoxide. It is the consistent and coherent evidence from epidemiologic and human clinical studies along with biological plausibility provided by the role of CO in limiting oxygen availability that is sufficient for the US EPA to conclude that a causal relationship is likely to exist between relevant short-term carbon monoxide exposures and cardiovascular morbidity.

Recent studies observed associations between ambient CO concentration and emergency department visits and hospital admissions for ischaemic heart disease, congestive heart failure and cardiovascular diseases as a whole in locations with mean 24-hour average CO concentrations ranged from 0.5 ppm to 9.4 ppm. All but one of these studies that evaluated these outcomes reported positive associations. Although CO is often considered a marker of the effects of other traffic related pollutants or mix of pollutants evidence indicates that CO associations generally remain robust in two pollutant models and supports a direct effect of short-term ambient CO exposure on cardiovascular morbidity. The known role of CO in limiting oxygen availability lends biological plausibility to ischaemia related health outcomes following CO exposure however it is not clear whether small changes in COHb associated with ambient CO exposures resulting substantially reduced oxygen delivery to the tissues.

4.4.4.2 Exposure Assessment

The air dispersion modelling conducted as part of the air quality assessment has provided daily 8-hour maximum CO levels for worst affected locations in the surrounding suburbs. This data is shown in Figure 7.

Figure 7: Daily maximum 8-hour CO concentrations from Stage 1 Proposal operations



The data shown in Figure 7 show that the predicted CO levels are slightly higher at the Wattle Grove location compared to Moorebank, Glenfield and Casula. All predicted CO concentrations are well below the NEPM standard of 10 mg/m^3. The data from all locations has been used in the calculation of risk from CO for the Stage 1 Proposal.

4.4.4.3 Risk Characterisation

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

- Increases in daily mortality
- Hospital Admissions
    - Cardiac disease
    - Cardiovascular disease

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

No studies investigating the long term effects of exposure to CO on health have been identified.

The groups that have been identified as being susceptible to the effects of CO are:

- Elderly
- People with existing cardiovascular disease
- Low socioeconomic groups
- Foetus

The exposure-response functions in Table 14 have been taken from Australian studies and in particular two multicity meta-analyses (Simpson et al., 2005; EPHC, 2006). The use of Australian meta-analyses is consistent with the NHMRC (2006) and NEPC (2011) recommendations for selecting exposure response functions for risk assessments for air pollution.

No exposure-response functions for long-term exposure to CO have been identified.

Table 14: Exposure response functions for CO selected health outcomes

Outcome	Averaging Period	Exposure Response Function per 1 mg/m³ increase in CO
Daily all-cause mortality(non-accidental) all ages	8 hours	0.001
Hospital Admissions cardiac disease 65+ years	8 hours	0.003
Hospital Admissions cardiovascular disease 65+ years	8 hours	0.0014

The number of attributable cases has been calculated using the following equation:

Number of attributable cases = exposure response function(increase in health outcome) per 1mg/m³ increase in CO x CO concentration x baseline health incidence rate/ 100,000 population x actual population

The number of cases for each outcome was calculated for the population in each of the potentially affected suburbs using the data from the worst impacted residential receptor in each area. The number of cases for each day of the year were calculated and then summed to give the annual total. Table 15 shows the results for each study population for CO.

Table 15: Number of Attributable Cases for the Stage 1 Proposal for CO

Health Outcome	Casula	Wattle Grove	Moorebank	Glenfield
Daily Mortality all causes all ages	$4x10^{-4}$	$4x10^{-4}$	$2x10^{-4}$	$1x10^{-4}$
Hospital Admissions Cardiac Disease 65+ years	$1x10^{-3}$	$5x10^{-4}$	$6x10^{-4}$	$4x10^{-4}$
Hospital Admissions Cardiovascular Disease 65+ years	$1.4x10^{-3}$	$7x10^{-4}$	$1x10^{-3}$	$1x10^{-3}$

The results shown in Table 15 show that the health effects attributable to CO arising from the Stage 1 Proposal is very low. The highest risk is for hospital admissions for cardiovascular disease in people 65 years of age and older with an additional 1.4 deaths in one thousand years due to the emissions from the Stage 1 Proposal. This risk is negligible. The increase in risk associated with the SIMTA emissions are

between 0.01 and 0.04 in a million well below the internationally agreed criteria for acceptable risk of between 1 in a million and 1 in 100,000.

4.4.5 Health Effects of Air Toxics

A number of air toxics are emitted from the truck and rail activity associated with the Stage 1 Proposal. As part of the Air Quality Impact Assessment (Environ, 2015) air dispersion modelling has been conducted for benzene, 1,3-butadiene and PAHs. This data has been used in the risk assessment for air toxics from the Proposal.

4.4.5.1 Literature Review of the Health Effects of Benzene

Acute (short-term) inhalation exposure of humans to benzene may cause drowsiness, dizziness, headaches, as well as eye, skin, and respiratory tract irritation, and, at high levels, unconsciousness. Chronic (long-term) inhalation exposure has caused various disorders in the blood, including reduced numbers of red blood cells and aplastic anaemia, in occupational settings. Reproductive effects have been reported for women exposed by inhalation to high levels, and adverse effects on the developing foetus have been observed in animal tests. Increased incidence of leukaemia (cancer of the tissues that form white blood cells) have been observed in humans occupationally exposed to benzene.

Benzene is a well-established cause of cancer in humans. The International Agency for Research on Cancer has classified benzene as carcinogenic to humans (Group 1). Benzene causes acute myeloid leukaemia (acute non-lymphocytic leukaemia), and there is limited evidence that benzene may also cause acute and chronic lymphocytic leukaemia, non-Hodgkin's lymphoma and multiple myeloma. Individuals who have experienced benzene poisoning requiring treatment show a substantially increased risk of mortality from leukaemia. Benzene is a genotoxic carcinogen and does not have a threshold for effect.

Chronic inhalation of certain levels of benzene causes disorders in the blood in humans. Benzene specifically affects bone marrow (the tissues that produce blood cells). Aplastic anaemia (a risk factor for acute non-lymphocytic leukaemia), excessive bleeding, and damage to the immune system (by changes in blood levels of antibodies and loss of white blood cells) may develop. In animals, chronic inhalation and oral exposure to benzene produces the same effects as seen in humans.

4.4.5.2 Literature Review of the Health Effects of 1,3-butadiene

Motor vehicle exhaust is a constant source of 1,3-butadiene. Although 1,3-butadiene breaks down quickly in the atmosphere, it is usually found in ambient air at low levels in urban areas. Acute (short-term) exposure to 1,3-butadiene by inhalation in humans results in irritation of the eyes, nasal passages, throat, and lungs. Epidemiological studies have reported a possible association between 1,3-butadiene exposure and cardiovascular diseases. Epidemiological studies of workers in rubber plants have shown an association between 1,3-butadiene exposure and increased incidence of leukemia. Animal studies have reported tumours at various sites from 1,3-butadiene exposure. The available epidemiological and toxicological data provide evidence that 1,3-butadiene is carcinogenic in humans and may also be genotoxic in humans. The IARC and USEPA have classified 1,3-butadiene as carcinogenic to humans by inhalation.

1,3-butadiene is of low acute toxicity in experimental animals. However, long-term exposure to 1,3-butadiene was associated with reproductive and developmental effects in mice (OEHHA, 1997). 1,3-Butadiene also induced a variety of effects on the blood and bone marrow of mice; although data are limited, similar effects have not been observed in rats. Animal studies have reported tumours at a variety of sites from inhalation exposure to 1,3-butadiene. (ATSDR, 1992,USEPA, 2009,OEHHA, 1997).

4.4.5.3 Literature Review of the Health Effects of PAHs

The health effects of polycyclic aromatic hydrocarbons (PAHs) have been reviewed extensively and summarised by NEPC (2003). One of the complexities in evaluating the health effects of PAHs is that they exist as a mixture of compounds not individual compounds. The toxicity of these compounds varies quite markedly, with the most toxic being benzo(a)pyrene (BaP), which is classified as a probable human carcinogen.

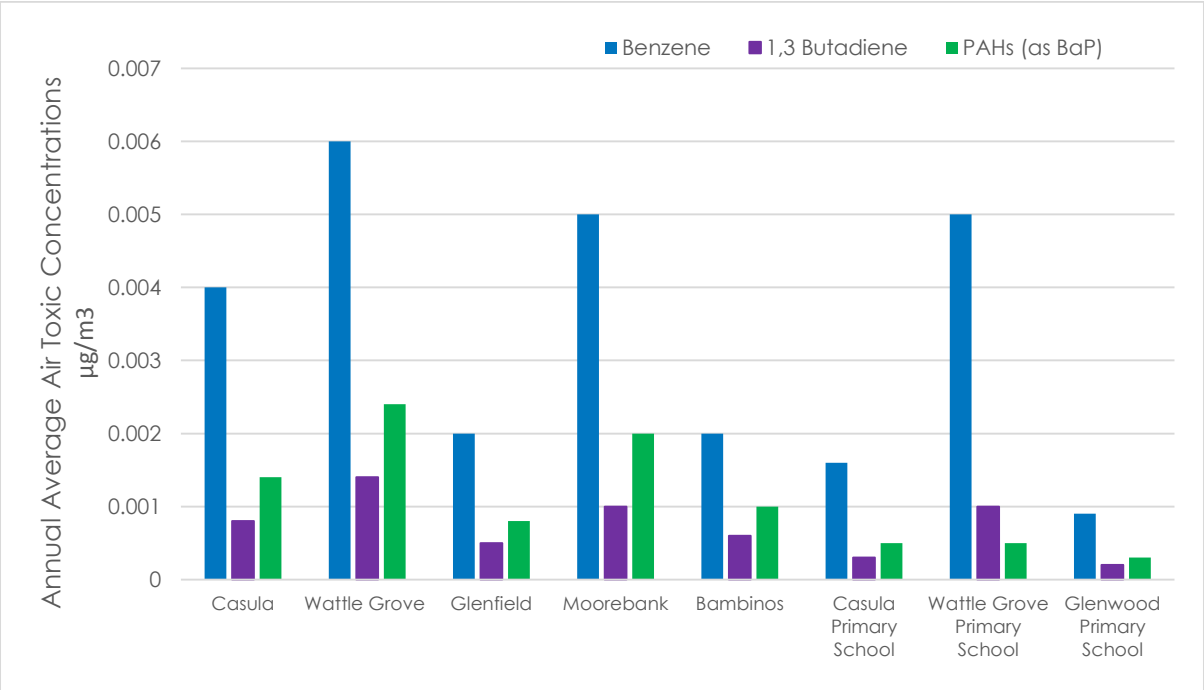
There is little information on human exposure to single, pure PAH. That which is available includes reports of accidental exposure to naphthalene and some data from defined short term studies of volunteers. All other reports are of exposure to mixtures of PAH, which also contained other (non-PAH) potentially carcinogenic chemicals, in occupational and environmental situations. Several epidemiological studies have shown increased mortality due to cancer, which has been associated with exposure to PAH-containing mixtures in humans exposed to coke oven emissions, roofing-tar emissions, and cigarette smoke. The cancers occur predominantly in the lungs and skin following inhalation and dermal exposure, respectively but can occur in other tissues away from the major route of exposure. It is thus impossible to evaluate the contribution of any individual PAH to the total carcinogenicity of these mixtures in humans because of the complexity of the mixtures and the presence of other carcinogens, and the potential interactions that could occur with other toxic substances in the mixtures. Despite these limitations, reports of this nature provide qualitative evidence of the potential for mixtures containing PAHs such as benzo(a)pyrene (BaP), chrysene, benz(a)anthracene, benzo(b)fluoranthene, and dibenz(a,h)anthracene to cause cancer in humans.

BaP is by far the most extensively studied PAH in experimental animals. It produces tumours of many different tissues, depending on the species tested and the route of application. BaP is the only PAH that has been tested for carcinogenicity following inhalation, and it produced respiratory tract tumours (not lung tumours) in hamsters, the only species tested (Thyssen et al, 1981). The lung carcinogenicity of BaP can be enhanced by co-exposure to other substances such as cigarette smoke, asbestos and airborne particles.

4.4.5.4 Exposure Assessment Air Toxics

From the review of the health effects of the air toxics presented above the critical health endpoint for evaluation is cancer. To enable the potential increased risk of cancer arising from the Stage 1 Proposal to be evaluated, annual average concentrations of benzene, 1,3-butadiene and PAHs have been modelled as part of the Air Quality Assessment (Environ, 2015). This data is shown in Figure 8 for the most affected location in each of the surrounding suburbs.

Figure 8: Annual Average Concentrations for Air Toxics SIMTA Stage 1 Proposal



The annual average concentrations are low and as shown in the air quality assessment all comply with relevant air quality standards.

The highest concentrations for each of the potentially affected areas have been used in the risk characterisation to give a worst case estimate for lifetime increased cancer risk attributable to these pollutants.

4.4.5.5 Risk Characterisation Air Toxics

The lifetime (assumed to be 70 years) increase in cancer risk has been calculated for the inhalation pathway. International agencies have published unit risk factors for a range of pollutants, including those assessed in this HRA, which is an estimate of the increase in risk with exposure to 1 µg/m^3 of the pollutant over a lifetime. To calculate the lifetime cancer risk associated with the concentrations of the air toxics predicted to arise from emissions from the operation of the Stage 1site the following equation has been used:

Increase in lifetime cancer risk = annual average concentration x unit risk factor (URF)

A review of the available unit risk factors has been undertaken. The derivation of these factors, like any standard or guideline, is based on a range of assumptions and key information on the concentrations at which these effects can be observed. To enable the risk from each of these substances to be compared it is important that as far as practicable all URFs are obtained from the same source and that the derivation of these values is well documented. Based on the review undertaken the URFs from the Californian EPA Office of Environmental Health Hazard Assessment (OEHHA) has been used in this study. The URFs used to calculate the increased risk of cancer in the HRA are summarised in Table 16:

Table 16: Unit Risk Factors (source OEHHA)

Pollutant	Unit Risk Factor (increase in risk per 1 µg/m³ increase in pollutant)
Benzene	2.9×10^{-5}
1,3 - butadiene	1.7×10^{-4}
PAHs (as BaP)	1.1×10^{-3}

Combining the annual average concentrations shown in Figure 8 the increase in cancer risk has been calculated and the results are shown in Table 17. In addition to the residential locations, the cancer risk has also been calculated for other sensitive receptors in proximity to the SIMTA site including schools and child care centres. These results are also shown in Table 17

Table 17: Lifetime increase in cancer risk for sensitive locations

	Lifetime Excess Cancer Risk		
	Benzene	**1,3 Butadiene**	**PAHs (as BaP)**
Casula	2.32×10^{-7}	1.36×10^{-7}	1.54×10^{-6}
Wattle Grove	1.74×10^{-7}	2.38×10^{-7}	2.64×10^{-6}
Moorebank	1.45×10^{-7}	1.70×10^{-7}	2.20×10^{-6}
Glenfield	5.8×10^{-8}	8.5×10^{-8}	8.8×10^{-7}
Bambinos	5.80×10^{-8}	1.02×10^{-7}	1.10×10^{-6}
Casula Primary School	4.64×10^{-8}	5.10×10^{-8}	5.50×10^{-7}
Wattle Grove Primary School	1.45×10^{-7}	1.70×10^{-7}	5.50×10^{-7}
Glenwood Primary School	2.6×10^{-8}	3.4×10^{-8}	3.3×10^{-7}

It is generally accepted by regulatory agencies that an increase in risk between 1×10^{-6} (1 in a million) and 1×10^{-5} (1 in 100,000) is considered to be a low risk and within acceptable criteria. As shown in Table 17 all predicted cancer risks are within this range with most well below. At the sensitive locations, schools and child care centres, the predicted level of risk is up to 100 times lower than the acceptable levels. Therefore the emissions from Proposal pose a very small increase in cancer risk which is well within the acceptable levels established by national and international agencies.

4.4.6 Diesel

Through the consultation on the Concept Approval for the SIMTA Project, concern was raised by several submitters about the potential health effects of diesel exhaust from trucks and locomotives associated with the SIMTA Project. Given that this issue was raised an assessment has been conducted on the potential cancer risk arising from exposure to diesel emissions associated with the Stage 1 Proposal.

4.4.6.1 Literature Review of the Health Effects of Diesel

Exposure to diesel exhaust can have immediate health effects. Diesel exhaust can irritate the eyes, nose, throat and lungs, and it can cause coughs, headaches, light headedness and nausea. In studies with human volunteers, diesel exhaust particles made people with allergies more susceptible to the materials to which they are allergic, such as dust and pollen. Exposure to diesel exhaust also causes inflammation in the lungs, which may aggravate chronic respiratory symptoms and increase the frequency or intensity of asthma attacks.

In experimental studies, healthy subjects have shown increased symptoms of irritation and compromised pulmonary function after short-term exposure to diesel exhaust. Additional studies have shown that diesel exhaust particles influence localized immunological components involved with allergic reactions. There have also been cases of newly developed asthma reported in workers exposed to diesel exhaust.

The inhalation or direct application of diesel into the respiratory tract of animals in acute and sub chronic studies induced inflammatory airway changes, lung function changes, and increased susceptibility of exposed animals to lung infection. The morphological effects observed in the lungs of animals in chronic inhalation exposures are mainly related to chronic inflammatory responses. Animal data indicate that chronic respiratory disease can result from long-term exposure to diesel exhaust. In rats, laboratory studies have shown that exposure to diesel exhaust can decrease resistance to infection and increase chronic inflammation. Rats, mice, rabbits, guinea pigs, and other primates all exhibit significant adverse pulmonary non-carcinogenic effects from long-term exposures to diesel exhaust.

Diesel exhaust and many individual substances contained in it (including arsenic, benzene, formaldehyde and nickel) have the potential to contribute to mutations in cells that can lead to cancer. A meta-analysis of 30 studies showed that occupational exposure to diesel exhaust is associated with an increased risk of lung cancer. Pooled relative risk estimates from 30 studies clearly reflect the existence of a positive relationship between diesel exhaust and lung cancer in a variety of diesel-exposed occupations. Based upon a review of these epidemiological studies conducted by OEHHA and the meta-analysis, it was concluded that these epidemiological studies provide evidence consistent with a causal relationship between occupational diesel exhaust exposure and lung cancer. The majority of these studies have reported elevated estimates of relative risk for lung cancer, many of which are statistically significant. The WHO and OEHHA have classified diesel particles and diesel exhaust as a known human carcinogen.

4.4.6.2 Exposure Assessment

The carcinogenic effects of diesel are associated primarily with particle fraction of the diesel. The unit risk factors for diesel are for diesel particles. The air modelling that has been done for $PM_{2.5}$ for the Stage 1 Proposal has been done for emissions from diesel trucks, locomotives and equipment used on within the Proposal site as well as non-diesel motor vehicle emissions. Advice from the air quality specialist is that the annual average $PM_{2.5}$ arising from the Proposal arises predominantly from the diesel sources. Therefore it has been assumed that all $PM_{2.5}$ is attributable to diesel for the purposes of the HRA. This is a conservative assumption but is justified on the basis of the inventory of $PM_{2.5}$ emission sources at the Proposal site.

The air dispersion modelling has predicted annual average $PM_{2.5}$ concentrations for the potentially affected areas. These values are summarised in Table 18:

Table 18: Annual Average $PM_{2.5}$ concentrations

Location	Annual Average $PM_{2.5}$ (µg/m³)
Casula	0.12
Wattle Grove	0.2
Moorebank	0.13
Glenfield	0.07
Bambinos	0.08
Casula Primary School	0.05
Wattle Grove Primary School	0.15
Glenfield Primary School	0.03

These concentrations represent the highest predicted concentrations within the surrounding suburbs as well as sensitive locations. These values have been used in the calculation of the cancer risk associated with diesel emissions from the operation of the Stage 1 Proposal. These predicted concentrations of $PM_{2.5}$ are based on the use of diesel powered reach stackers for container handling which provides the worst case scenario for diesel emissions.

4.4.6.3 Risk Characterisation

As with the risk characterisation for the air toxics, the URF from OEHHA has been used in the assessment of the increase in cancer risk associated with diesel particles from the operations at the Stage 1 site. The URF from OEHHA for diesel particles is $3x10^{-4}$ per 1 µg/m³ increase in diesel particles. The resultant cancer risk is shown in Table 19:

Table 19: Increase in Cancer Risk attributable to diesel particles

Location	Lifetime Excess Cancer Risk
Casula	3.6×10^{-5}
Wattle Grove	4×10^{-5}
Moorebank	3.9×10^{-5}
Glenfield	2.1×10^{-5}
Bambinos	2.4×10^{-5}
Casula Primary School	1.5×10^{-5}
Wattle Grove Primary School	4.5×10^{-5}
Glenwood Primary School	9×10^{-6}

Some of the results shown in Table 19 are the high end of what is generally accepted as acceptable risk. These risks are based on the conservative assumption that all $PM_{2.5}$ is due to diesel emissions. This is expected to lead to an overestimate of the risk. These results indicate that actions should be taken to reduce diesel emissions from vehicles and machinery used within the Stage 1 site and vehicles entering and leaving the Proposal. Options to reduce the impact of diesel emissions from locomotives should be implemented. A number of options are discussed in the Best Practice Review for air quality (Environ, 2015)

As discussed above, the air quality modelling scenario was based on the 'worst case' scenario, including the use of diesel powered reach stackers. The use of gantry cranes would be deployed progressively in the Stage 1 Proposal of the SIMTA development, resulting in a reduction in the use of diesel powered container handling equipment and an associated reduction in emissions. The Air Quality Assessment (Environ, 2015) estimates that a move to the use of electrified gantry cranes for container handling would lead to approximately a 45% reduction in diesel particles. If this reduction is applied to the annual average $PM_{2.5}$ concentrations in Table 18 the resultant risk estimates fall within the acceptable level for risk generally adopted by international agencies. The resulting risk estimates are presented in Table 20:

Table 20: Increase in Cancer Risk attributable to diesel particles with use of electrified gantry cranes for container handling

Location	Lifetime Excess Cancer Risk
Casula	$2x10^{-5}$
Wattle Grove	$3x10^{-5}$
Moorebank	2x10-5
Glenfield	$1.1x10^{-5}$
Bambinos	$1x10^{-5}$
Casula Primary School	$8x10^{-6}$
Glenwood Primary School	$4.5x10^{-6}$
Wattle Grove Primary School	$2x10^{-5}$

The results shown in Table 20 show the effectiveness of the move from diesel powered container handling equipment to gantry cranes at the site in reducing the potential risk to the health of the local community. Implementation of other Best Practice Measures, as identified within the review (Environ, 2015), would lead to further improvements to air quality.

4.4.7 Regional impacts

As discussed for PM_{10} and $PM_{2.5}$, the impact of emissions from the Stage 1 Proposal on regional air quality were not assessed quantitatively in the Air Quality Assessment (Environ, 2015). Therefore a quantitative assessment of risk cannot be undertaken. The Air Quality Assessment commentary on regional impacts was based on previous work done on the emissions from road vehicles and railway locomotives associated with the SIMTA Project on the Port Botany-Moorebank Corridor (Pacific Environment, 2013). This report used changes in total emissions to estimate the impact on regional air quality and found that the changes in emissions would be negligible when considered at a regional level. On this basis Environ (2015), concluded that the impacts on regional air quality would also be negligible. If there is no increase in emissions that would subsequently lead to an increase in regional air pollution associated with the Stage 1 Proposal, then it can be concluded that there will be no associated increase in health risk. It should be noted that the assessment conducted in the Pacific Environment (2013) report was based on a 1 million TEU annual throughput for the SIMTA project. The current Stage 1 proposal is for 250,000 TEU throughput per annum which will generate fewer emissions and lower impact on air quality than the earlier proposal with 1 million TEU per annum.

4.4.8 Cumulative impact scenario

The cumulative impacts of the Early Works associated with the MIC Proposal and the Stage 1 Proposal operating at 250,000TEU throughput per annum, have been modelled in the Air Quality Assessment (Environ, 2015) for PM_{10}, $PM_{2.5}$ and NO_2. Although the modelling for SO_2 and CO was conducted the results were not presented as the incremental increase was negligible. The annual average increases for PM_{10}, $PM_{2.5}$ and NO_2 are shown in Table21.

The data shown in Table 21 show that the combined impact of the Stage 1 Proposal and the MIC Proposal Early Works has a small impact on the receptors that with only small increases in the annual average PM_{10}, $PM_{2.5}$ and NO_2 levels in the surrounding suburbs. The combined annual averages have been used to calculate the cumulative health risk from both developments. These results are shown in Table 22.

The results for PM_{10} and $PM_{2.5}$ show that the addition of the emissions from the MIC site approximately doubles the number of health outcomes attributable to exposure to these pollutants. The risk however is still low with an increase in risk of mortality for the combined emissions of 1 in 100,000 which is within the acceptable risk levels established by national and international agencies.

For NO_2 the incremental risk is even lower. The combined emissions from the two developments does not significantly increase the predicted health risk. The most significant risk is for an increase in all-cause mortality with a predicted increase of 0.1% in attributable cases. For all other outcomes there is no difference with the emissions from the MIC site included.

The calculations shown in Table 22 are based on long-term chronic effects. Similar results would be observed for the short-term impacts.

Table 21 Incremental Increase from the Stage 1 Proposal and MIC Proposal Early Works

Pollutant	Stage 1 Proposal				MIC Proposal – Early Works				Cumulative			
	Casula	Wattle Grove	Moorebank	Glenfield	Casula	Wattle Grove	Moorebank	Glenfield	Casula	Wattle Grove	Moorebank	Glenfield
PM_{10} annual average	0.13	0.2	0.14	0.08	0.1	0.1	0.1	0.1	0.23	0.3	0.24	0.18
$PM_{2.5}$ annual average	0.12	0.2	0.13	0.07	0.1	0.1	0.1	0.1	0.22	0.3	0.23	0.17
NO_2 annual average	3.13	3.44	5.1	2.15	0.2	0.2	0.2	0.1	3.33	3.64	5.3	2.17

Table 22: Long-term health risk attributable to the combined emissions from the Stage 1 Proposal and MIC Proposal Early Works

HEALTH OUTCOME	Casula	+MIC	Wattle Grove	+MIC	Moorebank	+MIC	Glenfield	+MIC
PM_{10}								
All-cause mortality 30+years (long-term	0.04	0.07	0.03	0.05	0.02	0.04	0.02	0.03
$PM_{2.5}$								
All-cause mortality 30+years (long-term)	0.05	0.1	0.05	0.07	0.03	0.06	0.02	0.05
Cardiopulmonary mortality 30+years (long-term)	0.05	0.1	0.05	0.07	0.03	0.06	0.02	0.05
Ischemic Heart Disease 30+ years (long-term)	0.03	0.06	0.03	0.05	0.02	0.04	0.01	0.03
Lung cancer mortality 30+ years (long-term)	0.008	0.01	0.007	0.01	0.005	0.008	0.003	0.007
NO_2								
All-cause mortality 30+years (long-term)	0.7	0.71	0.6	0.6	0.4	0.4	0.3	0.3
Cardiovascular mortality 30+years (long-term)	0.2	0.24	0.2	0.2	0.14	0.15	0.1	0.1

HEALTH OUTCOME	Casula	+MIC	Wattle Grove	+MIC	Moorebank	+MIC	Glenfield	+MIC
Respiratory Mortality 30+ years (long-term)	0.06	0.07	0.05	0.05	0.04	0.04	0.03	0.03
Lung cancer mortality 30+ years (long-term)	0.05	0.06	0.05	0.05	0.03	0.03	0.02	0.02

4.4.9 Summary

The health risk assessment for the emissions to air from the operation of the Stage 1 Proposal has shown that the highest risk arises from exposure to NO_2 and diesel particles. The HRA is based on modelling undertaken without the best practice measures identified for the project being implemented. As shown in Table 20, the implementation of best practice measures identified in the Air Quality Assessment can effectively reduce the potential risk to the local community.

The health risk from PM_{10}, $PM_{2.5}$, CO is very low. The number of attributable cases from PM_{10} from the Stage 1 Proposal are low and well within internationally accepted guidelines for acceptable risk. For all outcomes the increased risk is between 1 in a million and 1 in 100,000 which is generally considered as acceptable levels of risk by international agencies such as WHO and USEPA. The risks for all outcomes for both PM_{10} and $PM_{2.5}$ are very low and the increase is unlikely to be observed within the community.

For CO the health effects attributable to CO arising from the Stage 1 Proposal is very low. The highest risk is for hospital admissions for cardiovascular disease in people 65 years of age and older with an additional 1.4 deaths in one thousand years due to the emissions from the Stage 1 Proposal. This risk is approximately 100 times lower than the levels of risk considered acceptable by international agencies.

The results of the HRA show that the risk from exposure to NO_2 from the Stage 1 Proposal operations is higher than that predicted for PM_{10} and $PM_{2.5}$. Based on the modelling data provided for the HRA, the highest risk is for long-term mortality in people over 30 years of age with approximately 7 additional hospital admissions per 10 years predicted. A similar risk is predicted for hospital admissions for respiratory and cardiovascular disease in the 65+ year age group. This correlates with an increase in risk of between 1 in 1 million and 8 in 100,000. These calculations are based on a number of conservative assumptions that are likely to overestimate the risk attributed from the Proposal. Firstly, the worst case concentrations for each suburb would apply to the whole population of that suburb and secondly that all NO_x is NO_2. Using the ratio of NO_2 to NO_x obtained from monitoring data from the Liverpool Air Monitoring Station reduces the risk by 30 %.

A number of these actions are discussed in the Best Practice Review conducted a part of the Air Quality Assessment (Environ, 2015) to reduce NO_2 levels and include the use of electric gantry cranes to replace the diesel powered equipment for container handling. A detailed discussion of best practice measures in contained in Appendix 1 of the Air Quality Assessment Report. It is estimated that the move to the use of gantry cranes will reduce NO_2 emissions by 30%. Applying this reduction to NO_2 concentrations predicted from the air quality modelling and the ratio of NO_2 to NO_x from the Liverpool Air Monitoring Station reduces the risk from exposure from NO_2 from the emissions from the SIMTA Stage 1 proposal reduces the risk level to between 1 in a million to 3 in 100,000. Implementation of other Best Practice Measures, such as avoiding the unnecessary idling of locomotives and trucks on site, as identified in the review would further reduce this risk.

The increased cancer risk from the air toxics assessed, benzene, 1,3-butadiene and PAHs is low. It is generally accepted by regulatory agencies that an increase in risk between 1×10^{-6} (1 in a million) and 1×10^{-5} (1 in 100,000) is considered to be a low risk and within acceptable criteria. All predicted cancer risks are within this range with most well below. At the sensitive locations, schools and child care centres, the predicted level of risk is up to 100 lower than the acceptable levels. Therefore the emissions from the Stage 1 Proposal pose a very small increase in cancer risk which is well within the acceptable levels established by national and international agencies.

For diesel emissions some of the predicted increases in cancer are the high end of what is generally accepted as acceptable risk. These risks are based on the conservative assumption that all $PM_{2.5}$ is

due to diesel emissions. This may lead to an overestimate of the risk. These results indicate that actions should be taken to reduce diesel emissions from vehicles and machinery used on site and trucks entering and leaving the site. Options to reduce the impact of diesel emissions from locomotives should be considered. A number of options are discussed in the best practice review for air quality (Environ, 2015).

The modelling scenario used as the basis of the HRA was based on the use of diesel powered reach stackers. The air quality assessment estimates that a move to the use of electrified gantry cranes for container handling would lead to approximately a 45% reduction in diesel particles. If this reduction is applied to the annual average $PM_{2.5}$ concentrations used in the risk calculations the resultant risk estimates fall within the acceptable level for risk generally adopted by international agencies. These results show the effectiveness of implementation of best practice measures at the site in reducing the potential risk to the health of the local community.

The Air Quality Assessment (Environ, 2015) concluded that the regional impacts of the SIMTA Stage 1 proposal were negligible. Based on the information provided in the Air Quality Assessment (Environ, 2015) that there will be no change to emissions to the regional airshed from the SIMTA Stage 1 proposal, then the risk to the health of the community from changes in regional air quality will also not change.

The cumulative assessment from the combined Stage 1 operations and associated transport emissions and the MIC Proposal Early Works has shown that the cumulative impacts do increase the risk to the health of the local community due to changes in air quality.

5 HEALTH RISK ASSESSMENT NOISE

5.1.1 Review of the Health Effects of Environmental Noise

In recent years evidence has accumulated regarding the health effects of environmental noise. Epidemiological studies have found that cardiovascular diseases are consistently associated with exposure to environmental noise. The WHO has released two reports on the health effects of environmental noise: *Guidelines for Community Noise* (1999) and the *Burden of Disease from Environmental Noise* (2011). In these documents the main health effects associated with environmental noise are:

- Cardiovascular disease
- Cognitive impairment
- Sleep disturbance
- Tinnitus
- Annoyance
- Hearing Impairment

An increasing body of literature has shown traffic noise to have adverse short- and long-term health effects (Babisch 2006; Berglund et al. 1999; Bluhm et al. 2007; Stansfeld et al. 2000, 2005). One of the suggested mechanisms by which noise affects nonauditory health is through indirect or direct activation of the sympathetic nervous system and endocrine systems (Ising and Kruppa 2004; Stansfeld and Matheson 2003), resulting in autonomic reactions, including increased blood pressure, heart rate, and arrhythmia (Berglund et al. 1999). Therefore, research has focused on the impact of transportation noise on cardiovascular health. There is suggestive evidence that transportation noise exposure is associated with an increase in ischemic heart disease (Babisch 2006). Associations between traffic noise and hypertension have been inconsistent (Babisch et al. 2006; Chang et al. 2009; Jarup et al. 2008; van Kempen et al. 2006).

5.1.1.1 Environmental noise and cardiovascular disease

Epidemiological studies suggest a higher risk of cardiovascular diseases, including high blood pressure and myocardial infarction, in people chronically exposed to high levels of road or air traffic noise. While there is evidence that road traffic noise and aircraft noise increase the risk of ischaemic heart disease, including myocardial infarction (WHO, 2011).

Non-auditory health effects of noise have been studies in animals and humans for several decades. Noise is a non-specific stressor that that arouses the autonomous nervous system and the endocrine system (WHO, 2011). Among other non-auditory health end-points, short-term changes in circulation, including blood pressure, heart rate, cardiac output and vasoconstriction, as well as stress hormones have been studies in experimental settings for many years. Classical biological risk factors have be shown to be elevated in subjects exposed to high levels of noise (WHO, 2011).

These studies have supported the hypothesis that persistent noise stress increases the risk of cardiovascular disorders, including hypertension and ischaemic hearty disease. According to the noise/stress reaction model, the arousal of the endocrine and autonomic nervous system affects classical biological risk factors (eg., blood pressure, blood lipids, glucose regulation, blood flow, haemostatic factors and cardiac output). Chronic metabolic changes or dysfunction due to noise increase the risk of diseases, including hypertension, arteriosclerosis and myocardial infarction (WHO, 2011).

A further study by Dratva et al (2012) found evidence of an adverse effect of railway noise on blood pressure in a cohort study in Switzerland. Traffic noise was associated with higher blood pressure only

in diabetics, possibly due to low exposure levels. The study results imply more severe health effects by transportation noise in vulnerable populations, such as adults with hypertension, diabetes, or CVD.

The study by Dratvic et al (2012) found significant positive associations of railway noise during day and night with systolic blood pressure and diastolic blood pressure in an adult cohort in Switzerland. Associations were particularly strong among subjects with reported physician-diagnosed hypertension, diabetes, or CVD. For traffic noise, no association with blood pressure was found in the study population as a whole, whereas stratified analyses yielded a significant positive association with blood pressure in participants with doctor-diagnosed diabetes. Traffic noise from heavily frequented streets corresponds to a continuous exposure without much variation in noise exposure levels, whereas railway noise is characterized by a discontinuous pattern of intermittent very high maximum sound pressure levels and steep slopes of rise. The currently used measure of noise [equivalent sound level (Leq)], based on the average sound level, therefore potentially underestimates the detrimental effect of rail and aircraft noise (Lercher et al. 2010). The intermittent pattern of noise exposure is thought to be more disruptive (Griefahn et al. 2008). Especially during night time railway noise causes repeated sleep disturbances and activation of the sympathetic nervous system, which may reduce the normal nocturnal blood pressure (Babisch et al. 2001; Griefahn et al. 2008; Haralabidis et al. 2010),

A study by Sorenson et al (2011) showed that long-term exposure to road traffic noise was weakly associated with a higher systolic BP in a cross-sectional design, whereas long-term exposure to road traffic noise was not associated with risk for self-reported development of hypertension in a prospective design. Exposure to railway noise of 60 dB or more was associated with an 8% higher risk for hypertension.

High blood pressure is a major risk factor for cardiovascular disease and, therefore, even small increases in BP from road traffic noise may have high impact on public health. Older people might be a susceptible group with regard to the hazardous effects of traffic noise as the relationship between exposure to road traffic noise and blood pressure was found to be strongest among the older participants (over 60 years) (Sorenson, 2011). Sleep disturbances contribute to cardiovascular risk, and it is therefore believed that night noise exposure is more harmful than daytime exposure. The sleep structure generally becomes more fragmented with age and older people are, thus, more susceptible to sleep disturbances.

5.1.1.2 Environmental Noise and Cognitive Impairment in Children

Children may be particularly vulnerable to the effects of noise because they may have less cognitive capacity to understand environmental issues and anticipate stressors and they may lack appropriate coping strategies to deal with noise. Additionally, noise may interfere with learning at a critical developmental stage.

The impact of environmental noise on children's learning and memory has been known for many years. Epidemiological studies show effects of chronic noise exposure on tasks involving central processing and language, such as reading, comprehension, memory and attention. Experimental studies investigating acute (short-term) exposures have found similar effects. Exposure during critical periods of learning at school could potentially impair development and have a lifelong effect on educational attainment.

Cognitive impairment has been defined as '*delayed psychomotor development and impaired performance in language skills, motor skills, and coordination equivalent to a 5- to 10- point deficit in IQ*' (Lopez etal, 2006). None of the epidemiological studies on noise and cognitive impairment have used IQ as a health endpoint.

The WHO (2011) has defined cognitive impairment as '*Reduction in the ability in school-age children that occurs while noise exposure persists and will persist for some time after the cessation of the exposure*'. A notable characteristic of this definition is that the cognitive impairment is assumed to

show itself during the noise exposure as well as some time after the exposure has stopped. Two other large studies have also found effects of road and aircraft noise at levels between 46 to 62 dB(A) L_{dn}. Long-term noise exposure was significantly related to both intentional and incidental memory.

5.1.1.3 Environmental Noise and Sleep Disturbance

Sleep disturbance in one of the most common complaints raised by noise exposed communities and can have a major impact on health and quality of life (WHO (2011)). Studies have shown that noise affects sleep in terms of immediate effects (eg., arousal responses, sleep stage changes, awakenings, body movements, total wake time, autonomic responses), after effects (eg., sleepiness, daytime performance cognitive function deterioration) and long-term effects (eg., self-reported chronic sleep disturbance).

Sufficient undisturbed sleep is necessary to maintain performance during the day as well as for good health (WHO (2011)). Humans recognise, evaluate and react to environmental sounds even when asleep. These reactions are part of an integral activation process of the human body and express themselves as changes in sleep structure or increases in heart rate. Although they are natural responses to noise, it is assumed that substantial increase in the number of these effects constitutes a health issue. Environmental noise may reduce the restorative power of sleep by means of repeatedly occurring activations (so called sleep fragmentation). Acute and chronic sleep restrictions or fragmentation has been shown to affect waking psychomotor performance, memory consolidation, creativity, risk-taking behaviour, signal detection performance and risks of accidents (WHO, 2011).

5.2 Exposure Assessment

As part of the approvals process for the Stage 1 Proposal a Noise and Vibration Assessment has been undertaken (Wilkinson-Murray, 2015). The exposure data for the noise HRA been taken from this report. The sources of noise that have been considered in this HRA are operational and rail noise. The noise modelling has been undertaken for the worst case scenario without mitigation measures to reduce noise being implemented.

The Noise Assessment conducted for the Stage 1 Proposal (Wilkinson-Murray, 2015) identified a number of residential noise catchment areas (NCA) in proximity to the Proposal site. Several non-residential receivers have also been identified. Table 23 presents the sensitive receivers identified in Noise and Vibration Assessment (Wilkinson Murray, 2015), and their proximity to key aspects of the Proposal.

Table 23: Sensitive Receivers used in Noise Assessment

Receiver ID	Description	Distance (m)		
		Stage 1 Operational Area	Stage 1 Construction Area	Rail link
NCA1	Wattle Grove, south of Anzac Road	770	600	790
NCA2	Wattle Grove, north of Anzac Road	1,050	900	1,900
NCA3	Casula	960	220	220
NCA4	Glenfield	1,700	750	760
S1	All Saints Senior College	1,250	260	260
S2	Casula Powerhouse	930	380	690

I1	DNSDC	370	80	1,010

Rating background noise levels (RBL) at sensitive receiver locations representative of each of the four Noise Catchment Areas (NCA) were established in accordance with the *NSW Industrial Noise Policy* (INP) (EPA, 2000). The RBL are presented in Table 24.

Table 24: Existing $L_{Aeq, period}$ noise levels:

Receiver Catchment	Day (7am – 6pm)	Evening (6pm – 10pm)	Night (10pm – 7am)
NCA1	49	49	46
NCA2	49	47	45
NCA3	58	57	55
NCA4	53	53	53

The Noise and Vibration Assessment (Wilkinson Murray , 2015) has assumed that the Stage 1 Proposal will operate on a 24/7 basis with activity levels varying throughout the day. It has been assumed that the Stage 1 Proposal will operate at a constant rate throughout the day, equal to the average anticipated activity levels. This assumption is not applied to the assessment of $L_{Aeq, 15min}$ operational noise levels, which are based on peak operational activity levels (Wilkinson-Murray, 2015). Rail noise predictions are made for all trains travelling between the Stage 1 site and the SSFL. Previous assessments and approval of the SSFL are understood to account for freight movements generated by an intermodal terminal facility in the Moorebank area. Therefore, no assessment was undertaken of noise emissions from movements on the SSFL generated by the Proposal (Wilkinson Murray, 2015).

The predicted $L_{Aeq, period}$ and L_{Amax} operational noise levels from noise generated by the Stage 1 site, at nearby sensitive receivers are presented below in Table 25.

Table 25: Predicted $L_{Aeq, period}$ Operational Noise Levels and Predicted L_{Amax} Noise Levels at Sensitive Receivers

Receiver	Predicted $L_{Aeq, period}$ Noise Level (dBA)	Predicted Level due to Transient Events (dBA – L_{Amax})
NCA1	33	48
NCA2	20	38
NCA3	33	48
NCA4	25	41
S1	32	
S2	29	
I1	26	

One of the key issues raised during consultation for the Concept Approvals with respect to noise is the noise associated with the rail movements associated with the SIMTA Project. Table 26 shows the $L_{Aeq, period}$ and L_{Amax} rail noise levels for the northern section of the Rail link to the site. It is expected that all freight movements generated by Stage 1 will access the northern connection (Wilkinson Murray, 2015).

Table 26: Predicted $L_{Aeq, period}$ and L_{Amax} Rail Noise Levels – Northern connection

Receiver	Predicted Level L_{Aeq} (dBA)		Predicted Level L_{max} (dBA)	
	Excluding Curve Gain	Including Curve Gain	Excluding Curve Gain	Including Curve Gain
NCA1	38	44	59	67
NCA2	29	34	49	56
NCA3	44	46	66	68
NCA4	39	44	60	67
S1	43	45	67	70
S2	37	40	60	63
I1	34	38	56	62

The data for the southern connection for rail noise are shown in Table 27:

Table 27: Predicted $L_{Aeq, period}$ and L_{Amax} Rail Noise Levels – Southern connection

Receiver	Predicted Level (dBA)		Predicted Level L_{max} (dBA)	
	Excluding Curve Gain	Including Curve Gain	Excluding Curve Gain	Including Curve Gain
NCA1	38	44	59	67
NCA2	29	34	49	56
NCA3	45	51	72	81
NCA4	38	44	58	67
S1	41	47	64	72
S2	36	41	57	64
I1	34	38	56	62

The data shown in Tables 26 and 27 have been used to characterise the health risk associated with exposure to noise from the operations at the Proposal site. The details of the noise modelling is contained within the Noise and Vibration Assessment (Wilkinson-Murray, 2015).

5.3 Risk Characterisation

The WHO has established guidelines for community noise to protect against the key health outcomes described in Section 5.1. The relevant WHO guidelines are summarised in Table 28:

Table 28: WHO Guidelines for Community Noise

Specific Environment	Critical health effect(s)	L_{Aeq} [dB(A)]	Time base [hours]	L_{Amax} fast [dB]
Outdoor Living area	Serious annoyance, daytime and evening Moderate annoyance , daytime and evening	55 50	16 16	- -
Dwelling, indoors Inside bedrooms	Speech intelligibility & moderate annoyance, Daytime & evening Sleep disturbance, night time	35 30	16 8	 45
Outside bedrooms	Sleep disturbance, window open (outdoor values)	45	8	60
School class rooms & pre-schools, Indoors	Speech intelligibility, disturbance of information extraction, Message communications	35	During class	-
Pre-school bedrooms, indoor	Sleep disturbance	30	Sleeping-time	45
School, playground outdoor	Annoyance (external source)	55	During play	-

The purpose of the risk characterization is to estimate potential risks associated with exposure to noise form the Stage 1 Proposal. For the assessment of health effects where there is a known threshold for effect, the predicted noise level for each averaging period is compared to the health based guideline values as set out in Table 28. The ratio of the predicted level to the guideline is termed the hazard quotient (HQ):

HQ = predicted noise level / health based guideline

The hazard quotients are estimated for each of the averaging periods relevant to the guidelines. The WHO community noise guidelines apply to total noise, including background, not just the increment from a particular source. Background noise levels for the suburbs in proximity to the SIMTA Stage 1 site are shown in Table 24.

Table 29 shows that resultant Hazard Quotients for operational noise associated with the Stage 1 Proposal for each of the sensitive locations assessed in the Noise and Vibration Assessment (Wilkinson-Murray, 2015).

Table 29: Hazard Quotients for Operational Noise for key Health Outcomes

	Hazard Index			
OPERATIONS	**Annoyance**	**Sleep Disturbance**		**Cognitive Function**
Sensitive Receptor	**LAeq**	**LAeq**	**LAmax**	**LAeq**
NCA1 Wattle Grove, south of Anzac Road	0.7	0.7	0.8	0.9
NCA2 Wattle Grove, north of Anzac Road	0.4	0.4	0.6	0.6
NCA3 Casula	0.7	0.7	0.8	0.9
NCA4 Glenfield	0.5	0.6	0.7	0.7
S1 All saints Senior College	0.6	0.7		0.9
S2 Casula Powerhouse	0.6	0.6		0.8
I1 DNSDC	0.5	0.6		0.7

As can be seen from Table 29, all hazard quotients are less than 1 indicating that the risk from the operational noise from the Stage 1 site alone does not pose an unacceptable risk. Hazard quotients less than 1 are considered to be an acceptable level of risk (enHealth, 2012).

Table 30 shows the hazard quotients for the existing noise levels (shown in Table 24).

Table 30: Hazard Quotients for Existing Noise

Receiver Catchment	Annoyance	Sleep Disturbance	Cognitive Function
NCA1	0.9	1	1.1
NCA2	0.9	1	1.1
NCA3	1.1	1.2	1.3
NCA4	1	1.2	1.2

Sleep disturbance has been assessed using the night time noise levels while both annoyance and cognitive function have been assessed against the measured daytime levels. The daytime noise levels were adjusted by 10dB for assessment against the WHO guideline to account for the attenuation of noise by structures. The WHO guideline applies indoors for this outcome while the noise measurements were taken outdoors. The data in Table 30 show that the existing noise levels in the suburbs surrounding the SIMTA site already exceed the hazard quotient of 1. The existing levels are higher than those predicted for operational noise from SIMTA Stage 1.

As some of the hazard quotients shown in Table 29 are close to 1 it is likely that with the addition of background noise there may be some hazard quotients that exceed 1. The results shown in Table 29 indicate that actions should be implemented to minimise noise impacts from the site to ensure that the health based criteria are not exceeded. It should be noted that a hazard quotient greater than

1 does not necessarily mean that adverse health effects will be observed, it just means that there is an increased risk of effects occurring. The Noise and Vibration Assessment (Wilkinson Murray, 2015) provides a discussion on best practice measures that would be implemented to reduce from the operations at the Stage 1 Proposal.

Tables 31 and 32 show the hazard quotients for the rail noise from the northern and southern connections, respectively. Hazard quotients ≥ 1 are shown in bold. Note that the noise levels relevant for cognitive function in children have been adjusted by 10dB to account for the applicability of the WHO criteria indoors. The predicted noise levels have been modelled for outdoor receptors.

Table 31: Hazard quotients for Rail Noise Northern Connection

	Hazard Index without curve				Hazard Index with curve			
Rail Noise North	**Annoyance**	**Sleep Disturbance**		**Cognitive Function**	**Annoyance**	**Sleep Disturbance**		**Cognitive Function**
Sensitive Receptor	**LAeq**	**LAeq**	**LAmax**	**LAeq**	**LAeq**	**LAeq**	**LAmax**	**LAeq**
NCA1 Wattle Grove, south of Anzac Road	0.8	0.8	**1.0**	0.8	0.9	**1.0**	**1.1**	**1.3**
NCA2 Wattle Grove, north of Anzac Road	0.6	0.6	0.8	0.5	0.7	0.8	0.9	**1.0**
NCA3 Casula	0.9	**1.0**	**1.1**	**1**	0.9	**1.0**	**1.1**	**1.3**
NCA4 Glenfield	0.8	0.9	**1.0**	0.8	0.9	**1.0**	**1.1**	**1.3**
S1 All saints Senior College	0.9	**1.0**	**1.1**	0.9	0.9	**1.0**	**1.2**	**1.3**
S2 Casula Powerhouse	0.7	0.8	**1.0**	0.7	0.8	0.9	**1.1**	**1.1**
I1 DNSDC	0.7	0.8	0.9	0.7	0.8	0.8	**1.0**	**1.1**

Table 32: Hazard Quotients Rail Noise for Southern Connection

	Hazard Index without curve				Hazard Index with curve			
Rail Noise South	**Annoyance**	**Sleep Disturbance**		**Cognitive Function**	**Annoyance**	**Sleep Disturbance**		**Cognitive Function**
Sensitive Receptor	**LAeq**	**LAeq**	**LAmax**	**LAeq**	**LAeq**	**LAeq**	**LAmax**	**LAeq**
NCA1 Wattle Grove, south of Anzac Road	0.8	0.8	**1.0**	0.8	0.9	**1.0**	**1.1**	**1**
NCA2 Wattle Grove, north of Anzac Road	0.6	0.6	0.8	0.5	0.7	0.8	0.9	0.7
NCA3 Casula	0.9	**1.0**	**1.2**	**1**	**1.0**	**1.1**	**1.4**	**1.2**
NCA4 Glenfield	0.8	0.8	**1.0**	0.8	0.9	**1.0**	**1.1**	0.8
S1 All saints Senior College	0.8	0.9	**1.1**	0.9	0.9	**1.0**	**1.2**	**1**
S2 Casula Powerhouse	0.7	0.8	**1.0**	0.7	0.8	0.9	**1.1**	0.9
I1 DNSDC	0.7	0.8	0.9	0.7	0.8	0.8	**1.0**	0.8

As can be seen from Tables 31 and 32 there are some hazard quotients that are greater than 1 for sleep disturbance, especially with L_{Aeqmax}, and cognitive function. These values only marginally exceed 1 which indicates that the noise from the rail noise associated with the operations from the Stage 1 Proposal may result in a small increase in the risk of the health outcomes in the local community. As discussed in the Noise and Vibration Impact Assessment (Wilkinson-Murray, 2015) the noise predictions for rail movements does not include the noise from the existing rail movements in the local area or background noise. The best practice review conducted as part of the Noise and Vibration Assessment recommends a range of options that can be implemented to reduce noise levels from the rail movements. The reduction in noise through the implementation of these measures will result in reducing the risk to the health of the local community.

The combined night time $L_{Aeq,\ period}$ operational and rail noise levels associated with the Stage 1 Proposal are shown in Table 33 together with the combined background and project noise:

Table 33: Cumulative Noise Levels for Stage 1Proposal

Receiver Catchment	Operational noise (dB)	Rail (excluding squeal) (dB)	Cumulative (Operations + Rail) (dB)	Total (cumulative + existing) (dB)	Increase over existing (dB)
NCA1	33	38	39	47	1
NCA2	20	29	30	45	0
NCA3	33	44	44	55	0
NCA4	25	39	39	53	0

The cumulative night time $L_{Aeq,\ period}$ noise levels from both operational and rail noise activities presented above are the logarithmic sum of the highest predicted levels from the respective activities in each receiver catchment. It should be noted that the locations within each catchment where the highest predictions for operational and rail noise levels are typically not coincident. Therefore, the addition of the highest predicted levels as described above is conservative. The data shown in Table 33 shows that the combined noise from the Stage 1 operations and associated rail noise has a minimal impact on existing noise levels in the surrounding suburbs. The worst case increase in noise is 1dB over existing noise which would not be detected in the local community.

5.3.1 Regional Impacts

There was no assessment undertaken in the Noise and Vibration Assessment (Wilkinson Murray, 2015) of the impact of the noise from the Stage 1 proposal on regional noise. Therefore a quantitative assessment of the associated health risk cannot be undertaken.

The Noise and Vibration Assessment (Wilkinson Murray, 2015) has shown that the impact of the Stage 1 operations and associated rail noise would have a minor impact on noise levels in the local area. The data shown in Table 33 show that the combined impact of the Stage 1 operations and rail noise together with the existing background has a minimal impact on noise in the local area. Given that the local impacts are insignificant, the impact on regional noise will also be insignificant. Based on this the associated health risk would also be insignificant.

5.3.2 Cumulative Impacts

An assessment of the cumulative impacts from the Stage 1 Proposal and the MIC Proposal Early Works was undertaken as part of the noise impact assessment (Wilkinson Murray, 2015). This assessment was undertaken taking into account the most likely scenario where the cumulative noise impacts could

occur. The most likely scenario where sensitive receivers could experience cumulative impacts from the Stage 1 site and the MIC Proposal would be during concurrent operational activities within the Stage 1 site (at 250,000 TEU throughput per annum) and Early Works construction activities within the MIC site.

Predicted construction noise levels at nearby residential receivers during Early Works on the MIC site are presented in Chapter 12 of the *Moorebank Intermodal Terminal Environmental Impact Statement* and are reproduced in Table 34 below.

Table 34 Predicted Construction Noise Levels – MIC Proposal Early Works

Construction Activity	**Maximum Predicted Noise Levels, dBA L_{Aeq}**		
	Casula	Wattle Grove	Glenfield
Heavy vehicles within the main MIC site	30-42	29-36	30-38
Service utility terminations and diversions	29-41	28-35	29-37
Lifting	24-36	23-30	24-31
Landscaping	32-44	31-38	32-40

It is understood that the majority of Early Works construction activities for the MIC Proposal are to be conducted within standard construction hours (Wilkinson Murray, 2015). Therefore, the cumulative impact assessment is based on daytime noise goals.

Table 35 presents cumulative $L_{Aeq, period}$ noise levels at nearby receivers during the daytime. These levels are the logarithmic sum of the predicted daytime $L_{Aeq, period}$ operational noise levels from the Stage 1 site, and the maximum values presented above in Table 34. The hazard quotients for the cumulative impacts is also included in Table 35:

Table 35 Predicted Cumulative $L_{Aeq, period}$ Noise Levels

Receiver	**Predicted Noise Levels**			**Hazard Quotients**	
	Stage 1 Operations	**MIC Early Works**	**Cumulative**	**Annoyance**	**Cognitive Function**
NCA1	31	38	39	0.7	0.8
NCA2	<20	*	<20	<0.4	<0.3
NCA3	31	44	44	0.8	1
NCA4	23	40	40	0.7	0.9
S1	30	44	44	0.8	1
S2	27	44	44	0.8	1

I1	25	*	25	0.5	0.4

*no significant contribution from MIC site

The data in Table 35 shows that the hazard quotients for the health outcomes associated with daytime noise, annoyance and cognitive development in children, are equal to or less thanI1 at all receivers. This indicates that cumulative $L_{Aeq,\ period}$ noise levels from Stage 1 operations and MIC Proposal Early Works will not pose a risk to the health of the local community.

5.3.3 Summary

The health risk assessment for noise has shown that there is potentially a small increase in risk of sleep disturbance associated with the rail noise. The predicted noise levels used in this assessment have been derived without mitigation measures considered and represent the worst case exposures.

The results of the HRA for noise has shown that the incremental operational noise from the Stage 1 Proposal generally meet the health guidelines although there are some exceedances for the rail noise associated with the Proposal especially for the guidelines for sleep disturbance. This is shown by the resultant hazard quotients being just greater than 1 in some cases. A hazard quotient greater than 1 does not imply that an impact on health will occur just that the risk of such an effect occurring is increased. The results for existing (background) noise in the area surrounding the Stage 1 site indicate that existing noise levels already exceed the health guidelines. An assessment of the combined impact of the noise from the Stage 1 operations and associated rail noise and existing background shows that the Stage 1 Proposal does not significantly change the existing noise levels in the local area with only a 1dB increase predicted for one receiver. For all other locations no difference in noise levels was predicted. The cumulative impact of noise from the Stage 1 Proposal and the MIC Proposal Early Works show that all hazard quotients are less than or equal to 1 for the relevant health outcomes indicating that the combined operations will not pose a risk to the health of the local community.

The results for the Proposal associated noise alone, especially the rail noise, indicate that management actions should be considered to minimise the impact on the local community. The implementation of measures included in the Best Practice Review for noise will result in a reduction in noise levels, mitigating the potential risk to the health of the community.

6 CONCLUSIONS

A Screening Health Impact Assessment (HIA) has been conducted for the Stage 1 Proposal. The HIA has considered the issues raised by the community through consultation for the Concept Plan Approval and has examined the potential impacts of the Stage 1 Proposal operation on the local community.

The Proposal site is surrounded by the suburbs of Casula, Wattle Grove, Glenfield and Moorebank in south western Sydney. A review of the demographics of the population and the baseline health status has found that there are no significant differences between the indicators within these communities and the rest of Sydney and NSW. There are no underlying health issues that would make these communities more vulnerable to the effects of environmental factors, such as air pollution or noise from the Proposal's operations than the rest of Sydney.

A health risk assessment (HRA) has been conducted to assess the air quality and noise impacts on the health of the local community. For the Air Quality Assessment a range of health outcomes were investigated including increases in mortality and morbidity such as hospital admissions as well as increases in cancer risk due to exposure to air toxics and diesel emissions. The results of the HRA found that the increase in risk due to air pollution from the operations of the Proposal are low and in most cases are negligible. The cancer risk form the air toxics are well below acceptable risk level set by international agencies. The implementation of best practice measures as outlined in the Air Quality Assessment report will lead to further reductions in air pollution levels and the associated health risks. This has been demonstrated through the reduction in cancer risk from diesel emissions by the implementation of some of the measures. These measures include the move from diesel powered reach stackers to electrified gantry cranes for container handling and the implementation of strategies to reduce the idling times of trucks and locomotives.

The noise HRA has investigated the impact of noise from the operations of the Proposal including rail noise on sleep disturbance and cognitive development in children using the WHO community noise guidelines. The noise from the operations of the Proposal meets the WHO criteria. There are some small exceedances of the sleep disturbance criteria from the rail noise however with the implementation of the best practice measures outlined in the Noise and Vibration Impact Assessment, these exceedances will be minimised and the risk to the local community reduced. As discussed in the Noise and Vibration Assessment (Wilkinson Murray, 2015) it has been recommended that friction modifying agents are applied to the Rail link to mitigate rail squeal noise and that the extent and frequency of application of friction modifiers should be developed during detailed design of the Rail link and should be implemented prior to the commencement of rail operations. Given that the rail squeal noise is the major contributor to the hazard quotients for sleep disturbance exceeding 1, reduction in rail squeal will lead to a reduction in the health risk from noise associated with the rail noise.

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