

Moorebank Precinct East -Stage 2 Proposal

Human Health Risk Assessment





SYDNEY INTERMODAL TERMINAL ALLIANCE

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MOOREBANK PRECINCT EAST STAGE 2 HUMAN HEALTH RISK ASSESSMENT



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EXECUTIVE SUMMARY

Introduction

A Health Risk Assessment has been prepared to support the Environmental Impact Statement for approval of the Moorebank Precinct East (MPE) Stage 2 Proposal (the Proposal), comprising warehousing and distribution facilities on the MPE site.

Overall study approach

A Health Risk Assessment (HRA) uses information about pollutants to estimate a theoretical level of risk for people who might be exposed to defined levels of these substances. The objective of this HRA is to assess potential health risk posed by the air emissions and noise on the surrounding community.

The HRA process comprises five components: issues identification, exposure assessment, toxicity assessment, risk characterisation, and uncertainty assessment. The approach to this HRA was in accordance with approved Australian guidance for performing risk assessments, in particular:

- Health Impact Assessment A Practical Guide Centre for Health Equity Training, Research and Evaluation (CHETRE, 2007).
- Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth, 2012a).

It is noted that a Health Impact Assessment (HIA) has not been conducted for the Proposal. HIA has previously been undertaken for the MPE Stage 1 Project (Pacific Environment, 2015) and MPW Concept Plan Approval (EnRisks 2014b). The community consultation underpinning these previous HIA, and the conclusions, remain applicable and relevant to the Proposal.

Overview of the air quality health risk assessment

The focus of the air quality HRA was on the health impacts of emissions from the operational phase of the Proposal. The key air pollutants evaluated in the local air quality assessment were considered as chemicals of potential concern (COPCs) and inhalation of air was the only exposure pathway evaluated.

The human receptors of concern included commercial/industrial workers, residents, school or day care students and recreational users located in the suburbs of Casula, Wattle Grove, Glenfield, and Moorebank. Annual average ground level concentrations (GLCs) of COPCs emitted from operation of the Proposal were predicted by air modelling in the local air quality assessment. A cumulative Proposal scenario was also considered for concurrent operation of the Proposal, the MPE Stage 1 Project and the MPW Stage 2 Project.

Health endpoints and associated exposure-response relationships were previously approved by NSW Health as part of the consultation undertaken for MPE Stage 1, and are therefore also adopted for this HRA. The air quality HRA evaluated a range of health endpoints associated with the key air pollutants, including increases in mortality and morbidity as well as excess lifetime cancer risks.

Summary of air quality HRA results

Short-term and long-term exposure to PM_{10} and $PM_{2.5}$ result in low health impacts in the surrounding communities (i.e., fewer than one increased case per year of premature mortality, hospital admissions, and emergency department visits associated with cardiovascular and respiratory diseases or asthma). Short-term and long-term exposure to NO_x , following adjustment for the fraction attributable to NO_2 , result in low health impacts in the surrounding communities (i.e. less than or equal to one increased case per year of premature mortality, hospital admissions, and emergency department visits associated with cardiovascular and respiratory diseases or asthma). Short-term exposure to SO_2 and CO results in negligible impacts in the surrounding communities (i.e. orders of magnitude below the acceptable risk of one increased case per year, for premature mortality, hospital admissions, and emergency department visits associated with cardiovascular and respiratory diseases or asthma).

Excess lifetime cancer risks for residents/school students, commercial/industrial workers, and recreational populations within the study area are below levels of acceptable risk (i.e. within or below the established acceptable cancer risk range of 10^{-6} to 10^{-4}).

In summary, there are no significant adverse health effects expected in relation to short-term and long-term exposure to key air pollutants associated with the operation of the Proposal alone, and also a cumulative assessment scenario.

Overview of health risk assessment for noise

The main health effects associated with environmental noise include cardiovascular disease, cognitive impairment, sleep disturbance, tinnitus, annoyance, and hearing impairment.

The exposure data for the noise HRA were obtained from the Noise and Vibration Impact Assessment (Wilkinson-Murray, 2016). The risk characterisation was conducted by comparing the predicted noise levels to the corresponding health-based World Health Organisation guideline values for annoyance, sleep disturbance and cognitive impairment (WHO, 1999).

The noise from both operation of the Proposal and cumulative assessment scenario meets the WHO community noise guidelines at all residential receivers. A HQ greater than 1 was predicted for annoyance and cognitive impairment at the nearest industrial receiver, however, the HQs for existing ambient noise already exceed 1 for annoyance and cognitive impairment. Similarly, although total noise exceed WHO community noise guidelines, the existing ambient noise levels alone are already above these guidelines and on this basis the Proposal related noise is expected to have a minimal impact on the local residential area.

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Calculation of Increased Annual Incidence for Mortality and Morbidity Endpoints

Appendix 3

Calculation of Excess Lifetime Cancer Risk

ACRONYMS AND ABBREVIATIONS

ABS	Australian Bureau of Statistics	
AQIA	Air Quality Impact Assessment	
BaP	Benzo(a)pyrene	
Cal/EPA	California Environmental Protection Agency	
CCME	Canadian Council of Ministers for the Environment	
CO	Carbon Monoxide	
COPC	Chemical of Potential Concern	
CSM	Conceptual Site Model	
dB	decibel	
DEMS	Diesel Exhaust in Miners Study	
DoD	Department of Defense	
DPM	Diesel Particulate Matter	
EIS	Environmental Impact Statement	
EP&A	Environmental Planning and Assessment	
EPBC	Environmental Protection Biodiversity Conservation	
EPC	Exposure Point Concentration	
EPHC	Environment Protection and Heritage Council	
GFA	Gross Floor Area	
GLC	Ground Level Concentration	
HEI	Health Effects Institute	
HIA	Health Impact Assessment	
HQ	Hazard Quotient	
HRA	Health Risk Assessment	
IARC	International Agency for Research on Cancer	
IMEX	Import Export	
IMT	Intermodal Terminal	
LGA	Local Government Area	
m ²	square meter	
m ³	cubic meter	
MAF	Modelling Adjustment Factor	
MIC	Moorebank Intermodal Company	
MPE	Moorebank Precinct East	
MPW	Moorebank Precinct West	
NCA	Noise Catchment Area	
NEPC	National Environment Protection Council	
NEPC	National Environment Protection Measure	
NHMRC	National Health and Medical Research Council	
NO ₂	Nitrogen Dioxide	
NSW	New South Wales	
OEHHA	Office of Environmental Health Hazard Assessment	
OEH	Office of Environment and Heritage	
PAH	Polycyclic Aromatic Hydrocarbon	
PM	Particulate Matter	
RBL	Rating Background Level	
REMM	Revised Environmental Mitigation Measure	
SEAR	Secretary's Environmental Assessment Requirement	
SIMTA	Sydney Intermodal Terminal Alliance	
SIMTA SO ₂	Sulfur Dioxide	
SPR	Source-Pathway-Receptor	
SSD	State Significant Development	
SSFL	Southern Sydney Freight Line	
SWSLHD	South Western Sydney Local Health District	

TEF	Toxicity Equivalent Factor	
TEQ	Toxicity Equivalent	
TEU	Twenty-Foot Equivalent Unit	
μg	microgram	
μm	micrometer	
URF	Unit Risk Factor	
USEPA	United States Environment Protection Agency	
VOC	Volatile Organic Compound	
WHO	World Health Organization	

1. INTRODUCTION

Concept Plan Approval (MP 10_0193) for an intermodal terminal (IMT) facility at Moorebank, NSW (the Moorebank Precinct East Project (MPE Project) (formerly the SIMTA Project)) was received on 29 September 2014 from the NSW Department of Planning and Environment (DP&E). The Concept Plan for the MPE Project involves the development of an IMT, including a rail link to the Southern Sydney Freight Line (SSFL) within the Rail Corridor, warehouse and distribution facilities with ancillary offices, a freight village (ancillary site and operational services), stormwater, landscaping, servicing, associated works on the eastern side of Moorebank Avenue, Moorebank, and construction or operation of any part of the project, which is subject to separate approval(s) under the *Environmental Planning and Assessment Act 1979* (EP&A Act).

This Air Quality Impact Assessment (AQIA) forms part of the Environmental Impact Statement (EIS) seeking approval, under Part 4, Division 4.1 of the EP&A Act, for the construction and operation of Stage 2 of the MPE Project (herein referred to as the Proposal) under the Concept Plan Approval for the MPE Project, being the construction and operation of warehouse and distribution facilities.

This EIS has been prepared to address:

- The Secretary's Environmental Assessment Requirements (SEARs) (SSD 16-7628) for the Proposal, issued by NSW DP&E on 27 May 2016.
- The relevant requirements of the Concept Plan Approval MP 10_0913 dated 29 September 2014 (as modified).
- The relevant requirements of the approval under the *Environment Protection and Biodiversity Conservation Act 1999* (EPBC Act) (No. 2011/6229, granted in March 2014 by the Commonwealth Department of the Environment (DoE)) (as relevant).

This AQIA also gives consideration to the MPE Stage 1 Project (SSD 14-6766) including the mitigation measures and conditions of consent as relevant to this Proposal.

This AQIA has been prepared to provide a complete assessment of the potential air quality impacts associated with the construction and operation of the Proposal. This AQIA proposes measures to mitigate these issues and reduce any unreasonable impacts on the environment and surrounding community.

1.1 Report purpose

This report presents a human health risk assessment (HRA) to address the potential health risks from exposure to air emissions and noise from the Proposal. The report supports the Environmental Impact Statement (EIS) for the Proposal and has been prepared as part of a State Significant Development (SSD) Application for which approval is sought under Part 4, Division 4.1 of the EP&A Act. This report has been prepared to address:

- The Secretary's Environmental Assessment Requirements (SEARs) (SSD 16-7628) for the Proposal, issued by NSW DP&E on 27 May 2016.
- The relevant requirements of Concept Plan Approval MP 10_0913 dated 29 September 2014 (as modified).
- The relevant requirements of the approval under the *Environment Protection and Biodiversity Conservation Act 1999* (EPBC Act) (No. 2011/6229, granted in March 2014 by the Commonwealth Department of the Environment (DoE)) (as relevant).

The SEARs and the Concept Plan Conditions of Approval and Statement of Commitments relevant to this report, and the section of this report where they have been addressed, are provided in **Table 1-1** and **Table 1-2**.

Table 1-1: Summary of SEARs for human health risk			
General requirements	Where addressed in this		
	report		
a health risk assessment of local and regional impacts associated with the development, including those health risks associated with relevant key issues. The assessment should be undertaken with reference to the Centre for Health Equity Training, Research, an Evaluations' practical guide to impact assessment (August 2007) and shall include:	This report presents a health risk assessment for local impacts associated with air and noise emissions. Regional impacts have		
 a discussion of the known potential developments in the local region; an assessment of the impact on the environmental values of public health; and an assessment of local and regional impacts including health risks. 	been previously considered for the Concept Plan Approval for both the MPW and MPE sites. Refer Section 3 for methodology.		

Table 1-2: Concept Plan conditions of approval or statement of commitments

Statement of commitments	Where addressed in this report		
 The Proponent will undertake further health impact assessments for lodgement with each of the detailed planning applications for the three major stages of the development, including: Discussion of the known and potential developments in the local region Assessment of the impact on the environmental values of public health. Assessment of local and regional impacts including health risks Health impact assessments will be undertaken with reference to the Centre for Health Equity Training, Research, and Evaluations' practical guide to impact assessment (August 2007) 	This report presents a health risk assessment for local impacts associated with air and noise emissions. Regional impacts have been previously considered for the Concept Plan Approval for both the MPW and MPE sites. Refer Section 3 for methodology.		

1.2 Overview of the Proposal

The Proposal involves the construction and operation of Stage 2 of the MPE Project, comprising warehousing and distribution facilities on the MPE site and upgrades to approximately 1.4 kilometres of Moorebank Avenue between the northern MPE site boundary and 120 metres south of the southern MPE site boundary.

Key components of the Proposal include:

- Warehousing comprising approximately 300,000m² GFA, additional ancillary offices and the ancillary freight village
- Establishment of an internal road network, and connection of the Proposal to the surrounding public road network
- Ancillary supporting infrastructure within the Proposal site, including:
 - Stormwater, drainage and flooding infrastructure
 - Utilities relocation and installation
 - Vegetation clearing, remediation, earthworks, signage and landscaping
 - Subdivision of the MPE Stage 2 site
- The Moorebank Avenue upgrade would be comprised of the following key components:
 - Modifications to the existing lane configuration, including some widening
 - Earthworks, including construction of embankments and tie-ins to existing Moorebank Avenue road level at the Proposal's southern and northern extents
 - Raking of the existing pavement and installation of new road pavement
 - Establishment of temporary drainage infrastructure, including temporary basins and / or swales
 - Raising the vertical alignment by about two metres from the existing levels, including kerbs, gutters and a sealed shoulder
 - Signalling and intersection works
 - Upgrading existing intersections along Moorebank Avenue, including:
 - Moorebank Avenue / MPE Stage 2 access
 - Moorebank Avenue / MPE Stage 1 northern access
 - Moorebank Avenue / MPE Stage 2 central access
 - MPW Northern Access / MPE Stage 2 southern emergency access

The Proposal would interact with the MPE Stage 1 Project (SSD_6766) via the transfer of containers between the MPE Stage 1 IMT and the Proposal's warehousing and distribution facilities. This transfer of freight would be via a fleet of heavy vehicles capable of being loaded with containers and owned by SIMTA. The fleet of vehicles would be stored and used on the MPE Stage 2 site, but registered and suitable for on-road use. The Proposal is expected to operate 24 hours a day, seven days per week.

To facilitate operation of the Proposal, the following construction activities would be carried out across and surrounding the Proposal site (area on which the Proposal is to be developed):

- Vegetation clearance
- Remediation works
- Demolition of existing buildings and infrastructure on the Proposal site
- Earthworks and levelling of the Proposal site, including within the terminal hardstand
- Drainage and utilities installation
- Establishment of hardstand across the Proposal site, including the terminal hardstand
- Construction of a temporary diversion road to allow for traffic management along the Moorebank Avenue site during construction (including temporary signalised intersections adjacent to the existing intersections) (the Moorebank Avenue Diversion Road)
- Construction of warehouses and distribution facilities, ancillary offices and the ancillary freight village
- Construction works associated with signage, landscaping, stormwater and drainage works.

Construction works associated with signage, landscaping, stormwater and drainage works. The Proposal would operate 24 hours a day, 7 days a week.

An overview of the Proposal is shown in **Figure A1-1 (Appendix 1)**.

1.3 Key terms relevant to the proposal

Table 1-3 provides a summary of the key terms relevant to the Proposal, which are included throughout this report.

Table 1-3: Summary of key terms used throughout this document Terms			
Term	Definition		
General terms			
The Moorebank Precinct	Refers to the whole Moorebank intermodal precinct, i.e. the MPE site and the MPW site		
Moorebank Precinct West (MPW) Project	The MPW Intermodal Terminal Facility as approved under the MPW Concept Plan Approval (SSD_5066) and the MPW EPBC Approval (No.		
(formerly the MIC Project)	2011/6086).		
Moorebank Precinct West (MPW) site (formerly the MIC site)	The site which is the subject of the MPW Concept Plan Approval, MPW EPBC Approval and MPW Planning Proposal. The MPW site does not include the rail link as referenced in the MPW Concept Plan Approval or MPE Concept Plan Approval.		
Moorebank Precinct East (MPE) Concept Plan Approval (formerly the SIMTA Concept Plan Approval)	MPE Concept Plan Approval (SSD_0193) granted by the NSW Department of Planning and Environment on 29 September 2014 for the development of former defence land at Moorebank to be developed in three stages; a rail link connecting the site to the Southern Sydney Freight Line, an intermodal terminal, warehousing and distribution facilities and a freight village.		
Moorebank Precinct East (MPE) Project (formerly the SIMTA Project)	The MPE Intermodal Terminal Facility, including a rail link and warehouse and distribution facilities at Moorebank (eastern side of Moorebank Avenue) as approved by the Concept Plan Approval (MP 10_0913) and the MPE Stage 1 Approval (14_6766).		
Moorebank Precinct East (MPE) Site (formerly the SIMTA Site)	Precinct East (MPE) Including the former DSNDC site and the land owned by SIMTA whi subject to the Concept Plan Approval. The MPE site does not include rail corridor, which relates to the land on which the rail link is to be		
Statement of Commitments (SoC)	Recommendations provided in the specialist consultant reports prepared as part of the MPE Concept Plan application to mitigate environmental impacts, monitor environmental performance and/or achieve a positive environmentally sustainable outcome in respect of the MPE Project. The Statement of Commitments have been proposed by SIMTA as the Proponent of the MPE Concept Plan Approval.		
MPE Stage 1 Project-specific te	erms		
MPE Stage 1	Stage 1 (14-6766) of the MPE Concept Plan Approval for the development of the MPE Intermodal Terminal Facility, including the rail link at Moorebank. This reference also includes associated conditions of approval and environmental management measures which form part of the documentation for the approval.		
PE Stage 1 site Includes the MPE Stage 1 site and the Rail Corridor, i.e. the area for which approval (construction and operation) was sought within the Stage 1 Proposal EIS.			

Table 1-3: Summary of key terms used throughout this document			
Term	Definition		
MPE Stage 2 Proposal/ the Proposal	The subject of this EIS; being Stage 2 of the MPE Concept Plan Approval including the construction and operation of 300,000m ² of warehousing and distribution facilities on the MPE site and the Moorebank Avenue upgrade within the Moorebank Precinct.		
MPE Stage 2 site The area within the MPE site which would be disturbed by the MPE 2 Proposal (including the operational area and construction area). MPE Stage 2 site MPE Stage 2 site includes the former DSNDC site and the land own SIMTA which is subject to the MPE Concept Plan Approval. The MPE does not include the rail corridor, which relates to the land on whice rail link is to be constructed.			
The Moorebank Avenue site	The extent of construction works to facilitate the construction of the Moorebank Avenue upgrade.		
The Moorebank Avenue upgrade	Raising of the vertical alignment of Moorebank Avenue for 1.5 kilometres of its length by about two metres, from the northern boundary of the MPE site to approximately 120 metres south of the MPE site. The Moorebank Avenue upgrade also includes upgrades to intersections, ancillary works and the construction of an on-site detention basin to the west of Moorebank Avenue within the MPW site.		
Construction area Extent of construction works, namely areas to be disturbed durin construction of the MPE Stage 2 Proposal (the Proposal).			
Operational area	Extent of operational activities for the operation of the MPE Stage 2 Proposal (the Proposal).		

Table 1-3: Summary of key terms used throughout this document

2. PROPOSAL OVERVIEW

2.1 Regional context

The MPE site, including the Proposal site, is located approximately 27 km south-west of the Sydney Central Business District (CBD) and approximately 26 km west of Port Botany. The MPE site is situated within the Liverpool Local Government Area (LGA), in Sydney's South West subregion, approximately 2.5 km from the Liverpool City Centre.

The MPE site is located approximately 800 m south of the intersection of Moorebank Avenue and the M5 Motorway. The M5 Motorway provides the main road link between the MPE site, and the key employment and industrial areas within Sydney's West and South-Western subregions, the Sydney orbital network and the National Road Network. The M5 connects with the M7 Motorway to the west, providing access to the Greater 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 regional context of the Proposal is shown in **Figure A1-2 (Appendix 1)**.

2.2 Local context

The Proposal site is located approximately 2.5 km south of the Liverpool City Centre, 800 m south of the Moorebank Avenue/M5 Motorway interchange and one kilometre to the east of the SSFL providing convenient access to and from the site for rail freight (via a dedicated freight rail line) and for trucks via the Sydney Motorway Network.

The land surrounding the Proposal site comprises:

- The MPW site, formerly the School of Military Engineering (SME), on the western side of Moorebank Avenue directly adjacent to the MPE site (subject to the MPW Concept Plan Approval), which is owned by the Commonwealth;
- The East Hills Rail Corridor to the south of the MPE site, which is owned and operated by Sydney Trains;
- The Holsworthy Military Reserve, to the south of the East Hills Rail Corridor, which is owned by the Commonwealth; The Boot Land, to the immediate east of the MPE site between the eastern site boundary and the Wattle Grove residential area, which is owned by the Commonwealth.
- The southern Boot Land, to the immediate south of the MPE site between the southern site boundary and the East Hills Rail Corridor, which is owned by the Commonwealth.

Glenfield Waste Services, south-west of the Proposal is proposing to develop a Materials Recycling Facility on land owned by the Glenfield Waste Services Group within the boundary of the current landfill site at Glenfield. The facility is proposed to recycle a maximum of 450,000 tonnes of material per year. The Glenfield Waste Services Proposal is the subject of a DA (SSD_6249) under Part 4, Division 4.1 of the EP&A Act. A number of residential suburbs are located in proximity to the Proposal site. The approximate distances of these suburbs to the MPE Stage 2 site and the Moorebank Avenue site are provided in **Table 2-1**.

Suburb	Distance to MPE Stage 2 site	Distance to Moorebank Avenue site
Wattle Grove	360 m to the north-east	865 m to the north-east
Moorebank	1300 m to the north	1430 m to the north
Casula	820 m to the west	760 m to the west
Glenfield	1830 m to the south-west	1540 m to the south-west

Table 2-1: Distance to residential suburbs from the Proposal site

The closest industrial precinct to the Proposal is at Moorebank, comprising around 200 hectares of industrial development. This area includes (but is not limited to) the Yulong and ABB sites to the south of the M5 Motorway and the Goodman MFive Business Park and Miscellaneous industrial and commercial development to the north of the M5 Motorway. The majority of this development is located to the north of the M5 Motorway between Newbridge Road, the Georges River and Anzac Creek. The Moorebank Industrial Area supports a range of industrial and commercial uses, including freight and logistics, heavy and light manufacturing, offices and business park developments.

There are other areas of industrial development near the Proposal at Warwick Farm to the north, Chipping Norton to the north-east, Prestons to the west and Glenfield and Ingleburn to the southwest. The local context of the Proposal is shown in **Figure A1-3 (Appendix 1)**.

2.3 Construction overview

Construction of the Proposal is proposed to take between 24 and 36 months, commencing in the final quarter of 2017, with the completion of construction in the third quarter of 2019 (should construction take 24 months). The final construction program will depend on the market demand for warehouses to be constructed on the MPE Stage 2 site.

The construction works have been divided into seven 'works periods' which are interrelated and also may overlap, as shown below. Subject to confirmation of construction staging, the order of these construction works periods may shift slightly.

- Works period A pre-construction activities.
- Works period B site preparation activities.
- Works period C construction of the Moorebank Avenue diversion road
- Works period D bulk earthworks, drainage and utilities.
- Works period E pavement works along Moorebank Avenue.
- Works period F warehouse construction and internal fit-out.
- Works period G miscellaneous construction and finishing works.

An indicative construction programme and full description of the activities included in each works period is outlined in the main body of the EIS. Construction works would generally be undertaken during standard daytime construction working hours, being:

- 7 am to 6 pm Monday to Friday
- 8 am to 1 pm Saturday
- No works on Sunday or Public Holidays.

Bulk earthworks activities and construction works to facilitate the Moorebank Avenue upgrade during peak construction periods may be undertaken outside of standard construction hours, but not during the night-time (i.e. 10pm to 7am). An overview of the construction layout is shown in **Figure A1-4 (Appendix 1)**.

2.4 Operations overview

The Proposal involves the construction and operation of Stage 2 of the MPE Project, comprising warehousing and distribution facilities on the MPE site and upgrades to approximately two kilometres of Moorebank Avenue between Anzac Road and 200 metres south of the MPE site.

The Proposal would interact with the MPE Stage 1 Project (SSD_6766) via the transfer of containers between the MPE Stage 1 IMT and the Proposal's warehousing and distribution facilities. The vehicle movements associated with the transfer of containers between the MPE Stage 1 IMT and the Proposal would be within the Proposal site only, and would not impact on the surrounding road network.

The Proposal is expected to operate 24 hours a day, seven days per week.

2.4.1 Warehousing

The Proposal would provide up to 300,000m² of warehousing across the MPE Stage 2 site, with ancillary offices attached. The Proposal would include eight warehouses, which would be up to 21 metres in height and would range in size from 20,350m² to 61,500m². The Proposal would also include some internal fitout of the warehouses, namely the installation of racking and associated services. The Proposal would seek approval for the construction of these warehouses and also the operation of these warehouses by future tenants.

The indicative layout of the warehouses are shown in **Figure A1-4 (Appendix 1)**.

2.4.2 Freight village

A freight village including amenities would be provided on the MPE site as part of the Proposal. The ancillary freight village would be located in the north-west of the Proposal site, directly north of Warehouse 1 and east of Moorebank Avenue. The freight village would include five buildings which would provide for a mixture of retail, commercial and light industrial land uses, with a combined GFA of approximately 8,000m².

2.4.3 Vehicle movements and access

Access to and from the Proposal site would be via the existing DSNDC northern access, to the north of the MPE Stage 1 Project. Site access at this location would allow for vehicular access to warehouse and distribution facilities to enable the direct delivery and dispatch of goods to the warehouses.

Internal roads

The MPE Stage 2 site includes two main internal roads, which provided the main east-west and north-south traffic movements throughout the MPE Stage 2 site. On entering the MPE Stage 2 site, light and heavy vehicles would travel along an east-west oriented internal road (internal road 1). Internal road 1 would connect at its easternmost point to a second north-south oriented internal road (internal road 2).

Internal roads 1 and 2 would connect to three service roads which would provide vehicle access to warehouses, loading docks and car parking.

Internal road 2 would provide for traffic movements along the entire eastern perimeter of the Proposal, and would have a cul-de-sac at both the northern and southern ends to allow vehicles to turn around. The internal roads would be two lanes wide (one lane in each direction) and would be wide enough to accommodate heavy vehicle turning movements, including B-doubles.

Service roads

Three service roads would connect to the internal roads within the MPE Stage 2 site. The service roads would provide access to loading docks at warehouses for heavy vehicles to park and be packed with materials which have been received and stored within the warehouses. Service roads would also enable access to light vehicle parking for users of the warehouses. Each service road would have a cul-de-sac for vehicles to turn around, which would be able to accommodate turning movements of B-doubles.

Service road 1 would connect to internal road 1 via a T-intersection, and would provide access to Warehouse 1, Warehouse 2 and the ancillary freight village. Two additional service roads would connect to internal road 2 via t-intersections; service road 2 would provide access for warehouses 3, 4 and 5, and service road 3 would provide access to warehouses 6, 7 and 8.

Transfer roads

There would be three Transfer roads within the MPE Stage 2 site. These roads would provide connections between the warehouses and the MPE Stage 1 IMT. It is intended that the transfer of freight between the Stage 1 IMT and warehouses would be via an internal fleet of vehicles which would remain on the MPE Stage 2 site and would not use the external road network.

Transfer road 1 would travel mostly along the same path as internal road 1 and provide access between the Stage 1 IMT facility and Warehouses 1, 2 and 3. Transfer road 2 would travel through the centre of the MPE Stage 2 site and would provide access between the Stage 1 IMT facility and Warehouses 4, 5, 6 and 8. Transfer road 3 would travel along the southern boundary of the MPE site, and provide access between the Stage 1 IMT facility and Warehouses 7 and 8.

With the exception of transfer road 1, which travels along the same path as internal road 1, the movement of internal fleet vehicles along transfer roads would be separated from light and heavy vehicles entering and exiting the MPE Stage 2 site to maintain efficiency and to provide for a safe internal road network.

2.4.4 Roadworks – Moorebank Avenue

As part of the Proposal, Moorebank Avenue would be upgraded for about 1.4 kilometres. The Moorebank Avenue upgrade commences from approximately 95 metres south of the northern boundary of the MPE site to approximately120 metres south of the southern MPE site boundary. The Moorebank avenue upgrade is located within the existing Moorebank Avenue road corridor and along the eastern boundary of the MPW site (refer to **Figure A1-1 (Appendix 1)** for extent of works).

The Moorebank Avenue upgrade would be comprised of the following key components:

- Modifications to the existing lane configuration, including some widening
- Signalling and intersection works.
- Raising the vertical alignment by about two metres from the existing levels, including kerbs, gutters and a sealed shoulder

3. APPROACH TO ASSESSMENT

3.1 Health Risk Assessment

A Health Risk Assessment (HRA) uses information about pollutants to estimate a theoretical level of risk for people who might be exposed to defined levels of these substances. HRAs 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 HRA to ensure protection of the public. In a HRA, the most vulnerable people (e.g. children, the sick and elderly) are carefully considered to make sure that all members of the public are protected.

HRA is used extensively as a tool in Australia and overseas to assist in decision making on the acceptability of the risks associated with the presence of contaminants in the environment and for the evaluation of projects with potential risks to the public.

The HRA process comprises five components: issues identification, exposure assessment, toxicity assessment, risk characterisation, and uncertainty assessment. Some of the key factors and questions that are taken into consideration at each of these components include the following:

- **Issue Identification** Identifies issues that can be assessed through a risk assessment and assists in establishing a context for the risk assessment.
- **Exposure Assessment** identifies the groups of people who may be exposed to hazardous agents and quantifies the exposure concentrations.
- **Toxicity 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.
- **Risk Characterisation** provides the quantitative evaluation of potential risks to human health. The characterisation of risk is based on the review of exposure-response relationship and the assessment of the magnitude of exposure.
- **Uncertainty Assessment** identifies potential sources of uncertainty and qualitative discussion of the magnitude of uncertainty and expected effects on risk estimates.

The objective of this HRA was to assess potential health risk posed by air emissions and noise generated from the Proposal on surrounding communities. The HRA was undertaken in accordance with approved Australian guidance for performing risk assessments including:

- enHealth. 2012a. Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards.
- enHealth. 2012b. Exposure Factors Guide.
- National Health and Medical Research Council (NHMRC). 2006. Approach to Hazard Assessment for Air Quality.
- National Environment Protection Council (NEPC). 2011. Methodology for Setting Air Quality Standards in Australia.

Where considered appropriate, guidance from international authorities have been referred to during preparation of the HRA, such as the World Health Organization (WHO) and the United States Environment Protection Agency (USEPA).

Local air quality impacts have been evaluated in detail in an Air Quality Impact Assessment (Ramboll Environ, 2016), referred to in this document as the Air Quality Impact Assessment or AQIA. Local noise impacts have been evaluated in detail in the report "MPE Stage 2 Noise and Vibration Impact Assessment" prepared by Wilkinson-Murray (2016). The HRA has drawn on information presented in AQIA as well as the noise and vibration impact assessment report, and as such, should be read in conjunction with these two reports. The HRA focusses on risk characterisation using the model-predicted air and noise data for the Proposal. A comprehensive discussion of background and toxicity assessment, is provided in previous health risk assessment

reports for the MPE Concept Plan (Toxicos, 2012), the MPE Stage 1 Project (Pacific Environment 2015) and MPW Concept Plan Approval (EnRisks 2014a).

3.2 Health Impact Assessment

A Health Impact Assessment (HIA) has not been conducted for the Proposal. A screening HIA has previously been undertaken for the MPE Stage 1 Project (Pacific Environment, 2015) and MPW Concept Plan Approval (EnRisks 2014b). The community consultation underpinning these previous HIAs, and their conclusions, remain applicable and relevant to the Proposal.

Pacific Environment (2015) considered the issues raised by the community through consultation for the Concept Plan Approval and examined the potential impacts of the MPE Stage 1 Project on the local community. A review of the demographics of the population and the baseline health status 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. The findings of the Social Impact Commentary conducted for the Concept Plan Approval concluded that the development of the MPE Project would provide employment opportunities for the local community and provide opportunities to improve community services and economic benefits in the form of employment and local and regional productivity (Pacific Environment 2015).

EnRisks (2014b) identified a number of potential impacts (both positive and negative) on the health and wellbeing of the local communities (including sensitive receptors). These impacts relate to the economic environment, transport, the natural environment, sustainability, lifestyle, and the social environment. It was concluded that the negative impacts identified can be effectively mitigated through a wide range of measures.

4. HEALTH RISK ASSESSMENT – AIR QUALITY

The air quality HRA has been conducted to evaluate the potential health risks to surrounding communities from exposure to air emissions from the Proposal, with a focus on the operational phase. Emissions to air from the construction sources were not evaluated in this HRA, consistent with previous air quality HRA prepared for the MPE Stage 1 Project (Pacific Environment, 2015). Construction phase impacts for the Proposal would be temporary, able to be managed with the implementation of standard mitigation measures, and are demonstrated in the AQIA to comply with the relevant air quality standards.

The air quality data used in the HRA has been generated through air modelling included in the AQIA. Prior to the evaluation of health risk, the existing health of the local populations and the existing air quality in the local areas were evaluated in the baseline assessment.

4.1 Baseline assessment

The air quality HRA has focused on key air pollutants associated with emission sources from the operation of the Proposal. For each pollutant, there are a large number of other sources in the local area that have the potential to affect the health of local communities, including other combustion sources, other local construction/earthworks, and personal exposures (such as smoking). The health of the community is also influenced by a complex range of interacting factors including age, socio-economic status, social capital, behaviours, beliefs and lifestyle, life experiences, country of origin, genetic predisposition, and access to health and social care.

It is necessary therefore to review existing health statistics and air quality for the local areas surrounding the proposed site and compare them to the greater Sydney area and NSW, prior to an evaluation of the health impacts attributed to the Proposal.

4.1.1 Surrounding area and population

The Proposal site is located within the Liverpool LGA in the Sydney south-western region. The study area considered within the AQIA encompasses the local air shed in which the construction and operation of the Proposal would likely have influence.

It includes the proposed MPE site and surrounding land zoned for commercial, industrial, and Defence purposes. Surrounding residential suburbs include Casula, Moorebank, Glenfield and Wattle Grove, comprising low to medium density housing (**Figure 4-1**).

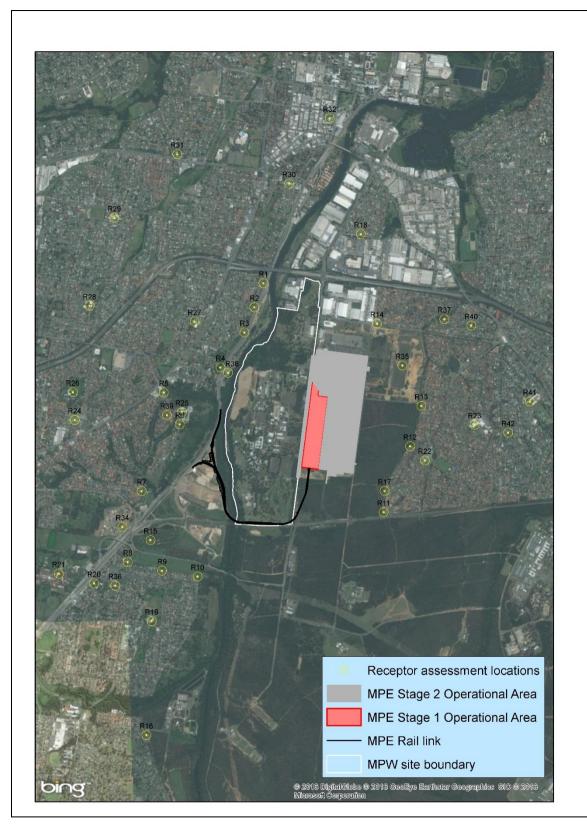


Figure 4-1: Surrounding residential suburbs and study area

4.1.2 Population statistics

Population statistics for the surrounding suburbs of Casula, Glenfield, Wattle Grove, and Moorebank were obtained from the Australian Bureau of Statistics (ABS) for the census year 2011 and are summarised in **Table 4.1**.

Table 4.1: Summary of population statistics							
Lesstien	Total	% of population by key age group					
Location	population	< 5 years	5-14 years	15-64 years	65+ years	30+ years	
Casula	14,696	7.9	15	67	10	49	
Wattle Grove	8,192	8.7	18	69	5.2	45	
Moorebank	7,595	8.4	13	66	13	60	
Glenfield	7,558	6.6	12	67	14	67	
Sydney South West	360,166	7.1	15	68	11	50	
Greater Sydney	4,391,674	6.8	12	68	13	60	
Rest of NSW (excluding Sydney)	2,512,949	6.3	13	63	18	63	

Based on this general population data, the population composition in the suburbs of Moorebank, Casula, and Glenfield are largely similar to Sydney Southwest and Greater Sydney, while Wattle Grove is characterised by a lower proportion of people aged 65 years and over, reflecting the presence of a higher percentage of military families in this suburb (EnRisk 2014a).

According to the Liverpool Community Health Profile (South Western Sydney Local Health District [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 4-2**, and the most significant population growth is predicted for people in age groups less than 69 years of age.

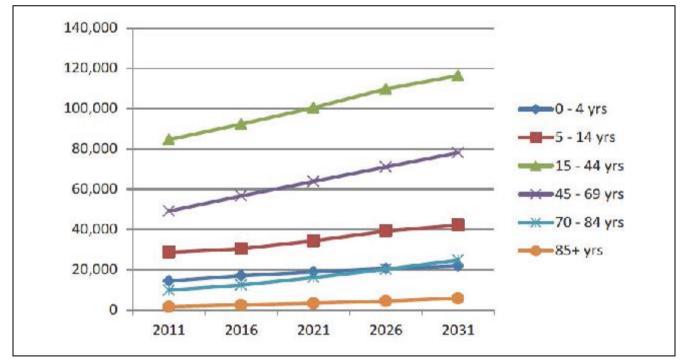


Figure 4-2: Predicted population growth in Liverpool LGA (Source: SWSLHD 2014) (Source: SWSLHD 2014)

4.1.3 Existing health of population

Most of the health indicators presented in this report are not available for each of the smaller suburbs surrounding the proposed MPE site. Health indicators are only available from a mix of larger areas (that incorporate the study area) that comprise the Liverpool LGA, the larger Sydney South West Area, Greater Sydney and NSW. The health statistics for these larger areas are assumed to be representative of the smaller population located in the vicinity of the Proposal.

Information on the incidence of health-related behaviours, the key mortality indicators and hospitalisations, and the prevalence and management of asthma in children in local areas, as well as in Greater Sydney and NSW, was discussed in previous HRA reports (Pacific Environment, 2015; EnRisks, 2014a).

Table 4.2 presents the data on baseline health incidence for the local population as well as in Sydney South West, Greater Sydney and NSW. The data in **Table 4.2** indicates that the baseline health status of the local population does not differ significantly from the data for NSW as a whole.

Based on the available information, there are no underlying health issues that would make the local communities more vulnerable to the effects of environmental factors, such as air pollution or noise from the Proposal, when compared with the rest of Sydney and NSW.

Health indicator	Incidence for population (rate per 100,000 population)					
	Liverpool LGA	Sydney South West Area	Greater Sydney	NSW		
Mortality	-					
All causes-all ages	556 ^a	543 ^b	587 ^c	529	b	
All causes-30+ years				1065	b	
Cardiovascular disease- all ages ¹	162 ^a	160 ^a		155	b	
Cardiovascular disease- 30+ years ²				299	b	
Cardiopulmonary 30+ years				490	d	
Ischemic heart disease 30+ years ³	71 ^a	72 ^a		67	b	
Respiratory disease all ages		52 ^e		50	f	
Respiratory disease 30+ years ⁴		52 ^e		50	f	
Lung cancer 30+ years ⁵	38 ^g	36 ^f		35	f	
Hospital Admissions	-		•			
Respiratory disease 65+ years				4476	h	
Respiratory disease 15-64 years ⁶				899	h	
Cardiac disease 65+ years ⁷				9159	h	
Cardiovascular disease 65+ years ¹				9159	h	
Pneumonia and bronchitis 65+ years ⁸				1236	h	
Ischemic heart disease 65+ years ⁹		2805 ^h		3331	h	
COPD 65+ years	1678 ⁱ	1482 ^h	1194 ^j	1489	h	
Asthma	·					
ED Visits 1-14 years ¹⁰				804		
 Notes: ^{1.} Used circulatory disease mortality data. ^{2.} Used circulatory disease mortality data for 25+ ye ^{3.} Used coronary heart disease mortality data for all ^{4.} Used respiratory disease mortality data for all ages. ^{5.} Used lung cancer mortality data for all ages. ^{6.} Used respiratory disease hospitalisation data for 1 ^{7.} Used data for cardiovascular disease hospitalisation ^{8.} Used all pneumonia and influenza hospitalisation data for 1 ^{9.} Used coronary heart disease hospitalisation data for 0-17 	 ^a 2012-2013 data (NSW HealthStats¹) ^b 2013 data (NSW HealthStats). ^c 2006-2007 data (Table 2.3 in EnRisks 2014a). ^d 2005-2007 data (Table 2.3 in EnRisks 2014a). ^e 2010-2011 data (NSW HealthStats). ^f 2011 data (NSW HealthStats). ^g 2004-2008 data (SWS LHD 2014). ^h 2013-2014 data (NSW HealthStats). ⁱ 2009-2011 data (Table 2.3 in EnRisks 2014a). ^j 2010-2011 data (Table 2.3 in EnRisks 2014a). 					

Abbreviations: COPD: Chronic Obstructive Pulmonary Disease; ED: Emergency Department; LGA: Local Government SWS LHD: South Western Sydney Local Health District

¹ Available at: http://www.healthstats.nsw.gov.au/

4.1.4 Existing air quality

Existing air quality in the local area has been evaluated in the AQIA. Local air quality is influenced by a number of industrial and non-industrial sources, including existing industries surrounding the site, the Glenfield Waste Disposal facility, traffic emissions from the existing road network, locomotive emissions from the East Hills rail line (south of the site) and the Southern Sydney Freight Line (SSFL)/Main Southern rail line (to the west), and emissions from aircraft at Bankstown Airport (northeast of the site).

Background air quality is described in the AQIA with reference to monitoring data from a nearby monitoring station at Liverpool, operated by the Office of Environment and Heritage (OEH). The Liverpool OEH monitoring site is located on Rose Street, situated in a mixed residential and commercial area. The monitoring station measures Particulate Matter (PM_{10} and $PM_{2.5}$), oxides of nitrogen (NO_x), ozone (O_3) and carbon monoxide (CO). The Liverpool OEH monitoring site does not include monitoring for sulphur dioxide (SO_2) and reference is therefore also made to the OEH monitoring site at Chullora, located approximately 12 km northeast of the Proposal site.

Annual mean PM_{10} concentrations range from 18 µg/m³ to 21 µg/m³ and on average over the past 5 years, baseline concentrations are 77% of the National Environment Protection (Ambient Air Quality) Measure (AAQ NEPM) standards. Annual mean $PM_{2.5}$ concentrations range from 6 µg/m³ to 9 µg/m³ and on average over the past 5 years baseline concentrations are 103% of the AAQ NEPM standard. Exceedances of the 24-hour average reporting standards for both PM_{10} and $PM_{2.5}$ have occurred in three of the past five years. Existing concentrations of PM_{10} and $PM_{2.5}$ for the Liverpool area are strongly influenced by vehicle emissions and wood heaters.

Although $PM_{2.5}$ concentrations for the Liverpool area are currently non-compliant with the NEPM AAQ standards, regulatory initiatives such as the NSW EPA Clean Air Plan outline potential actions for wood heaters and transport emissions, which are expected to play an important role in driving down long term ambient concentrations by 2027.

For NO₂, SO₂ and CO there have been no exceedances of the air quality standards for the previous five years and in general, background air quality for these pollutants is considered good. On average over the past five years, baseline concentrations for NO₂ are 33% of the AAQ NEPM standard for annual mean and 42% for maximum 1 hour average. Relative to the AAQ NEPM standards, baseline concentrations for CO and SO₂ are even lower. For example, maximum 1-hour baseline concentrations are 12% of the AAQ NEPM standard for CO and 10% for SO₂.

4.2 Air modelling

4.2.1 Assessment scenario

The cumulative impacts on the local area from all sources were compared against the relevant impact assessment criteria or NEPM air quality standards in the AQIA. Results indicated that the predicted increase in concentrations of key air pollutants from the construction and operation of the Proposal were considered minor when compared against existing background levels. The predicted cumulative ground level concentrations were below the air quality standards for short-term and long-term impacts, except for the annual average concentrations of $PM_{2.5}$; however, such exceedances were because the background concentrations of $PM_{2.5}$ already exceeded the NEPM air quality standard.

The HRA, therefore, focuses on the change in health outcomes as a result of new emission sources in the area that are added to existing background concentrations; that is from the operation of the Proposal. As previously described, emissions from construction were not evaluated in this HRA. The focus of this air quality HRA is on the health impacts of emissions from the operational sources related to the Proposal.

4.2.2 Emission sources

The air quality data used in the HRA has been generated through air modelling in the AQIA. Emissions to air from the operation of the Proposal have been quantified for the following operational scenarios:

- Scenario 1: Operation of the Proposal, as described in Section 2.4.
- Scenario 2: Cumulative operation, incorporating a combined precinct total 750,000 TEU (250,000 TEU for the MPE Stage 1 and 500,000 TEU for the MPW Stage 2) plus 515,000 m² of warehousing (300,000 m² for MPE Stage 2 and 215,000 m² for MPW Stage 2).

These sources result in emissions to air that are derived from diesel and other fossil fuel consumption. Hence, the assessment of impacts to air was focused primarily on health hazards associated with combustion emissions. The key air pollutants the AQIA has evaluated included:

- PM₁₀ and PM_{2.5};
- Nitrogen oxides (in particular NO₂);
- SO₂;
- CO;

L

- Volatile organic compounds (VOCs); and
- Polycyclic aromatic hydrocarbons (PAHs).

4.2.3 Modelled locations

The populations that may be exposed to air emissions from the Proposal are communities in the surrounding suburbs of Casula, Moorebank, Glenfield, and Wattle Grove. A total of 41 locations representative of the surrounding suburbs and other sensitive receptors (e.g., schools, day care centres, and aged care homes/facilities) were identified and selected as discrete sensitive receptors in the AQIA.

These locations are the same as those modelled in the EIS for the MPE Stage 1 Project in order to ensure consistency in interpretation of results and facilitate assessment of cumulative impact based on previous modelling. The modelled locations are shown in **Figure 4-1** and listed in **Table 4.3**.

Table 4.3: Off-site sensitive receptors				
Receptor location	Suburb	Receptor population		
Lakewood Crescent	Casula	Residential		
St Andrews Boulevard	Casula	Residential		
Buckland Road	Casula	Residential		
Dunmore Crescent	Casula	Residential		
Leacocks Lane	Casula	Residential		
Leacocks Lane_Mid	Casula	Residential		
Slessor Road	Casula	Residential		
Canterbury Road	Glenfield	Residential		
Ferguson Street	Glenfield	Residential		
Goodenough Street	Glenfield	Residential		
Wallcliff Court	Wattle Grove	Residential		
Corryton Court	Wattle Grove	Residential		
Martindale Court	Wattle Grove	Residential		
Anzac Road	Moorebank	Residential and Commercial/Industrial		
Cambridge Avenue	Glenfield	Residential		
Yallum Court	Wattle Grove	Residential		
Church Road	Moorebank	Residential		
Glenwood Public School	Glenfield	Residential/School		
Glenfield Public School	Glenfield	Residential/School		
Hurlstone Agricultural School	Glenfield	Residential/School		
Wattle Grove Public School	Wattle Grove	Residential/School		
St Marks Coptic College	Wattle Grove	Residential/School		
Maple Grove Retirement Village	Casula	Residential		
All Saints Catholic College	Casula	Residential/School		
Casula High School	Casula	Residential/School		
Casula Primary School	Casula	Residential/School		
Glenfield Rise Development	Glenfield	Residential		

Table 4.3: Off-site sensitive receptors				
Receptor location	Suburb	Receptor population		
New DNSDC Facility	Moorebank	Commercial/Industrial		
Playground Learning Centre	Glenfield	Residential		
Wattle Grove Long Day Care Centre	Wattle Grove	Residential and Recreational		
Casula Powerhouse Arts Centre	Casula	Recreational		
Little Peters Child Care	Casula	Residential		
Anzac Village Pre School	Wattle Grove	Residential/School		
St Christophers. Holsworthy	Wattle Grove	Residential/School		
Learn and Play Pre School	Wattle Grove	Residential/School		

4.2.3.1 Regional Air Quality

An assessment of regional air quality impacts is not a requirement of the SEARs for air quality, therefore no quantitative assessment has been undertaken for changes to regional air quality in this HRA (i.e. assessment of photochemical smog (ozone) on a regional scale). Regional air quality has been previously considered for the Concept Plan Approval for both the MPE and MPW Project. It is expected that changes in regional air quality as a result of the operation of the cumulative Proposal would be negligible, or may even result in a reduction in regional emissions, as a result of the efficiencies achieved by replacing road freight with rail freight.

4.3 Health risk assessment methodology

4.3.1 Issue identification

To identify the issues that can be assessed through the risk assessment and assist in establishing a context for the risk assessment, a conceptual site model (CSM) was developed. A CSM is a sitespecific qualitative description of the chemical source(s), the pathway(s) by which chemicals may migrate through the environmental media, and the populations that may potentially be exposed. This relationship is commonly known as a Source-Pathway-Receptor (SPR) linkage. Where one or more elements of the SPR linkage are missing, the exposure pathway is considered to be incomplete and no further assessment is required.

Source and chemicals of potential concern

The key air pollutants evaluated in the AQIA (see **Section 4.2.2**) were considered as chemicals of potential concern (COPCs) in this HRA. These key chemicals are discussed further below to refine the COPCs for the assessment.

Most of the VOC emissions comprise a range of hydrocarbons that are of low toxicity (such as methane, ethylene, ethane, butenes, butanes, pentenes, pentanes, and heptanes etc.) (USEPA 2012). From a toxicity perspective, the key VOCs that have been considered for the vehicle emissions in this HRA were benzene and 1,3-butadiene.

Hundreds of PAHs and nitro-PAHs exist in diesel exhaust (USEPA 2012). The toxicity of individual PAHs varies significantly, with the most toxic being benzo(a)pyrene (BaP), which is classified as a probable human carcinogen. Carcinogenic PAHs are commonly assessed as a group using the toxicity equivalent factor (TEF) approach. In this approach, the toxicity contribution of each individual carcinogenic PAH in diesel exhaust is calculated by multiplying its air concentration by its TEF based on relative toxicity potency to BaP, and then the results are summed to obtain BaP Toxicity Equivalent (BaP TEQ)(enHealth 2012a). Therefore, the carcinogenic effect of PAHs was evaluated as BaP TEQ in this HRA, and the TEFs presented by Canadian Council of Ministers for the Environment (CCME 2010) have been adopted to calculate BaP TEQ. This is consistent with the approach to assessing PAHs adopted in the NEPM (NEPC 2013).

The potential health effects of diesel exhaust from trucks are associated primarily with particle fraction of diesel. Diesel particulate matter (DPM) has not been specifically modelled in the AQIA; rather DPM was part of the PM_{2.5} assessment for emissions from diesel trucks and non-diesel light

vehicles. For the purposes of this HRA, it has been conservatively assumed that 100 percent of the incremental $PM_{2.5}$ is derived from diesel sources, which is a conservative assumption for Proposal which includes emissions from light vehicles and natural gas combustion associated with warehousing.

The data provided for the HRA also assumed that all NOx is NO₂ which is also a conservative assumption. A review of NO₂ and NO_x monitoring data from the OEH Liverpool monitoring site for the past 5 years indicates that, on average, ambient ratios of NO₂:NO_x range from 0.6 to 0.8 on an annual basis (average of 0.7 over the 5 year period). In other words, ambient NO₂ is typically approximately 70% of NO_x.

In summary, the COPCs identified for this HRA included:

- PM₁₀ and PM_{2.5} (including DPM);
- NO₂;
- SO₂;
- CO;
- Benzene and 1,3--butadiene; and
- PAHs (as BaP TEQ).

4.3.2 Exposure assessment

Human receptors

The human receptors of concern for this HRA included commercial/industrial workers, residents, school or day care students, and recreational users located in the vicinity of the Proposal. For residents, it was assumed that they may live all day every day in the local area for 35 years (enHealth 2012b). Therefore, exposure is assumed to occur 24 hours per day, 365 days per year. For school or day care students, since they may live and attend school all day every day in the local area, the same assumptions were also applied.

For commercial/industrial workers, it was assumed that exposure may occur eight hours per day, 240 days per year for 30 years (NEPC, 2013). For recreational users, it was assumed that exposure may occur four hours per day, 104 days per year (two days per week) for 35 years (enHealth 2012b, EnRisks 2014a).

Exposure pathways

For a human receptor to be exposed to COPCs, there needs to be an exposure pathway linking the source and the exposed population. An exposure pathway describes the course a chemical takes from the source to the exposed individual and generally includes the following elements (USEPA 1989):

- A source and mechanism of chemical release
- A retention or transport medium (or media where chemicals are transferred between media)
- A point of potential human contact with the contaminated media
- An exposure route (e.g., ingestion, inhalation) at the point of exposure.

The transport mechanisms for COPCs are atmospheric emissions to air and deposition to soil and surface water. Consistent with the previous risk assessments for the MPE Stage 1 Project, oral (non-inhalation) exposure routes related to deposition were not evaluated. Inhalation of air was the only exposure route evaluated in this HRA. A detailed assessment of the potential exposure pathways and human receptors is presented in **Table 4.4**.

Table 4.4: Exposur	e Pathway A	ssessment		
Evroquina	Po	tentially Com	plete?	
Exposure Pathway	Resident/ Student	Commercial Worker	Recreational User	Justification
Air	-			
Inhalation	Y	Y	Y	Populations in the surrounding communities may be exposed to COPCs by inhaling air emissions from operation of the Proposal.
Soil		I	L	
Incidental Ingestion	L	L	L	Populations in the surrounding communities may be exposed to chemicals in particulate matter (PM) deposited from air to soil through non-inhalation exposure routes.
Dermal Contact	L	L	L	However, consistent with the HRA for the MPE Stage 1 Project, non-inhalation exposure routes related to chemicals in deposited particulate matter were not assessed in this HRA.
Surface Water		÷	•	·
Incidental Ingestion	L	L	L	Populations in the surrounding communities may be exposed to chemicals in particulates and dust deposited from air to surface water through non-inhalation exposure routes.
Dermal Contact	L	L	L	However, consistent with the HRA for the MPE Stage 1 Project, non-inhalation exposure routes related to chemicals in deposited particulate matter were not assessed in this HRA.

Y – Pathway complete and quantitatively evaluated in the HRA

L – Pathway complete but considered less significant, therefore not quantitatively evaluated in the HRA

Exposure Point Concentrations

The annual average ground level concentrations (GLCs) of COPCs emitted from operation of the Proposal were predicted by air modelling in the AQIA at selected sensitive receptor locations in the surrounding suburbs. It was assumed that the Proposal would operate at the proposed capacity for no less than 35 years. The annual average GLCs were calculated by averaging the predicted air concentrations (concentrations over the minimum time period of operation) from the source over a continuous time period of 24 hours per day and 365 days per year.

All activities associated with the Proposal would occur 24/7, therefore the annual average GLCs are equal to the predicted air concentrations from the source and used directly as exposure point concentrations (EPCs) for all human receptors (i.e. residents, school or day care students, commercial/industrial workers, recreational users).

At each sensitive receptor location, the EPCs from all the sources were added together to obtain the EPCs from the operation of the Proposal and cumulative Proposal, which are shown along with the HRA results in **Section 4.4**.

4.3.3 Toxicity Assessment

The toxicity assessment involved both the qualitative evaluation of the adverse health effects associated with inhalation exposure to COPCs and the quantitative evaluation of the exposure-response relationships. Based on the available information, the most robust health endpoints (effects or outcomes) for the assessment of inhalation exposure to COPCs have been identified, and the exposure-response relationships for these health endpoints were derived from published peer-reviewed sources.

The adverse health effects from each COPC were discussed in detail in the MPE Stage 1 HIA (Pacific Environment, 2015), however a brief summary of some of the relevant health effects are provided in **Table 4.5**.

The health endpoints and associated exposure-response relationships adopted for this HRA (presented in **Section 4.4**) are consistent with those used in the MPE Stage 1 HIA, approved by NSW Health as part of the consultation undertaken for MPE Stage 1.

COPC	Summary of health effects	Source	
Particles	 Short term effects include: Lung inflammation Respiratory symptoms Adverse effects on the cardiovascular system Increased medication use Increased hospitalisations Increased mortality Long term effects include: Increased lower respiratory symptoms Reduced lung function in children Increased chronic obstructive pulmonary disease Reduced lung function in adults Reduced life expectancy, mainly due to cardiopulmonary mortality and probably to lung cancer 	Morgan et al, (2013)	
Nitrogen dioxide	 Short term effects include: Effects on lung function, especially in asthmatics Increased airway allergic inflammatory reactions Increased hospitalisations Increased mortality Long term effects include: Reduced lung function Increased probability of respiratory symptoms 	(2013)	
Sulphur dioxide	 Short term effects include: Effects on lung function, especially in asthmatics Increased hospitalisations Increased mortality 		
Carbon monoxide	CO can be harmful to humans because its affinity for haemoglobin is more than 200 times greater than that of oxygen. When CO is inhaled, it is taken up by the blood and therefore reduces the capacity of the blood to transport oxygen, although this process is reversible. Ambient (outdoor) levels of CO are unlikely to be elevated, however high levels in outdoor air can be of particular concern for people with some types of heart disease, whereby short-term exposure may result in reduced oxygen to the heart accompanied by chest pain also known as angina.	United States Environment Protection Agency (US EPA) ²	
Benzene	Known human carcinogen, associated with leukemia, especially acute myelogenic leukemia.		
1,3 – butadiene	Classified as a human carcinogen, associated with hematolymphopoietic cancers	Agency for Toxic Substances and Disease Registry (ATSDR) ³ .	
PAHs	The International Agency for Research on Cancer (IARC) has classified two PAHs as probable carcinogens and four PAHs as possible carcinogens. Similarly, the US EPA has determined that seven PAHs are probable human carcinogens. In particular, exposure to PAHs is linked with lung and skin cancer.		

4.3.4 Risk Characterisation

Potential health impacts from inhalation exposure to COPCs have been evaluated based the annual average GLCs predicted in the AQIA. The health effects of both short-term and long-term exposure to PM_{10} , $PM_{2.5}$, NO_2 , SO_2 and CO have been assessed for increased annual incidence, in terms of health endpoints of mortality and morbidity.

 $^{^{2}\} https://www.epa.gov/co-pollution/basic-information-about-carbon-monoxide-co-outdoor-air-pollution \# Effects$

³ https://www.atsdr.cdc.gov/substances/index.asp

The increased annual incidence was calculated using the following equation where a linear exposure-response was assumed (Burgers and Walsh 2002, Ostro 2004, USEPA 2005, 2010), which is consistent with the methodology used in previous health assessment for the MPE Project (EnRisks, 2014a; Pacific Environment, 2015):

$E = \beta * EPC_{suburb} * B * P_{suburb}$

Where:

E	 Increased annual incidence (number of cases per year for a given health endpoint attributable to the Proposal)
β	Exposure-response function (change in health outcome) per 1 μg/m ³ increase in EPC for a given health endpoint ([μg/m ³] ⁻¹ , as discussed in Section 4.4)
EPC _{suburb}	 Exposure point concentration associated with the operation of the Project, calculated as mean of annual average GLCs for all locations modelled within a suburb (µg/m³)
В	 Baseline health incidence rate per person per year for a given health endpoint (unitless, calculated based on the values presented in Table 4.2)
P _{suburb}	Population within a suburb for the age group relevant to the health endpoint being evaluated (as presented in Table 4.1)

Both long-term and short-term exposure were assessed on the basis of annual average GLCs. Annual average GLCs are used to assess short term exposures because the concentration-response functions are linear, and the same outcome will be obtained by assuming that the annual change in concentrations was derived by 365 equal daily changes or by 365 varied daily changes with the same average value. In other words, the results of the annual incidence for short-term exposure calculated using the following two approaches should be the same mathematically (Ostro 2004, USEPA 2010):

- Calculate the daily incidence based on the 24-hour average concentration and daily health incidence rate, and then sum the daily incidence to get the annual incidence or risk.
- Calculate the annual incidence based on the annual average concentration and annual health incidence rate.

In addition, the excess lifetime cancer risks from inhalation exposures to air toxics, such as DPM, benzene, 1,3-butadiene, and PAHs (as BAP TEQ), associated with the Proposal were calculated for the human receptors in the local area using the following equation (USEPA 2009):

Where:

EPC	=	Exposure point concentration associated with the operation of the Proposal at each sensitive receptor location (μ g/m ³)
ET	=	Exposure time (hours per day0
EF	=	Exposure frequency (days per year)
ED	=	Exposure duration (years)
AT	=	Averaging time for cancer risk (i.e. 70 years or 25550 days)
CF	=	Conversion factor (24 hours/day)
URF	=	Unit risk factor ([µg/m ³] ⁻¹).

Acceptable Risk

A discussion is provided in enHealth (2012a) on the acceptable risk levels for excess lifetime cancer risks or non-threshold risks. However, no direct recommendation on the use of a target level is provided. The enHealth (2012a) document indicates that while a target risk level of 1×10^{-6} is one of the more commonly used, the target risk level has varied between 10^{-6} and 10^{-3} in different types of risk management situations.

The USEPA (1991) states the following:

- The target risk level of 1 in 1,000,000 for an excess lifetime cancer risk is for exposure to an individual carcinogen by multiple pathways (e.g. ingestion, dermal exposure, and inhalation);
- Action is generally warranted when the excess lifetime cancer risk is greater than 1 in 10,000 for exposure to multiple carcinogens and pathways. This means that when all of the risks for individual carcinogens and pathways posed are added together, the excess lifetime cancer risk should not be greater than one in ten thousand people who have been exposed; and
- When the cumulative risk for a medium is within the range of 1 in 1,000,000 to 1 in 10,000, a decision about whether or not to take action is a site-specific determination.

The revised NEPM (NEPC, 2013) uses an acceptable target risk level of 1×10^{-5} (i.e., 1 in 100,000) to assess all pathways of exposure. Consistent with the previous risk assessments for the MPE Project, the excess lifetime cancer risks have been considered acceptable in the range of 10^{-6} to 10^{-4} .

The increased annual incidence of mortality or morbidity endpoints has been considered to be negligible when it was less than one case per year, which is not detectable above the normal fluctuations in health statistics. For health endpoints or populations where there is great variability in annual incidence, increases in incidence much greater than one case per year may not be detected above the normal variability observed in health statistics.

4.4 Evaluation of health risk for the Proposal

The HRA results for each COPC associated with the operation of the Proposal are summarised in the following sections. Detailed calculations are presented in **Appendix 2** for increased annual incidence for mortality and morbidity endpoints, and **Appendix 3** for excess lifetime cancer risk.

4.4.1 Particulate matter

The health endpoints and exposure-response functions (β values) adopted in this HRA for the evaluation of PM₁₀ and PM_{2.5} are presented in **Table 4.6** and **Table 4.7**, respectively.

Health Endpoint	Exposure period	β (Exposure Response Function per 1 µg/m ³ Increase in PM ₁₀)
All-cause mortality 30+ years	Annual Average	0.004
All-cause mortality all ages	24-Hour Average	0.002
Mortality cardiovascular disease all ages	24-Hour Average	0.002
Hospital admissions respiratory disease 65+ years	24-Hour Average	0.003
Hospital admissions cardiac disease 65+ years	24-Hour Average	0.002
Hospital admissions pneumonia and bronchitis 65+ years	24-Hour Average	0.0013
Hospital admissions respiratory disease 15-64 years	24-Hour Average	0.003
ED visits asthma 1-14 years	24-Hour Average	0.015

Table 4.6: Health endpoints and exposure-related functions for PM_{10}

Note: Exposure response functions were obtained from Environment Protection and Heritage Council (EPHC 2010) and Health Effects Institute (HEI 2009).

Abbreviations: ED: Emergency Department; PM: Particulate Matter

Health Endpoint	Exposure period	β (Exposure Respons Function per 1 μg/m Increase in PM _{2.5})	
All-cause mortality 30+ years	Annual Average	0.006	
Cardiopulmonary mortality 30+	Annual Average	0.014	
Mortality ischemic heart disease 30+ years	Annual Average	0.024	
Mortality lung cancer 30+ years	Annual Average	0.014	
All-cause mortality all ages	24-Hour Average	0.0023	
Mortality cardiovascular disease- all ages	24-Hour Average	0.0013	
Hospital admissions respiratory disease 65+ years	24-Hour Average	0.004	
Hospital admissions cardiac disease 65+ years	24-Hour Average	0.005	
Hospital admissions cardiovascular disease 65+ years	24-Hour Average	0.003	
Hospital admissions ischemic heart disease 65+ years	24-Hour Average	0.004	
Hospital admissions COPD 65+ years	24-Hour Average	0.004	
Hospital admissions pneumonia and bronchitis 65+ years	24-Hour Average	0.005	
Hospital admissions respiratory disease 15-64 years	24-Hour Average	0.003	
ED visits asthma 1-14 years	24-Hour Average	0.0015	

Table 4.7: Health and noints and exposure-related functions for PMar

Abbreviations: COPD: Chronic Obstructive Pulmonary Disease ; ED: Emergency Department; PM: Particulate Matter

The increased annual incidences for the health endpoints evaluated due to PM_{10} and $PM_{2.5}$ exposure for each suburb are summarised in Table 4.8 and Table 4.9. The health outcomes evaluated for exposure to PM include:

- Premature mortality
 - All-causes (ages 30+ years and all ages)
 - Cardiopulmonary (ages 30+ years)
 - Ischemic heart disease (ages 30+ years)
 - Lung cancer (ages 30+ years)
 - Cardiovascular disease (all ages)
- Hospital admissions
 - Respiratory disease (ages 65+ years and ages 15-64 years)
 - Cardiac disease (ages 65+ years)
 - Cardiovascular disease (ages 65+ years)
 - Ischemic heart disease (ages 65+ years)
 - Chronic obstructive pulmonary disease (ages 65+ years)
 - Pneumonia and bronchitis (ages 65+ years)
- Emergency department visits associated with asthma (ages 1-14 years)

The increased annual incidences for the health endpoints evaluated due to Proposal related PM₁₀ and PM_{2.5} exposure were all well below one case per year. For the most sensitive health endpoint of PM_{10} , the highest incidence is an additional 0.01 asthma-related emergency department visit per year among 1-14 year-olds in Wattle Grove (equivalent to one additional emergency department visit per 100 years). For the most sensitive health endpoints of $PM_{2.5}$, there would be an additional 0.02 hospital admission per year associated with cardiac disease among 65+ year-olds in Moorebank (equivalent to two additional hospital admissions per 100 years), which may be attributed to daily exposure to emissions of $PM_{2.5}$ from the operation of the Proposal. Based on the estimated increased annual incidence for multiple health endpoints contributing to mortality and morbidity for the Proposal, there are no significant adverse health effects expected in relation to short-term and long-term exposure to PM₁₀ and PM_{2.5} in the surrounding local area.

Health and wint	Evenence novied	Incre	Increased annual incidence (case per year)				
Health endpoint	Exposure period	Casula	Glenfield	Moorebank	Wattle Grove		
All-cause mortality 30+ years	Annual Average	0.005	0.004	0.009	0.007		
All-cause mortality all ages	24-Hour Average	0.003	0.002	0.004	0.004		
Mortality cardiovascular disease all ages	24-Hour Average	0.0008	0.0005	0.001	0.001		
Hospital admissions respiratory disease 65+ years	24-Hour Average	0.003	0.003	0.006	0.003		
Hospital admissions cardiac disease 65+ years	24-Hour Average	0.005	0.004	0.008	0.003		
Hospital admissions pneumonia and bronchitis 65+ years	24-Hour Average	0.0004	0.0003	0.0007	0.0003		
Hospital admissions respiratory disease 15-64 years	24-Hour Average	0.004	0.003	0.006	0.007		
ED visits asthma 1-14 years	24-Hour Average	0.007	0.003	0.009	0.01		

Uselth and sint	Exposure	Increased annual incidence (case per year)				
Health endpoint	period	Casula	Glenfield	Moorebank	Wattle Grove	
All-cause mortality 30+ years	Annual Average	0.008	0.006	0.01	0.01	
Cardiopulmonary mortality 30+	Annual Average	0.008	0.007	0.01	0.01	
Mortality ischemic heart disease 30+ years	Annual Average	0.002	0.002	0.003	0.003	
Mortality lung cancer 30+ years	Annual Average	0.0006	0.0003	0.001	0.0008	
All-cause mortality all ages	24-Hour Average	0.003	0.002	0.004	0.004	
Mortality cardiovascular disease- all ages	24-Hour Average	0.0005	0.0003	0.0007	0.0007	
Hospital admissions respiratory disease 65+ years	24-Hour Average	0.004	0.004	0.008	0.003	
Hospital admissions cardiac disease 65+ years	24-Hour Average	0.01	0.009	0.02	0.009	
Hospital admissions cardiovascular disease 65+ years	24-Hour Average	0.007	0.006	0.01	0.005	
Hospital admissions ischemic heart disease 65+ years	24-Hour Average	0.003	0.003	0.006	0.002	
Hospital admissions COPD 65+ years	24-Hour Average	0.001	0.001	0.003	0.001	
Hospital admissions pneumonia and bronchitis 65+ years	24-Hour Average	0.002	0.001	0.003	0.001	
Hospital admissions respiratory disease 15-64 years	24-Hour Average	0.004	0.003	0.006	0.007	
ED visits asthma 1-14 years	24-Hour Average	0.0007	0.0003	0.0009	0.001	

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4.4.2 Nitrogen Dioxide

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The health endpoints and exposure-response functions (β values) adopted in this HRA for the evaluation of NO₂ (modelled as NO_x) are presented in **Table 4.10**.

Health endpoint	Exposure period	β (Exposure response function per 1 µg/m ³ increase in NO ₂)
All-cause mortality 30+ years	Annual Average	0.0028
Cardiovascular mortality 30+ years	Annual Average	0.0028
Respiratory mortality 30+ years	Annual Average	0.0028
All-cause mortality all ages	24-Hour Average	0.001
Mortality respiratory disease all ages	24-Hour Average	0.0023
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
Note:	aroni at al. (2013)	<u>.</u>
Exposure response functions were obtained from EPHC (2010) and Ces Abbreviations: µg/m ₃ : microgram per cubic meter; ED: Emergency D		Novida

The increased annual incidences for the health endpoints evaluated due to Proposal related NO_2 exposure for each suburb are summarised in **Table 4.11**. Health outcomes evaluated for exposure to NO_2 included:

- Premature mortality
 - All-causes (ages 30+ years and all ages)
 - Cardiovascular (ages 30+ years and all ages)
 - Respiratory (ages 30+ years and all ages)
- Hospital admissions
 - Respiratory disease (ages 65+ years and ages 15-64 years)
 - Cardiovascular disease (ages 65+ years)
- Emergency department visits associated with asthma (ages 1-14 years)

The increased annual incidences for the Proposal were below one case per year for all health endpoints and in all locations. The highest increased annual incidence would be 0.2 for all-cause mortality among 30+ year-olds and hospital admissions for cardiovascular and respiratory disease among 65+ year olds in Moorebank (equivalent to two additional deaths or hospital admissions per 10 years).

Based on the estimated increased annual incidence for multiple health endpoints contributing to mortality and morbidity, there are no significant adverse health effects expected in relation to short-term and long-term exposure to NO_2 for the Proposal in the surrounding local area.

Table 4.11: Summary of increased annual incidence associated with exposure to NO ₂ from the operation of Proposa Use the exploring term incidence (case per year)					
Health endpoint	Exposure period	Casula	Glenfield	Moorebank	Wattle Grove
All-cause mortality 30+ years	Annual Average	0.1	0.07	0.2	0.1
Cardiovascular mortality 30+ years	Annual Average	0.03	0.02	0.05	0.03
Respiratory mortality 30+ years	Annual Average	0.005	0.003	0.008	0.006
All-cause mortality all ages	24-Hour Average	0.04	0.02	0.05	0.05
Mortality respiratory disease	24-Hour Average	0.008	0.004	0.01	0.01
Mortality cardiovascular disease all ages	24-Hour Average	0.01	0.006	0.02	0.01
Hospital admissions respiratory disease 65+ years	24-Hour Average	0.1	0.07	0.2	0.06
Hospital admissions cardiovascular disease 65+ years	24-Hour Average	0.09	0.07	0.2	0.06
Hospital admissions respiratory disease 15-64 years	24-Hour Average	0.04	0.02	0.06	0.06
ED visits asthma 1-14 years	24-Hour Average	0.008	0.003	0.01	0.01
Abbreviations: ED: Emergency Department, NO ₂ : Nitrogen Dioxide					

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4.4.3 Sulfur Dioxide

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The health endpoints and exposure-response functions (β values) adopted in this HRA for the evaluation of SO₂ are presented in **Table 4.12**.

Table 4.12: Health endpoints and exposure-response functions for SO_2				
Health endpoint	Exposure period	β (Exposure response function per 1 μg/m ³ increase in SO ₂)		
All-cause mortality all ages	24-Hour Average	0.0006		
Mortality respiratory disease- all ages	24-Hour Average	0.0013		
Mortality cardiovascular disease- all ages24-Hour Average0.0008				
Hospital admissions respiratory disease 65+ years	1- Hour Maximum	0.002		
ED visits asthma 1-14 years 24-Hour Average 0.008				
Note: Exposure response functions were obtained from Jalaudin et al. (2008), Katsouyanni (2006), and Simpson et al. (2005). Abbreviations: ED: Emergency Department. SO ₂ : Sulfur Dioxide				

Only short-term exposure was evaluated for SO_2 . The increased annual incidences for the health endpoints evaluated due to Proposal related SO_2 exposure for each suburb are summarised in **Table 4.13**. Health outcomes evaluated for exposure to SO_2 included:

- Premature mortality
 - All-causes (all ages)
 - Respiratory disease (all ages)
 - Cardiovascular disease (all ages)
- Hospital admissions
 - Respiratory disease (ages 65+ years)
- Emergency department visits associated with asthma (ages 1-14 years)

The increased annual incidences for the health endpoints evaluated due to Proposal related SO_2 exposure were all well below one case per year. For the most sensitive health endpoint, there would be an additional 0.004 asthma-related emergency department visit per year among 1-14 year-olds in Wattle Grove (equivalent to four additional emergency department visits per 1,000 years), which may be attributed to daily exposure to emissions of SO_2 from the operation of the Proposal.

Based on the estimated increased annual incidence for multiple health endpoints contributing to mortality and morbidity, there are no significant adverse health effects expected in relation to short-term exposure to SO_2 from the Proposal in the surrounding local area.

Table 4.13: Summary of increased annual inciProposal	dence associated wit	h exposure to SO_2 from the operation of the
Health endpoint	Exposure period	Increased annual incidence (case per year)

Hoalth and nount Exposure pariod						
Health endpoint	Exposure period	Casula	Glenfield	Moorebank	Wattle Grove	
All-cause mortality all ages	24-Hour Average	0.0005	0.0003	0.0007	0.0008	
Mortality respiratory disease- all ages	24-Hour Average	0.0001	0.00007	0.0002	0.0002	
Mortality cardiovascular disease- all ages	24-Hour Average	0.0002	0.0001	0.0003	0.0003	
Hospital admissions respiratory disease 65+ years	1- Hour Maximum	0.002	0.001	0.003	0.003	
ED visits asthma 1-14 years	24-Hour Average	0.002	0.001	0.003	0.004	
Abbreviations: ED: Emergency Department. SO ₂ : Sulfur Dioxide						

4.4.4 Carbon Monoxide

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The health endpoints and exposure-response functions (β values) adopted in this HRA for the evaluation of CO are presented in **Table 4.14**.

Health endpoint	Exposure period	β (Exposure response function per 1 μg/m ³ increase in CO)
All-cause mortality 30+ years	0.000001	
Hospital admissions cardiac disease 65+ years	8-Hour Average	0.000003
Hospital admissions cardiovascular disease 65+ years	8-Hour Average	0.0000014
Note: Exposure response functions were obtained from EPHC (2010) a Abbreviations: μg/m ³ : microgram per cubic meter CO: Carbon Monoxide	nd Simpson et al. (2005).	

Only short-term exposure was evaluated for CO. The increased annual incidences for the health endpoints evaluated due to Proposal related CO exposure for each suburb are summarised in **Table 4.15**. Health outcomes evaluated for exposure to CO included:

- Premature mortality
 - All-causes (ages 30+ years)
- Hospital admissions
 - Cardiac disease (ages 65+ years)
 - Cardiovascular disease (ages 65+ years)

The increased annual incidences for the health endpoints evaluated due to Proposal related CO exposure were all well below one case per year. For the most sensitive health endpoint, there would be an additional 0.0006 hospital admission per year associated with cardiac disease among 65+ year-olds in Moorebank (equivalent to six additional hospital admissions per 10,000 years), which may be attributed to 8-hour exposure to emissions of CO from the operation of the Proposal.

Based on the estimated increased annual incidence for multiple health endpoints contributing to mortality and morbidity, there are no significant adverse health effects expected in relation to short-term exposure to CO from the Proposal in the surrounding local area.

Table 4.15: Summary of increased annual incidence associated with exposure to CO from the operation of the	
Proposal	

Health and a sint	Exposure Increased annual incidenc				e (case per year)	
Health endpoint	period	Casula	Glenfield	Moorebank	Wattle Grove	
All-cause mortality 30+ years	8-Hour Average	0.00006	0.00005	0.0001	0.00009	
Hospital admissions cardiac disease 65+ years	8-Hour Average	0.0003	0.0003	0.0006	0.0003	
Hospital admissions cardiovascular disease 65+ years	8-Hour Average	0.00002	0.00002	0.00004	0.00002	
Abbreviations: CO: Carbon Monoxide. ED visits asthma 1-14 years						

4.4.5 VOCs, DPM and PAHs

The Unit Risk Factors (URFs) adopted in this HRA for VOCs (benzene and 1,3-butadiene), DPM and PAHs (as BAP TEQ) are presented in **Table 4.16**.

Table 4.16: Unit risk factors used for the calculation of excess lifetime cancer risk				
Chemical Unit risk factor (µg/m ³) ⁻¹				
Benzene	0.000029			
1,3-Butadiene 0.00017				
DPM	0.0003			
PAHs (as BaP TEQ) 0.0011				
Note: Unit risk factors were obtained from California Environmental Protection Agency (Cal/EPA) Office of Environmental				
Health Hazard Assessment (OEHHA). 2016. OEHHA Toxicity Criteria Database. Available at:				
http://oehha.ca.gov/tcdb/index.asp				
Abbreviations: µg/m ³ : microgram per cubic meter. BaP	: Benzo(a)pyrene. DPM: Diesel Particulate Matter. PAH: Polycyclic			
Aromatic Hydrocarbon. TEQ: Toxicity Equivalent				

Table 4.17 presents a summary of the excess lifetime cancer risks associated with exposure to benzene, 1,3-butadiene, DPM and PAHs (as BAP TEQ), for the maximum exposed receptor in each category (i.e. residential/school, commercial/industrial, or recreational).

The excess lifetime cancer risks associated with the Proposal related exposure to benzene, 1,3butadiene, and PAHs (as BAP TEQ) were all below the acceptable risk range of 10^{-6} to 10^{-4} . The excess lifetime cancer risks associated with the Proposal related DPM exposure were all within or well below the acceptable risk range of 10^{-6} to 10^{-4} .

Therefore, there are no unacceptable cancer risks are expected in relation to long-term exposure to VOCs, DPM and PAHs in the surrounding local area.

Table 4.17: Summary of excess lifetime cancer risks associated with exposure toBenzene, 1,3-Butadiene, PAHs, and DPM from the operation of the Proposal							
Chemical Excess lifetime cancer risk at maximum exposed receptor							
Residential/School	Recreational	Commercial/Industrial					
2.6E-07	4.2E-09	3.8E-08					
6.3E-07	1.0E-08	9.4E-08					
1.3E-05	2.5E-07	2.2E-06					
3.2E-09	6.2E-11	5.2E-10					
	diene, PAHs, and DPM Excess lifetime Residential/School 2.6E-07 6.3E-07 1.3E-05	diene, PAHs, and DPM from the operationExcess lifetime cancer risk at maxinResidential/SchoolRecreational2.6E-074.2E-096.3E-071.0E-081.3E-052.5E-07					

Abbreviations: BaP: Benzo(a)pyrene. DPM: Diesel Particulate Matter. PAH: Polycyclic Aromatic Hydrocarbon. TEQ: Toxicity Equivalent

4.5 Evaluation of health risks from the cumulative Proposal

The HRA results for each COPC associated with the operation of the cumulative Proposal are summarised in the following sections. The health endpoints and exposure-response functions (β values) are previously described in **Section 4.4**. The increased annual incidences for the health endpoints evaluated due to cumulative exposure for each COPC are summarised in **Table 4.18** to **Table 4.22**.

Based on the estimated increased annual incidence for multiple health endpoints contributing to mortality and morbidity, there are no significant adverse health effects expected in relation to short-term exposure to PM_{10} , $PM_{2.5}$, NO_2 , SO_2 or CO from the cumulative Proposal in the surrounding local area.

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		Increased annual incidence (case per year)			
Health endpoint	Exposure period	eriod Casula G		Moorebank	Wattle Grove
All-cause mortality 30+ years	Annual Average	0.07	0.04	0.07	0.07
All-cause mortality all ages	24-Hour Average	0.04	0.01	0.03	0.04
Mortality cardiovascular disease all ages	24-Hour Average	0.01	0.004	0.009	0.01
Hospital admissions respiratory disease 65+ years	24-Hour Average	0.05	0.02	0.05	0.02
Hospital admissions cardiac disease 65+ years	24-Hour Average	0.06	0.03	0.06	0.03
Hospital admissions pneumonia and bronchitis 65+ years	24-Hour Average	0.006	0.003	0.006	0.003
Hospital admissions respiratory disease 15-64 years	24-Hour Average	0.06	0.02	0.05	0.06
ED visits asthma 1-14 years	24-Hour Average	0.09	0.03	0.07	0.1

The increased annual incidences for the health endpoints evaluated due to cumulative Proposal related PM_{10} exposure were all well below one case per year. For the most sensitive health endpoint of PM_{10} , there would be an additional 0.1 asthma-related emergency department visit per year among 1-14 year-olds in Wattle Grove (equivalent to one additional emergency department visit per 10 years), which may be attributed to daily exposure to emissions of PM_{10} from the operation of the cumulative Proposal.

Uasth and aint	Exposure	Increased annual incidence (case per year)			
Health endpoint	period	Casula	Glenfield	Moorebank	Wattle Grove
All-cause mortality 30+ years	Annual Average	0.1	0.05	0.1	0.09
Cardiopulmonary mortality 30+	Annual Average	0.1	0.06	0.1	0.1
Mortality ischemic heart disease 30+ years	Annual Average	0.03	0.01	0.03	0.02
Mortality lung cancer 30+ years	Annual Average	0.008	0.004	0.008	0.007
All-cause mortality all ages	24-Hour Average	0.04	0.02	0.03	0.04
Mortality cardiovascular disease- all ages	24-Hour Average	0.007	0.003	0.005	0.007
Hospital admissions respiratory disease 65+ years	24-Hour Average	0.06	0.03	0.06	0.03
Hospital admissions cardiac disease 65+ years	24-Hour Average	0.2	0.08	0.2	0.08
Hospital admissions cardiovascular disease 65+ years	24-Hour Average	0.09	0.05	0.09	0.05
Hospital admissions ischemic heart disease 65+ years	24-Hour Average	0.05	0.02	0.05	0.02
Hospital admissions COPD 65+ years	24-Hour Average	0.02	0.01	0.02	0.01
Hospital admissions pneumonia and bronchitis 65+ years	24-Hour Average	0.02	0.01	0.02	0.01
Hospital admissions respiratory disease 15-64 years	24-Hour Average	0.06	0.02	0.05	0.06
ED visits asthma 1-14 years	24-Hour Average	0.009	0.003	0.007	0.01

The increased annual incidences for the health endpoints evaluated due to cumulative Proposal related $PM_{2.5}$ exposure were all well below one case per year. For the most sensitive health endpoint of $PM_{2.5}$, there would be an additional 0.2 hospital admissions per year associated with cardiac disease among 65+ year-olds in Casula or Moorebank (equivalent to two additional hospital admissions per 10 years), which may be attributed to daily exposure to emissions of $PM_{2.5}$ from the operation of the cumulative Proposal.

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Health endpoint	Exposure period	Increased annual incidence - case per year (values in brackets assume ambient ratio of NO ₂ to NO _x of 0.7)			
·		Casula	Glenfield	Moorebank	Wattle Grove
All-cause mortality 30+ years	Annual Average	1.4 (1)	0.8 (0.5)	1.4 (1)	1.2 (0.9)
Cardiovascular mortality 30+ years	Annual Average	0.4 (0.3)	0.2 (0.1)	0.4 (0.3)	0.3 (0.2)
Respiratory mortality 30+ years	Annual Average	0.07 (0.05)	0.04 (0.02)	0.07 (0.05)	0.06 (0.04)
All-cause mortality all ages	24-Hour Average	0.5 (0.4)	0.2 (0.1)	0.4 (0.3)	0.5 (0.3)
Mortality respiratory disease	24-Hour Average	0.1 (0.08)	0.04 (0.03)	0.09 (0.06)	0.1 (0.07)
Mortality cardiovascular disease all ages	24-Hour Average	0.1 (0.1)	0.06 (0.04)	0.1 (0.09)	0.1 (0.1)
Hospital admissions respiratory disease 65+ years	24-Hour Average	1.3 (0.9)	0.7 (0.5)	1.3 (0.9)	0.6 (0.4)
Hospital admissions cardiovascular disease 65+ years	24-Hour Average	1.3 (0.9)	0.7 (0.5)	1.3 (0.9)	0.6 (0.4)
Hospital admissions respiratory disease 15-64 years	24-Hour Average	0.6 (0.4)	0.2 (0.2)	0.5 (0.3)	0.6 (0.4)
ED visits asthma 1-14 years	24-Hour Average	0.1 (0.07)	0.03 (0.02)	0.08 (0.06)	0.12 (0.08)

The increased annual incidences for the health endpoints evaluated due to cumulative Proposal related NO₂ exposure were below one case per year for all health endpoints in Glenfield. The increased incidences for the cumulative Proposal were slightly above one case per year for three health endpoints in Casula and Moorebank (all-cause mortality for ages 30+ years, hospital admissions associated with respiratory disease for ages 65+ years due to daily exposure, and hospital admissions associated with cardiovascular disease for ages 65+ years due to daily exposure) as well as one health endpoint in Wattle Grove (all-cause mortality for ages 30+ years).

There would be an additional 1.4 deaths per year due to all causes among 30+ year-olds in Casula and Moorebank (equivalent to 14 additional deaths per 10 years) and an additional 1.2 deaths per year in Wattle Grove (equivalent to 12 additional deaths per 10 years), which may be attributed to annual exposure to emissions of NO_2 from the operation of the cumulative Proposal.

These calculations were based on a conservative assumption of 100% conversion of NO_x is NO₂. If this adjustment for fraction of NO_x that is NO₂ is applied to the ambient concentrations from the operation of the cumulative Proposal (refer to **Section 4.1.4**), the predicted NO_2 concentrations in air and the increased annual incidences would be reduced by 30%. That is, the greatest increased annual incidence would be at or below the threshold of one case per year. The increased annual incidences assuming this more realistic conversion are also presented in Table 4.20 (values shown in brackets).

It is also noted that the AQIA indicated that the cumulative NO₂ levels for all relevant averaging periods were below the current NEPM air quality standards.

Haalth and naint	Exposure period	Increa	Increased annual incidence (case per year)			
Health endpoint	Exposure period	Casula	Glenfield	Moorebank	Wattle Grove	
All-cause mortality all ages	24-Hour Average	0.002	0.0006	0.001	0.002	
Mortality respiratory disease- all ages	24-Hour Average	0.0003	0.0001	0.0003	0.0003	
Mortality cardiovascular disease- all ages	24-Hour Average	0.0006	0.0002	0.0006	0.0006	
Hospital admissions respiratory disease 65+ years	1- Hour Maximum	0.006	0.002	0.005	0.006	
ED visits asthma 1-14 years	24-Hour Average	0.007	0.002	0.006	0.008	

The increased annual incidences for the health endpoints evaluated due to the cumulative Proposal related SO₂ exposure were all well below one case per year. For the most sensitive health endpoint, there would be an additional 0.008 asthmarelated emergency department visit per year among 1-14 year-olds in Wattle Grove (equivalent to eight additional emergency department visits per 1,000 years), which may be attributed to daily exposure to emissions of SO₂ from the operation of the cumulative Proposal. Table 4.22: Summary of increased annual incidence associated with exposure to CO from the cumulative Proposal

Haalth and naint	Expective period	Increased annual incidence (case per year)			
Health endpoint	Exposure period	Casula	Glenfield	Moorebank	Wattle Grove
All-cause mortality 30+ years	8-Hour Average	0.0003	0.0002	0.0003	0.0003
Hospital admissions cardiac disease 65+ years	8-Hour Average	0.002	0.0009	0.002	0.0009
Hospital admissions cardiovascular disease 65+ years	8-Hour Average	0.0001	0.00006	0.0001	0.00006
Abbreviations: CO: Carbon Monoxide. ED visits asthma 1-14 years					

The increased annual incidences for the health endpoints evaluated due to the cumulative Proposal related CO exposure were all well below one case per year. For the most sensitive health endpoint, there would be an additional 0.002 hospital admission per year associated with cardiac disease among 65+ year-olds in Casula or Moorebank (equivalent to two additional hospital admissions per 1,000 years), which may be attributed to 8-hour exposure to emissions of CO from the operation of the Proposal or cumulative Proposal.

The excess lifetime cancer risks associated with the cumulative Proposal related exposure to benzene, 1,3-butadiene, and PAHs (as BAP TEQ), presented in **Table 4.23**, are all below or within the acceptable risk range of 10^{-6} to 10^{-4} .

The excess lifetime cancer risks associated with the cumulative Proposal related DPM exposure were all within the acceptable risk range of 10^{-6} to 10^{-4} . Therefore, there are no unacceptable cancer risks are expected in relation to long-term exposure to VOCs, DPM and PAHs in the surrounding local area.

Chemical	Excess lifetime cancer risk at maximum exposed receptor					
Chemical	Residential/School	Residential/School Recreational				
Benzene	5.1E-07	2.6E-08	1.0E-07			
1,3-Butadiene	1.0E-06	5.3E-08	2.0E-07			
DPM	9.1E-05	4.5E-06	1.7E-05			
PAHs (as BaP TEQ)	6.4E-09	2.2E-10	1.1E-09			
Abbreviations: BaP: Ben	zo(a)pyrene. DPM: Diesel Partic	ulate Matter. PAH: Polycyc	lic Aromatic Hydrocarbon. TEQ:			
Toxicity Equivalent						

Table 4.23: Summary of excess lifetime cancer risks associated with exposure toBenzene, 1,3-Butadiene, PAHs, and DPM from the cumulative Proposal

4.6 Uncertainties

It is important to evaluate uncertainties associated with the calculations and assumptions used in this air quality HRA so that the results of this risk assessment can be placed in perspective. This section identifies the potential sources of uncertainties, performs a sensitivity analysis for the key quantifiable uncertainties with plausible ranges, and presents a discussion of the significance and expected effects of sensitive variables on risk estimates.

4.6.1 Baseline health incidence

The baseline health incidence data used for the quantification of potential health risk are derived from statistics recorded by hospitals and doctors, reported by postcode of residence, and are dependent on the correct categorisation of health problems upon presentation at the hospital. There may be some individuals who do not seek medical assistance particularly with less serious conditions and hence there is expected to be some level of under-reporting of effects commonly considered in relation to morbidity. Quantitatively, the baseline health incidence data considered in this assessment is only a general indicator (not a precise measure) of the incidence of these health endpoints. Such limitation in baseline health incidence data may underestimate the risk.

4.6.2 Exposure point concentrations

The modelling of air emissions involved the use of a number of assumptions related to the operation of the Proposal. While the approach adopted in the AQIA utilised published peer-reviewed emission estimation techniques, currently available site-specific data, site-specific meteorology and terrain data, and approved dispersion models for the quantification of impacts in the surrounding areas, the overall approach adopted was generally conservative to ensure that where uncertainties are present, the impact is overestimated. In addition, use of the locations with maximum modelled air concentrations to estimate risks likely overestimates risks for the majority of people living, working, and recreating within the study area.

4.6.3 Toxicity assessment

<u>Health endpoints</u>

The health endpoints evaluated in this HRA are the health effects or outcomes where the most significant and robust positive associations with COPCs have been identified. These health endpoints do not include all possible subsets of effects that have been considered in various published studies, and may be insufficient to provide a thorough understanding of all of the potential toxic properties of air pollutants to which humans may be exposed. This uncertainty is considered inherent in any evaluation, but will be refined over time with the collection of additional data. The influence of this uncertainty may be either positive or negative.

Exposure-Response Function

As discussed in EnRisks (2014a), there is variability inherent in the studies used to estimate exposure-response functions. The variability is expected to reflect the local and regional variability in the characteristics of air pollutants to which the population is exposed and the variability within the exposed population. The exposure-response functions used in this HRA have been taken from the Australian studies or the most reliable international studies in the absence of Australian data. They are considered current, robust and relevant to the characterisation of impacts from COPCs.

This HRA assumed a linear exposure-response relationship between the air EPCs associated with the Proposal and the health endpoints evaluated. However, the shape of the exposure-response function and whether there is a threshold for some of the health endpoints remains uncertain.

Most currently available data have demonstrated a linear relationship and no evidence of a threshold; however, for long-term exposure-related mortality, a log-linear relationship is more plausible and should be considered if there is a potential for exposure to very high concentrations. In this assessment, the impact evaluated is localised with low level increases in concentration associated with the Proposal. At low levels, the assumption of a linear relationship is considered appropriate.

Unit Risk Factor for Diesel Particulate Matter

DPM is a complex mixture of thousands of gases and fine particles that contains more than 40 toxic air contaminants. Many of them are known or suspected carcinogens. In this HRA, the URF of 3 x 10^{-4} (µg/m³)⁻¹ published by OEHHA (1998) has been used in the calculation of excess lifetime cancer risks associated with DPM from the Proposal. This URF is consistent with the HIA for the MPE Stage 1 Project (Pacific Environment, 2015).

However, the URF derived by OEHHA was based on Garshick et al. (1988), a study that has been judged by several authoritative bodies to be inadequate for derivation of a discrete, quantitative estimate of human risk due to substantial uncertainties (HEI 1999, USEPA 2002, Hesterberg et al., 2011, WHO 1996). Therefore, this sensitivity analysis presents results using alternative USEPA approved URF.

An important issue in extrapolating results from the older epidemiology studies is that diesel exhaust in the older epidemiology studies are based on diesel exhaust composition that is very different to more contemporary diesel exhaust. Since 1990s, new and cleaner diesel engines, together with different diesel fuels, have replaced a substantial number of existing engines. Such changes have not only resulted in the quantitative reduction in mass emitted by new technology diesel engines, but have also resulted in qualitative differences in the composition of DPM emitted, with respect to both size and chemicals associated with the exhaust (Hesterberg et al. 2011). Therefore, the exposure-response relationship between DPM and lung cancer is likely to change as newer engines become more prevalent. USEPA evaluated the toxicology and epidemiology evidence related to carcinogenic effect of DPM in 2002, and concluded that even though the scientific evidence supported an association between exposure to diesel exhaust and lung cancer, the data available at that time were not sufficient to confidently estimate a URF. This conclusion

was based on a number of factors including equivocal evidence for the presence or absence of a dose-response relationship and uncertainties related to exposure (USEPA 2002).

USEPA estimated that the DPM URF could possibly range from 1×10^{-5} to $1 \times 10^{-3} (\mu g/m^3)^{-1}$, while acknowledging numerous uncertainties and assumptions in reaching this conclusion. USEPA has not revisited this issue since then.

WHO used data from studies in rats to estimate URF for DPM (WHO 1996). Using four different studies where lung cancer was the endpoint evaluated, WHO calculated a URF range of 1.6×10^{-5} to 7.1×10^{-5} per µg/m³ (mean value of 3.4×10^{-5} per µg/m³), which is near the low end of the USEPA URF range. Since the WHO values were derived based on animal studies, there are substantial uncertainties for applying them to humans.

The HEI Panel (2015) reviewed new epidemiology studies of diesel exhaust and lung cancer, including those that were key to the 2012 International Agency for Research on Cancer (IARC) evaluation of diesel exhaust (IARC 2012). This Panel focused on two studies, the Trucking Industry Particle Study (the Truckers Study; Garshick et al. 2012), and the Diesel Exhaust in Miners Study (DEMS) (Attfield et al. 2012, Silverman et al. 2012). In this evaluation, the Panel found that both the Truckers Study and DEMS were well-designed, well-conducted studies that made considerable progress toward addressing a number of the serious limitations identified in previous studies of diesel exhaust and lung cancer. The studies included better metrics to specifically quantify diesel exposure, and used better models of historical exposures. The HEI Panel concluded that the studies would be useful for quantitative estimates of historical exposures to diesel exhaust, and thus be appropriate to develop more robust URF values for quantitative HRA. Although there have been some attempts to use these studies to develop quantitative estimates of cancer risk (Vermeulen et al. 2014, Crump 2014, Morfeld and Spallek 2015), the numbers generated can vary considerably. USEPA will likely be following the recommendation by HEI Panel and developing URF values for DPM in the future, but the timing is uncertain.

Given the above uncertainties, a sensitivity analysis was conducted in this HRA by evaluating the excess lifetime cancer risks associated with DPM emitted from the operation of the Proposal using the USEPA URF range of 10^{-5} to 10^{-3} (μ g/m³)⁻¹. This range, which encompasses the various URF values developed by different regulatory agencies and research groups, better reflects the uncertainty of defining the exposure-response curve for assessing potential cancer risk from diesel exhaust, yet allows comparisons across different exposure scenarios.

Results of the sensitivity analysis are presented in **Table 4.24** (Proposal) and **Table 4.25** (cumulative Proposal).

The excess lifetime cancer risks calculated using the low end USEPA URF value of $10^{-5} (\mu g/m^3)^{-1}$ were all below or within the acceptable risk range of 10^{-6} to 10^{-4} . The excess lifetime cancer risks calculated using the high end USEPA URF value of $10^{-3} (\mu g/m^3)^{-1}$ were within the acceptable risk range of 10^{-6} to 10^{-4} for commercial/industrial and recreational receptors, but above the acceptable risk range for residential/school receptors. These calculations were based on a conservative assumption that all PM_{2.5} is DPM.

Table 4.24: Summary of excess lifetime cancer risks associated with exposure to DPM from the operation of the Proposal – Sensitivity analysis						
Chemical	Unit risk factor					
Chemical	4)	(µg/m ³) ⁻¹ Residential/ School		Recreational	Commercial/ Industrial	
DPM	1.0E-05	Low End (USEPA 2002)	4.3E-07	8.2E-09	7.2E-08	
DPM	1.0E-03	High End (USEPA 2002)	4.3E-05	8.2E-07	7.2E-06	
Source: USEPA.	2002. Health	Assessment Document	t for Diesel Engine Exhau	st. EPA/600/8-90/057F	. May.	

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Table 4.25: Summary of excess lifetime cancer risks associated with exposure to DPM from the cumulative Proposal – Sensitivity analysis					
Chemical Unit risk factor Excess lifetime cancer risk at maximum exposed receptor					naximum exposed
Cnemical	nemicai (μg/m ³) ⁻¹		Residential/ School	Recreational	Commercial/ Industrial
DPM	1.0E-05	Low End (USEPA 2002)	3.0E-06	1.5E-07	5.6E-07
DPM	1.0E-03	High End (USEPA 2002)	3.0E-04	1.5E-05	5.6E-05

4.7 Summary of health risks from air emissions

The air quality HRA evaluated a range of health endpoints associated with the key air pollutants, including increases in mortality and morbidity as well as excess lifetime cancer risks.

The HRA indicates the following:

- Health impacts from short-term and long-term exposure to PM₁₀ and PM_{2.5} as a result of the Proposal would be low in the surrounding communities and are below the acceptable risk level (i.e., fewer than one increased case per year of premature mortality, hospital admissions, and emergency department visits associated with cardiovascular and respiratory diseases or asthma);
- Health impacts from short-term and long-term exposure to NO_x, following adjustment for the fraction attributable to NO₂, as a result of the Proposal would be low in surrounding communities and are below the acceptable risk level (i.e., less than or equal to one increased case per year of premature mortality, hospital admissions, and emergency department visits associated with cardiovascular and respiratory diseases or asthma);
- Short-term exposure to SO₂ and CO results in negligible impacts in the surrounding communities and are below the acceptable risk level (i.e., orders of magnitude below one increased case per year of premature mortality, hospital admissions, and emergency department visits associated with cardiovascular and respiratory diseases or asthma);
- Excess lifetime cancer risks for residents/school students, commercial/industrial workers, and recreational populations within the HRA study area are below levels of concern (i.e., within or below the established acceptable cancer risk range of 10⁻⁶ to 10⁻⁴).

In summary, there are no significant adverse health effects expected in relation to short-term and long-term exposure to key air pollutants in the surrounding communities as a result of the Proposal. The increased annual incidences for the health endpoints evaluated were all below or equal to the acceptable risk of one additional case per year. The excess lifetime cancer risks were also within or below the acceptable risk range of 10^{-6} to 10^{-4} .

HEALTH RISK ASSESSMENT - NOISE 5.

The noise HRA has been conducted to evaluate the potential health risks to surrounding communities from exposure to operational noise. Construction phase impacts for the Proposal would be temporary and are demonstrated in the Noise and Vibration Impact Assessment (Wilkinson Murray, 2016) to comply with the relevant standards and are therefore not considered further in this HRA.

This noise HRA replicates the methodology for the MPE Stage 1 Project, which provides an overview of the health effects of environmental noise and presents risks for the MPE Stage 1 Project (Pacific Environmental, 2015).

5.1 **Exposure assessment**

Similar to the air HRA, the noise HRA considers the potential health risks to surrounding communities from exposure to operational noise for the following scenarios.

- Scenario 1: Operation of the Proposal, as described in Section 2.4. •
- Scenario 2: Cumulative operation, incorporating a combined precinct total 750,000 TEU (250,000 TEU for the MPE Stage 1 and 500,000 TEU for the MPW Stage 2) plus 515,000 m² of warehousing (300,000 m² for MPE Stage 2 and 215,000 m² for MPW Stage 2).

Residential receivers identified as potentially being the most affected in the vicinity of the Proposal site are located in the suburbs of Casula, Glenfield, and Wattle Grove. In addition to residential receivers, a number of potentially affected non-residential receivers have been assessed, including All Saints Senior College and the Casula Powerhouse (located to the west of the site across the Georges River). The nearest industrial receiver to the Proposal is the Defence Joint Logistics Unit (DJLU), located immediately north of the Proposal site. Table 5-1 presents a summary of the receivers potentially most affected by noise near the Proposal site.

Table 5-1: Potentially affected receivers				
Receiver/Suburb	Population	Distance (m) to Proposal site		
Casula	Residential	820		
Glenfield	Residential	1,830		
Wattle Grove	Residential	360		
All Saints Senior College (S1)	Educational	1,600		
Casula Powerhouse (S2)	Educational	1,020		
DJLU (I2)	Industrial	400		

5.1.1 Baseline noise

The existing ambient noise environment at locations representative of the potentially most affected residential receivers in Casula, Glenfield and Wattle Grove were established through long-term background noise monitoring conducted in accordance with the NSW Industrial Noise Policy. The existing ambient noise levels (the equivalent noise levels averaged over a time period [LA_{eq, period}]) are presented in **Table 5-2**.

Cuburb		LA _{eq, period} (dBA)	
Suburb	Day	Evening	Night Time
Casula	55	54	53
Glenfield	48	47	44
Wattle Grove	55	49	46

5.1.2 Operational noise

The predicted operational noise levels at nearby potentially affected receivers are presented in Table 5-3. Transient noise events may have the potential to cause sleep disturbance and the maximum noise levels (L_{Amax}) at sensitive residential receivers due to the transient noise events are also presented in Table 5-3.

Dessiver (Suburb		LA _{max} (dBA)		
Receiver/Suburb	Day	Evening	Night Time	Night Time
Casula	21	21	20	35
Glenfield	<20	<20	<20	26
Wattle Grove	25	25	23	53
All Saints Senior College (S1)	<20	<20	<20	NA
Casula Powerhouse (S2)	<20	<20	<20	NA
DJLU (I2)	44	44	38	NA
Notes:			· · ·	
Daytime 7:00am-6:00pm; Evening 6:00pr	n–10:00pm; N	light time 10:00pm	7:00am.	

The cumulative noise levels (LA_{eq, period}) at sensitive receivers has been predicted by combining the noise models developed for each site. The cumulative L_{Amax} at sensitive receivers during night time as a result of transient noise events associated with the operation of the cumulative Proposal has been taken as the higher value between the two sites.

The predicted cumulative operational noise levels are presented in Table 5-4.

Bessiver (Suburb		LA _{max} (dBA)			
Receiver/Suburb	Day	Evening	Night Time	Night Time	
Casula	33	33	36	47	
Glenfield	22	22	27	26	
Wattle Grove	27	27	29	53	
All Saints Senior College (S1)	29	29	34	NA	
Casula Powerhouse (S2)	26	26	29	NA	
DJLU (I2)	56	56	57	NA	

Night time noise levels were predicted under adverse meteorology conditions.

Abbreviations: dBA: A-weighted decibel

5.2 **Toxicity assessment**

Exposure to noise is associated with direct auditory and non-auditory health effects, including cardiovascular disease, cognitive impairment, sleep disturbance, tinnitus, annoyance, and hearing impairment (WHO, 1999; WHO, 2011).

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 (Pacific Environment, 2015; WHO, 2011). High blood pressure is a major risk factor for cardiovascular disease and small increases in blood pressure from road traffic noise may impact on public health, particularly for the elderly. Sleep disturbance also contributes to

cardiovascular risk with older people again the more susceptible risk group (Pacific Environment, 2015).

Children may be particularly vulnerable to the effects of noise on cognitive impairment and noise may interfere with learning at a critical developmental stage. Epidemiological studies show effects of chronic noise exposure on tasks involving central processing and language, such as reading, comprehension, memory and attention. Exposure during critical periods of learning at school could potentially impair development and have a lifelong effect on educational attainment (Pacific Environment, 2015). 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'.

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) (Pacific Environment, 2015).

The WHO has established guidelines for community noise to protect against the key health effects of annoyance, sleep disturbance, and cognitive impairment (WHO, 1999). The WHO guidelines are summarised in **Table 5-5**. The WHO community noise guidelines apply to total noise including existing ambient noise, not just the increment from a particular source.

Critical health effect	L _{Aeq, period} (dBA)	Time Base (hour)	L _{Amax} (dBA)
Serious annoyance, daytime and evening	55	16	
Moderate annoyance , daytime and evening	50	16	
Disturbance of speech intelligibility and moderate annoyance, daytime and evening	35	16	
Sleep disturbance, night time	30	8	45
Sleep disturbance, window open, night time	45	8	60
Disturbance of speech intelligibility, information extraction, and message communications, daytime	35	During class	
Sleep disturbance, sleep time	30	During sleep	45
Annoyance, during play, daytime	55	During play	
	Serious annoyance, daytime and evening Moderate annoyance , daytime and evening Disturbance of speech intelligibility and moderate annoyance, daytime and evening Sleep disturbance, night time Sleep disturbance, window open, night time Disturbance of speech intelligibility, information extraction, and message communications, daytime Sleep disturbance, sleep time	Critical nearth errect(dBA)Serious annoyance, daytime and evening55Moderate annoyance, daytime and evening50Disturbance of speech intelligibility and moderate annoyance, daytime and evening35Sleep disturbance, night time30Sleep disturbance, window open, night time45Disturbance of speech intelligibility, information extraction, and message communications, daytime35Sleep disturbance, sleep time30	Critical health effectInterpreter (dBA)Interpreter (hour)Serious annoyance, daytime and evening5516Moderate annoyance , daytime and evening5016Disturbance of speech intelligibility and moderate annoyance, daytime and evening3516Sleep disturbance, night time308Sleep disturbance, window open, night time458Disturbance of speech intelligibility, information extraction, and message communications, daytime35During classSleep disturbance, sleep time30During sleep

5.3 Risk Characterisation

The risk characterisation was conducted by comparing the predicted noise levels to the corresponding health-based WHO guideline values. The ratio of the predicted noise level to the guideline is termed as hazard quotient (HQ). The HQ was estimated for each health effect listed in **Table 5-5**, at each applicable sensitive receiver, except that sleep disturbance is not applicable to the non-residential receivers because no people will sleep at those locations.

Sleep disturbance has been assessed using the night time noise levels while both annoyance and cognitive function have been assessed using the daytime (and evening in some cases) noise levels.

Since the predicted noise levels have been modelled for outdoor receptors, the noise levels were reduced by 10 decibels (dB) when compared against the WHO guidelines for indoor environment to account for the attenuation of noise by structures. The maximum HQs for each of the three key health effect categories (i.e. annoyance, sleep disturbance, and cognitive impairment) at sensitive receivers are presented in this HRA to provide a conservative assessment of health risk associated with noise.

A HQ of less than or equal to 1 is considered to be an acceptable level (enHealth 2012b). It should be noted that a HQ of greater than 1 does not necessarily mean that adverse health effects will be observed; it just means that further assessment is warranted.

To place the results of the noise HRA in context, **Table 5.6** presents the HQs for existing ambient noise in the three nearby suburbs. All HQs are greater than 1 for annoyance, sleep disturbance, and cognitive impairment in the three surrounding suburbs, indicating that the existing ambient noise levels already exceed the health-based WHO guidelines.

Table 5.6: Hazard quotients for existing ambient noise											
Suburb	Annoyance	Sleep Disturbance	Cognitive Impairment								
Suburb	LA _{eq, period}	LA _{eq} , period	L _{Aeq} , period								
Casula	1.3	1.4	1.3								
Glenfield	1.1	1.1	1.1								
Wattle Grove	1.3	1.2	1.3								

Table 5-7 presents the HQs for operational noise associated with the Proposal at sensitive receivers. All HQs were less than or equal to 1 at all receivers, indicating that the operational noise alone from the Proposal does not pose an unacceptable risk to the health of these communities. When operational noise from the proposal is added to existing ambient noise, the existing ambient noise is the higher of the two values and HQs for the Proposal with existing noise would therefore be the same as those presented in **Table 5.6**.

Table 5-7: Hazard quotients for operational noise from the Proposal											
Dessiver (Coherth	Annoyance	Sleep Dist	turbance	Cognitive Impairment							
Receiver/Suburb	LA _{eq} , period	LA _{eq, period}	LAmax	L _{Aeq} , period							
Casula	0.4	0.4	0.6	0.3							
Glenfield	0.4	0.4	0.4	0.3							
Wattle Grove	0.5	0.5	1.0	0.4							
All Saints Senior College (S1)	0.4	NA	NA	0.3							
Casula Powerhouse (S2)	0.4	NA	NA	0.3							
DJLU (I2)	1.0	NA	NA	1.0							

Table 5-8 presents the HQs for operational noise from the cumulative Proposal. All HQs were less than or equal to 1 at the residential and educational receivers, indicating that the operational noise from the cumulative Proposal does not pose an unacceptable risk to the health of these communities.

The HQs were greater than 1 for annoyance and cognitive impairment at the nearest industrial receiver. It is noted, however, that the HQs for existing ambient noise already exceed 1 for annoyance and cognitive impairment and when background noise is added to the cumulative operational noise, there is no change in the HQ for total noise (refer **Table 5.10**).

Table 5-8: Hazard quotients for cumulative operational noise from the cumulative
Proposal

Dession (Cohord	Annoyance	Sleep Dist	turbance	Cognitive Impairment		
Receiver/Suburb	LA _{eq} , period	LA _{eq} , period	L _{Amax}	L _{Aeq} , period		
Casula	0.7	0.9	0.8	0.7		
Glenfield	0.4	0.6	0.4	0.3		
Wattle Grove	0.5	0.6	1.0	0.5		
All Saints Senior College (S1)	0.5	NA	NA	0.5		
Casula Powerhouse (S2)	0.5	NA	NA	0.5		
DJLU (I2)	1.3	NA	NA	1.3		

The WHO community noise guidelines apply to total noise, including existing ambient background, and the total noise levels were also evaluated in this HRA. The decibel is a log scale unit, therefore the total noise levels were calculated as the logarithmic sum of the predicted noise levels from all operations associated with the cumulative Proposal. This includes rail noise associated with MPE Stage 1, MPW Stage 2 plus the existing ambient background.

The data presented in **Table 5.9** show that the difference between the total noise level and the existing ambient noise level would not be detected in any suburb, indicating that the Proposal related noise has a minimal impact on the noise in the local area.

Daily LA _{eq, period} (dBA)											
Suburb Cumulative operational and rail noise		Existing Ambient	Total (cumulative Proposal + Existing Ambient)								
Casula	50	55	55								
Glenfield	43	48	48								
Wattle Grove	41	55	55								

 Table 5.9: Predicted total noise levels (from cumulative operational, rail and existing ambient background noise)

Abbreviations: dBA: A-weighted decibel.

Table 5.10 presents the HQs for total noise from cumulative Proposal and existing ambient background in the three nearby suburbs. All HQs were greater than 1 for annoyance, sleep disturbance, and cognitive impairment in the three surrounding suburbs. However, the HQs for total noise are similar to the HQs for existing ambient noise (refer **Table 5.6**), indicating that the existing ambient noise is the major contributor to the total noise, and the Proposal related noise

has a minimal impact on the noise in the local residential area (the HQs for existing ambient noise are unchanged).

Table 5.10: Hazard quotients for total noise levels from cumulative operation of MPE Stage 2, MPE Stage 1, MPW Stage 2 and existing ambient background										
Cuburb	Annoyance	Sleep Disturbance	Cognitive Impairment							
Suburb LA _{eq, period}		LA _{eq} , period	L _{Aeq} , period							
Casula	1.3	1.4	1.3							
Glenfield	1.1	1.1	1.1							
Wattle Grove	1.3	1.2	1.3							

5.4 Summary of health risks from noise

A HRA has been conducted to investigate the impact of operational noise associated with the Proposal on the annoyance, sleep disturbance, and cognitive impairment in local communities by comparing the predicted noise levels against the WHO community noise guidelines.

The HRA indicates the following:

- The existing ambient noise levels alone exceed the WHO community guidelines. •
- The noise from operation of Proposal, as well as cumulative Proposal meets the WHO community noise guidelines at all sensitive residential and educational receivers.
- A HQ greater than 1 was predicted for annoyance and cognitive impairment at the nearest • industrial receiver, however HQs for existing ambient noise already exceed in the area.
- There are multiple exceedances of the WHO community noise guidelines on annoyance, sleep ٠ disturbance, and/or cognitive impairment in the local communities from total noise, however HQs for existing ambient noise already exceed in the area.

6. CONCLUSIONS

A HRA has been conducted to assess potential health risk associated with air and noise emissions from the operation of the Proposal for surrounding communities and nearby non-residential sensitive receivers. The MPE site is surrounded by the suburbs of Casula, Wattle Grove, Glenfield, and Moorebank in southwestern Sydney. A review of the demographics of the population and the baseline health status has found that there are no significant differences in the health indicators between these communities and the rest of Sydney and NSW that would make these communities more vulnerable to the effects of environmental factors, such as air pollution or noise from the Proposal.

The air quality HRA evaluated a range of health endpoints associated with key air pollutants, $(PM_{10} \text{ and } PM_{2.5}, \text{ nitrogen oxides}, SO_2, CO, VOCs and PAHs), including increases in mortality and morbidity as well as excess lifetime cancer risks. The results of the air quality HRA found that the increases in mortality and morbidity due to the Proposal and cumulative Proposal were low and in most cases were negligible. The excess lifetime cancer risks were below or within the acceptable risk range. Therefore, there are no significant adverse health effects expected in relation to short-term and long-term exposure to key air pollutants associated with the operation of the Proposal and cumulative Proposal in the surrounding communities.$

The noise HRA has been conducted to investigate the impact of operational noise associated with Proposal on annoyance, sleep disturbance and cognitive impairment in local communities and nearby non-residential sensitive receivers. Predicted noise levels were compared against the WHO community noise guidelines. To place the results of the noise HRA in context, the existing ambient noise levels were reviewed and found to already exceed the WHO community guidelines in residential areas.

The noise from the Proposal and cumulative Proposal meet the WHO community noise guidelines at all sensitive residential and educational receivers. A HQ greater than 1 was predicted for annoyance and cognitive impairment at the nearest industrial receiver, however, the HQs for existing ambient noise already exceed 1 for annoyance and cognitive impairment and are unchanged with the addition of the Proposal.

The total noise (cumulative Proposal plus existing background) exceeds the WHO community noise guidelines on annoyance, sleep disturbance, and cognitive impairment in all the three surrounding suburbs, however the Proposal related noise is expected to have a minimal additional impact on the noise in the local area above existing baseline levels.

Mitigation measures and monitoring are considered in the AQIA and Noise Impact Assessment, in accordance with the MPE Concept Plan Approval and Statement of Commitments.

7. **REFERENCES**

Attfield MD, Schleiff PL, Lubin JH, Blair A, Stewart PA, Vermeulen R, et al. 2012. The diesel exhaust in miners study: A cohort mortality study with emphasis on lung cancer. J Natl Cancer Inst 104:869–883.

Burgers M, Walsh S. 2002, Exposure Assessment and Risk Characterisation for the Development of a PM2.5 Standard, National Environment Protection Council. September.

Canadian Council of Ministers for the Environment (CCME). 2010. Canadian Soil Quality Guidelines, Carcinogenic and Other Polycyclic Aromatic Hydrocarbons (PAHs) (Environmental and Human Health Effects), Scientific Criteria Document (revised), Quebec.

Cesaroni G, Badaloni C, Gariazzo C, Stafoggia M, Sozzi R, Davoli M, et al. 2013. Long-term exposure to urban air pollution and mortality in a cohort of more than a million adults in Rome. Environ Health Perspect 121:324–331.

CHETRE. 2007. Health Impact Assessment – A Practical Guide - Centre for Health Equity Training, Research and Evaluation. University of New South Wales, Research Centre for Primary Health Care and Equity.

Crump. 2014. Meta-Analysis of Lung Cancer Risk from Exposure to Diesel Exhaust: Study Limitations. Environmental Health Perspectives Volume 122 Number 9, September.

enHealth. 2012a. Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards. Department of Health and Ageing and enHealth Council, Commonwealth of Australia.

enHealth. 2012b. Exposure Factors Guide. enHealth Council.

EnRisks. 2014a. Technical Working Paper: Human Health Risk Assessment – Moorebank Intermodal Terminal. September.

EnRisks. 2014b. Technical Working Paper: Moorebank Intermodal Terminal Health Impact Assessment. September.

Environment Protection Authority. 2000. NSW Industrial Noise Policy. January.

Environment Protection and Heritage Council (EPHC). 2010. Expansion of the multi-city mortality and morbidity study. September.

Garshick E, Schenker MB, Muñoz A, Segal M, Smith TJ, Woskie SR, Hammond SK, Speizer FE. 1988. A retrospective cohort study of lung cancer and diesel exhaust exposure in railroad workers. Am Rev Respir Dis 137:820–825.

Garshick E, Laden F, Hart JE, Davis ME, Eisen EA, Smith TJ. 2012. Lung cancer and elemental carbon exposure in trucking industry workers. Environ Health Perspect 120:1301–1306.

Health Effects Institute (HEI). 1999. Diesel Emissions and Lung Cancer: Epidemiology and Quantitative Risk Assessment. June. HEI. 2009. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Research Report 140.

HEI. 2015. Diesel Emissions and Lung Cancer: An Evaluation of Recent Epidemiological Evidence for Quantitative Risk Assessment. November.

Hesterberg TW, Long CM, Sax SN, Lapin CA, McClellan RO, Bunn WB, Valberg PA. 2011. Particulate Matter in New Technology Diesel Exhaust (NTDE) is Quantitatively and Qualitatively Very Different from That Found in Traditional Diesel Exhaust (TDE). J Air Waste Manag Assoc. 61(9):894-913.

International Agency for Research on Cancer (IARC). 2012. Press Release No. 213. IARC: Diesel Engine Exhaust Carcinogenic. June.

Jalaudin B, Khalaj B, Sheppeard V, Morgan G. 2008. Air pollution and ED visits for asthma in Australian children: a case-crossover analysis. International Archives of Occupational and Environmental Health 81(8):967-74.

Katsouyanni K. 2006. APHEA Project: Air Pollution and Health: A European Approach. Epidemiology, Volume 17, Issue 6, p19.

Morgan, G., Broome, R., Jalaludin, B. (2013). Summary for Policy Makers of the Health Risk Assessment on air Pollution in Australia. Prepared for the National Environment Protection Council (NEPC). November 2013.

Morfeld P and Spallek M. 2015. Diesel engine exhaust and lung cancer risks – evaluation of the meta-analysis by Vermeulen et al. 2014. J Occup Med Toxicol. 10: 31.

National Environment Protection Council (NEPC). 2011. Methodology for Setting Air Quality Standards in Australia.

NEPC. 2013. National Environmental Protection (Assessment of Site Contamination) Amended Measure. Guideline on Derivation of Health-Based Investigation Levels.

National Health and Medical Research Council (NHMRC) Approach to Hazard Assessment for Air Quality, 2006.

Office of Environmental Health Hazard Assessment. 1998. Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant. Appendix III, Part B: Health Risk Assessment for Diesel Exhaust. May.

Ostro B. 2004. Outdoor Air Pollution: Assessing the environmental burden of disease at national and local levels., World Health Organisation.

Pacific Environment. 2015. Moorebank Precinct East (MPE) (formerly SIMTA) Intermodal Terminal Facility - Stage 1 Health Risk Assessment (SSD-6766). April.

Ramboll Environ (2016). Moorebank Precinct East Stage 2 - Air Quality Impact Assessment.

Silverman DT, Attfield MD. 2012. Response to: 'The Diesel Exhaust in Miners Study: A nested case-control study of lung cancer and diesel exhaust' and 'The Diesel Exhaust in Miners Study: A cohort mortality study with emphasis on lung cancer.' J Natl Cancer Inst 104:1848–1849.

Simpson R, Williams G, Petroeschevsky A, Best T, Morgan G, Denison L, Hinwood A, Neville G, Neller A. 2005. The short-term effects of air pollution on hospital admissions in four Australian cities. Aust N Z J Public Health. Jun;29(3):205-12.

South Western Sydney Local Health District (SWSLHD). 2014. Liverpool Local Government Area Health Profile 2014.

Toxicos (2012). Preliminary screening health risk assessment and literature review. Moorebank Intermodal Freight Terminal, Moorebank NSW. November 2012.

United States Environmental Protection Agency (USEPA). 1989. Risk assessment guidance for Superfund, Vol I. Human health evaluation manual (Part A) Interim final report, EPA/540/1-89/002, Washington, DC.

USEPA. 1991. Risk assessment guidance for Superfund, Vol I. Human health evaluation manual, Part B, Development of risk-based preliminary remediation goals. EPA/540/R-92/003, Washington, DC.

USEPA. 2002. Health Assessment Document for Diesel Engine Exhaust. EPA/600/8-90/057F. May.

USEPA. 2005. Particulate Matter Health Risk Assessment for Selected Urban Area. Office of Air Quality Planning and Standards.

USEPA. 2009. Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual, (Part F, Supplemental Guidance for Inhalation Risk Assessment), Washington, D.C.

USEPA. 2010. Quantitative Health Risk Assessment for Particulate Matter, Office of Air Quality Planning and Standards.

USEPA. 2012. Provisional Assessment of Recent Studies on Health Effects of Particulate Matter Exposure, National Center for Environmental Assessment RTP Division, Office of Research and Development.

Vermeulen R, Silverman DT, Garshick E, Vlaanderen J, Portengen L, Steenland K. 2014. Exposure-response estimates for diesel engine exhaust and lung cancer mortality based on data from three occupational cohorts. Environ Health Perspect 122:172–177.

Wilkinson-Murray. 2016. MPE Stage 2 Noise and Vibration Impact Assessment. Report No. 12186-S2. November 2016

World Health Organization (WHO). 1996. Diesel fuel and exhaust emissions. Environmental Health Criteria 171. Geneva.

WHO. 1999. Guidelines for Community Noise. April.

APPENDIX 1 FIGURES

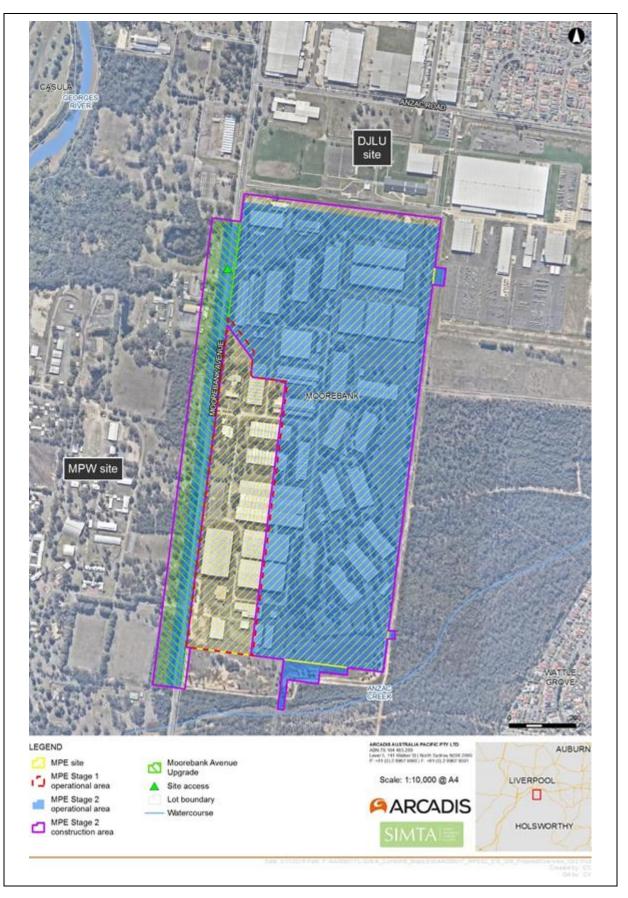


Figure A1-1: Overview of the proposal

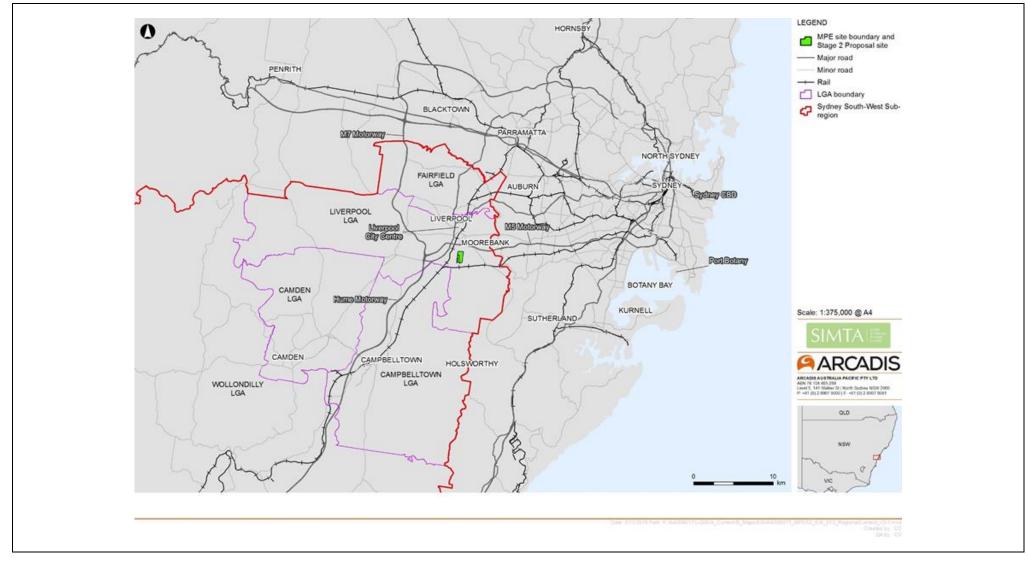


Figure A1-2: Regional context

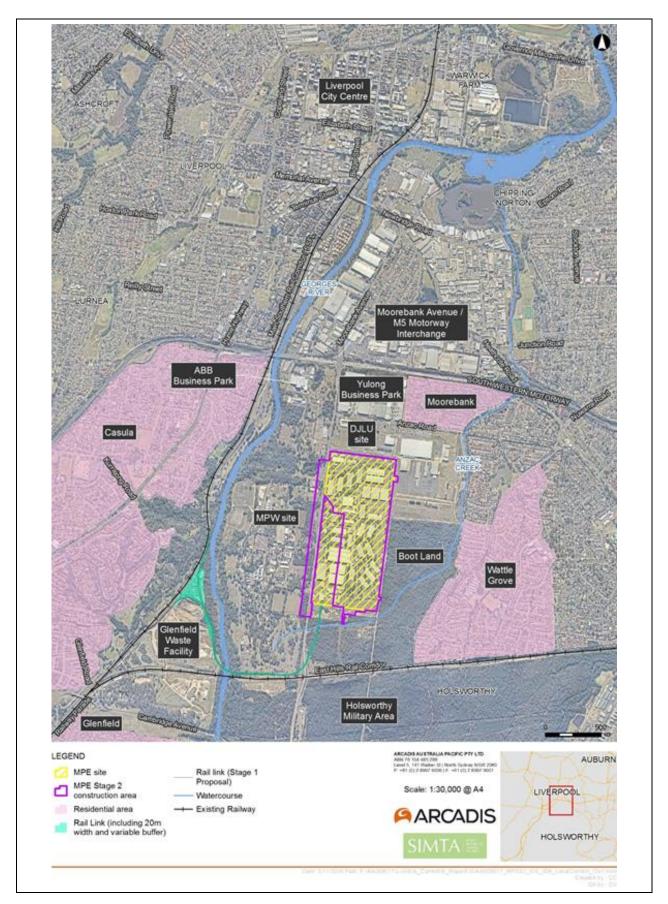


Figure A1-3: Local context

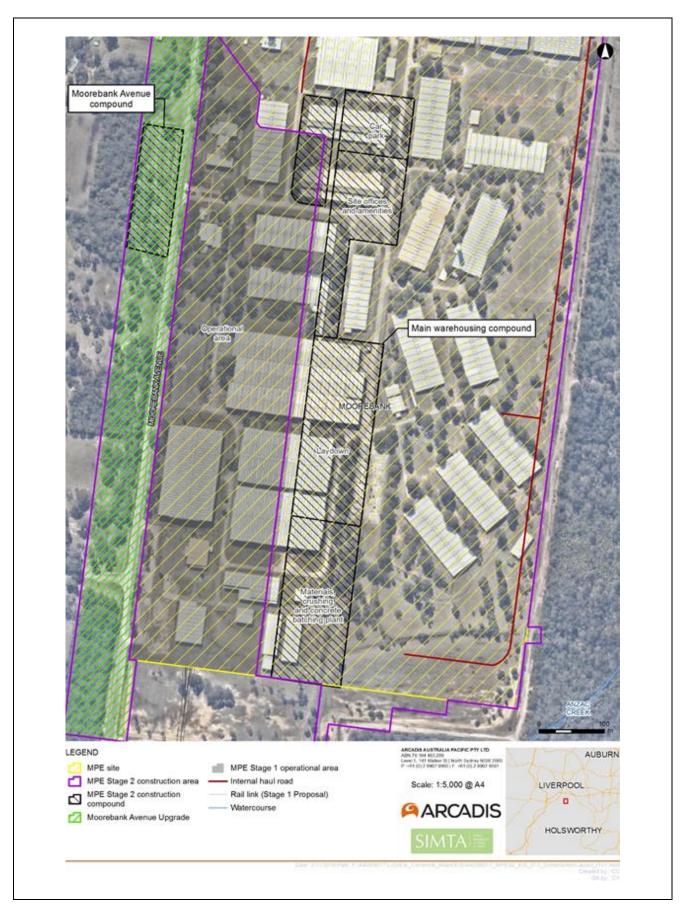


Figure A1-4: Construction layout

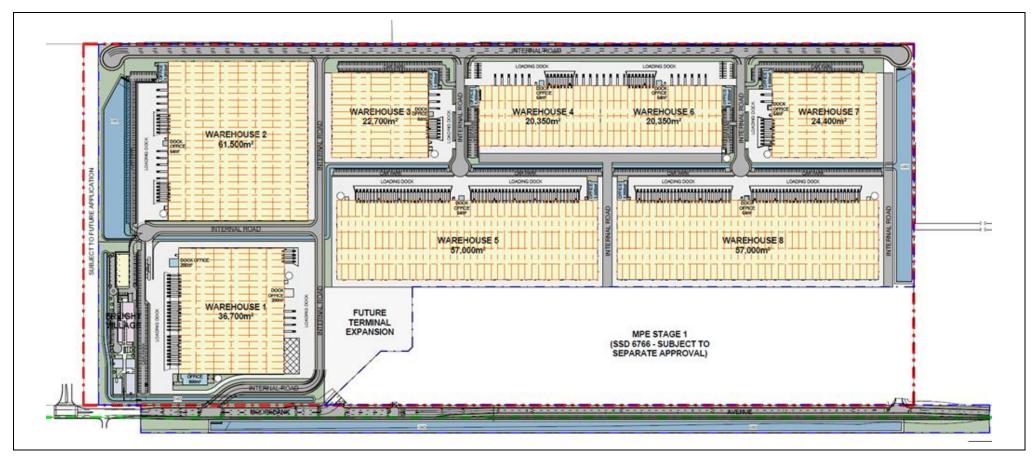


Figure A1-5: Warehouse layout

APPENDIX 2 CALCULATION OF INCREASED ANNUAL INCIDENCE FOR MORTALITY AND MORBIDITY ENDPOINTS

Table A-1 Calculation of Increased Annual Incidence for Mortality and Morbidity Endpoints - PM10 (Proposal)

Endpoint:	Mortality - All Causes	Mortality - All Causes	Mortality - Cardiovascular Disease	Hospital Admissions - Respiratory Disease	Hospital Admissions - Cardiac Disease	Hospital Admissions - Pneumonia and Bronchitis	Hospital Admissions - Respiratory Disease	ED Visits Asthma	
Exposure Period:	Annual Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	
Age Group:	30+ vears	All ages	All ages	65+ vears	65+ years	65+ years	15-64 years	1-14 vears	
β (Exposure Response Function per 1 μg/m ³ Increase in PM ₁₀):		0.002	0.002	0.003	0.002	0.0013	0.003	0.015	
Baseline Health Incidence Rate (per person)		0.005289	0.00155	0.04476	0.09159	0.01236	0.00899	0.00804	
	0.01005	0.003203	0.00135	0.01170	0.09139	0.01250	0.00033	0.00001	
Casula	-								
Total Population:	14,696	14,696	14,696	14,696	14,696	14,696	14,696	14,696	
% Population in Assessment Age-Group	49%	100%		10%		10%			
Average EPC within the Suburb (µg/m ³):	1.7E-02	1.7E-02	1.7E-02	1.7E-02	1.7E-02	1.7E-02	1.7E-02	1.7E-02	
Increased Annual Incidence (case per year):	0.01	0.00	0.00	0.00	0.00	0.000	0.00	0.01	
Glenfield				<u> </u>		<u> </u>		ļ	
Total Population:	7,558	7,558	7,558	7,558	7,558	7,558	7,558	7,558	
% Population in Assessment Age-Group	67%	100%	100%	14%	14%	14%	67%	19%	
Average EPC within the Suburb (µg/m ³):	2.0E-02	2.0E-02	2.0E-02	2.0E-02	2.0E-02	2.0E-02	2.0E-02	2.0E-02	
Increased Annual Incidence (case per year):	0.00	0.002	0.000	0.00	0.00	0.000	0.00	0.00	
Moorebank									
Total Population:	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595	
% Population in Assessment Age-Group	60%	100%	100%	13%	13%	13%	66%	22%	
Average EPC within the Suburb (µg/m ³):	4.7E-02	4.7E-02	4.7E-02	4.7E-02	4.7E-02	4.7E-02	4.7E-02	4.7E-02	
Increased Annual Incidence (case per year):	0.01	0.00	0.001	0.01	0.01	0.001	0.01	0.01	
Wattle Grove	<u> </u>			ļ		ļ			
Total Population:	8,192	8,192	8,192	8,192	8,192	8,192	8,192	8,192	
% Population in Assessment Age-Group	45%	100%		5%				26%	
Average EPC within the Suburb (µg/m ³):	4.5E-02	4.5E-02	4.5E-02	4.5E-02	4.5E-02	4.5E-02	4.5E-02	4.5E-02	
Increased Annual Incidence (case per year):	0.01	0.00	0.001	0.00		0.000		0.01	
Total Increased Annual Incidence (case per year) - All Suburbs	0.0	0.01	0.00	0.01	0.0	0.00	0.0	0.0	

Abbreviations: µg/m³: microgram per cubic meter ED: Emergency Department EPC: Exposure Point Concentration MPW: Moorebank Precinct West PM: Particulate Matter

Table A-2 Calculation of Increased Annual Incidence for Mortality and Morbidity Endpoints - PM2.5 (Proposal)

Endpoint:	Mortality - All Causes	Mortality - Cardiopulmonary	Mortality - Ischemic Heart Disease		Mortality - All Causes	Mortality - Cardiovascular Disease	Hospital Admissions - Respiratory Disease	Hospital Admissions - Cardiac Disease	Hospital Admissions - Cardiovascular Disease	Hospital Admissions - Ischemic Heart Disease	Hospital Admissions - COPD	Hospital Admissions - Pneumonia and Bronchitis	Hospital Admissions - Respiratory Disease	ED Visits Asthma
Exposure Period:	Annual Average	Annual Average	Annual Average	Annual Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average
Age Group:	30+ years	30+ years	30+ years	30+ years	All ages	All ages	65+ years	65+ years	65+ years	65+ years	65+ years	65+ years	15-64 years	1-14 years
β (Exposure Response Function per 1 µg/m ³ Increase in PM _{2.5}):	0.006	0.014	0.024	0.014	0.0023	0.0013	0.004	0.005	0.003	0.004	0.004	0.005	0.003	0.0015
Baseline Health Incidence Rate (per person)	0.01065	0.0049	0.00067	0.00035	0.00529	0.001551	0.04476	0.09159	0.09159	0.03331	0.01489	0.01236	0.00899	0.00804
Casula														
Total Population:	14,696									14,696	14,696			
% Population in Assessment Age-Group	49%					100%				10%	10 /		67%	
Average EPC within the Suburb (µg/m ³):	1.6E-02			1.6E-02		1.6E-02								
Increased Annual Incidence (case per year):	0.01	0.01	0.00	0.001	0.00	0.000	0.00	0.0	0.01	0.00	0.00	0.00	0.00	0.001
Glenfield				1				1		1		1	1	
Total Population:	7.558													
% Population in Assessment Age-Group	67%			67%	100%	100%				14%			67%	
Average EPC within the Suburb (µg/m ³):	1.9E-02			1.9E-02		1.9E-02							1.9E-02	
Increased Annual Incidence (case per vear):	0.01	0.01	0.002	0.000	0.002	0.000	0.00	0.01	0.01	0.00	0.001	0.001	0.00	0.000
Moorebank												1		
Total Population:	7,595	7,595	7,595	7,595		7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595	
% Population in Assessment Age-Group	60%		60%		100%	100%	13%	13%	13%	13%	13%	13%	66%	
Average EPC within the Suburb (µg/m ³):	4.6E-02			4.6E-02		4.6E-02			4.6E-02	4.6E-02	4.6E-02			
Increased Annual Incidence (case per year):	0.01	0.01	0.00	0.001	0.00	0.001	0.01	. 0.0	0.01	0.01	0.00	0.00	0.01	0.001
Wattle Grove	L	1	1	1	1	L	1	1	1	1	1	1	1	1
Total Population:	8.192			8,192						8.192	8.192	8.192	8.192	
% Population in Assessment Age-Group	45%	45%	45%	45%	100%	100%	5.2%	5.2%	5.2%	5.2%	5.2%	5.2%	69%	26%
Average EPC within the Suburb (ug/m ³):	4.4E-02	2 4.4E-02	4.4E-02	4.4E-02	4.4E-02	4.4E-02	4.4E-02	4.4E-02	4.4E-02	4.4E-02	4.4E-02	4.4E-02	4.4E-02	2 4.4E-02
Increased Annual Incidence (case per vear):	0.01	0.01	0.00	0.001	0.00	0.001	0.00	0.01	0.01	0.00	0.001	0.001	0.01	0.001
Total Increased Annual Incidence (case per year) - All Suburbs	0.0	0.0	0.01	0.00	0.01	0.00	0.0	0.0	0.0	0.01	0.01	0.01	0.0	0.00

Abbreviations: μq/m³: microgram per cubic meter COPD: Chronic Obstructive Pulmonary Disease ED: Emergency Department EPC: Exposure Point Concentration MPW: Moorebank Precinct West PM: Particulate Matter

Table A-3 Calculation of Increased Annual Incidence for Mortality and Morbidity Endpoints - NO2 (Proposal)

Endpoint:	Mortality - All Causes	Mortality - Cardiovascular	rdiovascular Respiratory		Mortality-	Mortality - Cardiovascular Disease	Admissions - Respiratory	Hospital Admissions - Cardiovascular Disease	Hospital Admissions - Respiratory Disease	ED Visits Asthma
Exposure Period:	Annual Average	Annual Average	Annual Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average
Age Group:	30+ years	30+ years	30+ years	All ages	All ages	All ages	65+ years	65+ years	15-64 years	1-14 years
β (Exposure Response Function per 1 µg/m ³ Increase in NO ₂):	0.0028	0.0028	0.0028	0.001	0.0023	0.001	0.003	0.0014	0.001	0.0006
Baseline Health Incidence Rate (per person)	0.01065	0.002987	0.00050	0.00529	0.00050	0.001551	0.04476	0.09159	0.00899	0.00804
Casula										
Total Population:	14,696	14,696	14,696	14,696		14,696	14,696	14,696	14,696	
% Population in Assessment Age-Group	49%	49%	49%	100%	100%	100%	10%	10%	67%	
Average EPC within the Suburb (µg/m ³):	4.8E-01	4.8E-01	4.8E-01	4.8E-01		4.8E-01	4.8E-01	4.8E-01	4.8E-01	
Increased Annual Incidence (case per year):	0.1	0.0	0.00	0.0	0.01	0.0	0.1	0.1	0.0	0.01
Glenfield										
Total Population:	7,558	7,558	7,558	7,558	7,558	7,558	7,558	7,558	7,558	7,558
% Population in Assessment Age-Group	67%	67%	67%	100%	100%	100%	14%	14%	67%	19%
Average EPC within the Suburb (µg/m ³):	4.8E-01	4.8E-01	4.8E-01	4.8E-01	4.8E-01	4.8E-01	4.8E-01	4.8E-01	4.8E-01	4.8E-01
Increased Annual Incidence (case per year):	0.1	0.0	0.00	0.0	0.00	0.01	0.1	0.1	0.0	0.00
Moorebank		ļ	ļ		ļ	ļ		ļ	ļ	
Total Population:	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595
% Population in Assessment Age-Group	60%	60%	60%	100%	100%	100%	13%	13%	66%	
Average EPC within the Suburb (ug/m ³):	1.3E+00	1.3E+00	1.3E+00	1.3E+00	1.3E+00	1.3E+00	1.3E+00	1.3E+00	1.3E+00	1.3E+00
Increased Annual Incidence (case per year):	0.2	0.0	0.01	0.1	0.01	0.02	0.2	0.2	0.1	0.01
Wattle Grove		<u> </u>	<u> </u>	ļ	<u> </u>	ļ		ļ	ļ	ļ
Total Population:	8,192	8,192	8,192	8.192	8.192	8,192	8,192	8,192	8,192	8,192
% Population in Assessment Age-Group	45%	45%	45%	100%	100%	100%	5.2%	5.2%	69%	
Average EPC within the Suburb (µg/m ³):	1.1E+00	1.1E+00					1.1E+00	1.1E+00	1.1E+00	
Increased Annual Incidence (case per year):	0.1	0.0	0.01	0.0		0.01	0.1	0.1	0.1	
Total Increased Annual Incidence (case per year) - All Suburbs	0.5	0.1	0.0	0.2	0.0	0.0	0.4	0.4	0.2	0.0

Abbreviations:

Jup/m³: microgram per cubic meter ED: Emergency Department EPC: Exposure Point Concentration MPW: Morebank Precinct West No₂: Nitrogen Dioxide Table A-4 Calculation of Increased Annual Incidence for Mortality and Morbidity Endpoints - SO2 (Proposal)

Endpoint:	Mortality - All Causes	Mortality- Respiratory	Mortality - Cardiovascular Disease	Hospital Admissions - Respiratory Disease	ED Visits Asthma
Exposure Period:	24-Hour Average	24-Hour Average	24-Hour Average	1-Hour Maximum	24-Hour Average
Age Group:	All ages	All ages	All ages	15-64 years	1-14 years
β (Exposure Response Function per 1 µg/m ³ Increase in SO ₂):	0.0006	0.0013	0.0008	0.002	0.008
Baseline Health Incidence Rate (per person)	0.00529	0.00050	0.001551	0.00899	0.00804
		-	•		
Casula					
Total Population:	14,696		14,696		
% Population in Assessment Age-Group	100%	100%	100%	67%	23%
Average EPC within the Suburb (µg/m ³):	1.0E-02	1.0E-02	1.0E-02	1.0E-02	1.0E-02
Increased Annual Incidence (case per year):	0.000	0.0001	0.0002	0.002	0.002
Glenfield					
Total Population:	7,558	7,558	7,558	7,558	7,558
% Population in Assessment Age-Group	100%	100%	100%	67%	
Average EPC within the Suburb (µg/m ³):	1.4E-02	1.4E-02	1.4E-02	1.4E-02	1.4E-02
Increased Annual Incidence (case per year):	0.0003	0.00007	0.0001	0.001	0.001
Moorebank					
Total Population:	7,595	7,595	7,595	7,595	7,595
% Population in Assessment Age-Group			100%		
Average EPC within the Suburb (µg/m ³):	3.1E-02	3.1E-02	3.1E-02	3.1E-02	3.1E-02
Increased Annual Incidence (case per year):	0.0007	0.0002	0.0003	0.003	0.003
Wattle Grove	1	1	1	1	1
Total Population:	8,192	8,192	8,192	8,192	8,192
% Population in Assessment Age-Group	100%	100%	100%	69%	26%
Average EPC within the Suburb (µg/m ³):	3.1E-02	3.1E-02	3.1E-02	3.1E-02	3.1E-02
Increased Annual Incidence (case per year):	0.0008	0.0002	0.0003	0.003	0.004
Total Increased Annual Incidence (case per year) - All Suburbs	0.002	0.0005	0.001	0.01	0.01

Abbreviations: µg/m³: microgram per cubic meter ED: Emergency Department EPC: Exposure Point Concentration MPW: Moorebank Precinct West SO₂: Sulfur Dioxide

 Table A-5 Calculation of Increased Annual Incidence for Mortality and Morbidity Endpoints - CO (Proposal)

Endpoint:	Mortality -	Hospital Admissions - Cardiac Disease	Hospital Admissions - Cardiovascular Disease
Exposure Period:	8-Hour Average	8-Hour Average	8-Hour Average
Age Group:	30+ years	65+ years	65+ years
β (Exposure Response Function per 1 µg/m ³ Increase in CO):	0.000001	0.000003	0.0000014
Baseline Health Incidence Rate (per person)	0.01065	0.09159	0.01236

Casula			
Total Population:	14,696	14,696	14,696
% Population in Assessment Age-Group	49%	10%	10%
Average EPC within the Suburb (μ g/m ³):	7.5E-01	7.5E-01	7.5E-01
Increased Annual Incidence (case per year):	0.0001	0.000	0.00002
Glenfield			
Total Population:	7,558	7,558	7,558
% Population in Assessment Age-Group	67%	14%	14%
Average EPC within the Suburb (µg/m ³):	9.7E-01	9.7E-01	9.7E-01
Increased Annual Incidence (case per year):	0.00005	0.0003	0.00002
Moorebank			
Total Population:	7,595	7,595	7,595
% Population in Assessment Age-Group	60%	13%	13%
Average EPC within the Suburb (µg/m ³):	2.1E+00	2.1E+00	2.1E+00
Increased Annual Incidence (case per year):	0.0001	0.0006	0.00004
Wattle Grove			
Total Population:	8,192	8,192	8,192
% Population in Assessment Age-Group	45%	5.2%	5.2%
Average EPC within the Suburb (µg/m ³):	2.3E+00	2.3E+00	2.3E+00
Increased Annual Incidence (case per year):	0.0001	0.0003	0.00002
Total Increased Annual Incidence (case per year) - All Suburbs	0.0003	0.001	0.0001

Abbreviations:

μg/m³: microgram per cubic meter CO: Carbon Monoxide EPC: Exposure Point Concentration MPW: Moorebank Precinct West

Table A-6 Calculation of Increased Annual Incidence for Mortality and Morbidity Endpoints - PM10 (cumulative Proposal)

Endpoint:	Mortality - All Causes	Mortality - All Causes	Mortality - Cardiovascular Disease	Hospital Admissions - Respiratory Disease	Hospital Admissions - Cardiac Disease	Hospital Admissions - Pneumonia and Bronchitis	Hospital Admissions - Respiratory Disease	ED Visits Asthma
Exposure Period:	Annual Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average
Age Group:	30+ vears	All ages	All ages	65+ vears	65+ years	65+ years	15-64 years	1-14 vears
β (Exposure Response Function per 1 μg/m ³ Increase in PM ₁₀):	0.004	0.002	0.002	0.003	0.002	0.0013	0.003	0.015
Baseline Health Incidence Rate (per person)		0.005289	0.00155	0.04476	0.09159	0.01236	0.00899	0.00804
	•							
Casula	•							
Total Population:	14,696	14,696	14,696	14,696	14,696	14,696	14,696	14,696
% Population in Assessment Age-Group	49%	100%	100%	10%	10%	10%	67%	23%
Average EPC within the Suburb (µg/m ³):	2.3E-01	2.3E-01	2.3E-01	2.3E-01	2.3E-01	2.3E-01	2.3E-01	2.3E-01
Increased Annual Incidence (case per year):	0.07	0.04	0.01	0.05	0.06	0.006	0.06	0.09
Glenfield								
Total Population:	7,558	7,558	7,558	7,558	7,558	7,558	7,558	7,558
% Population in Assessment Age-Group	67%	100%	100%	14%	14%	14%	67%	19%
Average EPC within the Suburb (µg/m ³):	1.7E-01	1.7E-01	1.7E-01	1.7E-01	1.7E-01	1.7E-01	1.7E-01	1.7E-01
Increased Annual Incidence (case per year):	0.04	0.01	0.004	0.02	0.03	0.003	0.02	0.03
Moorebank								
Total Population:	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595
% Population in Assessment Age-Group	60%	100%				13%		22%
Average EPC within the Suburb (µg/m ³):	3.6E-01	3.6E-01	3.6E-01	3.6E-01	3.6E-01	3.6E-01	3.6E-01	3.6E-01
Increased Annual Incidence (case per year):	0.07	0.03	0.009	0.05	0.06	0.006	0.05	0.07
Wattle Grove	1	I	1	1		1	I	
Total Population:	8,192	8,192	8,192	8,192	8,192	8,192	8,192	8,192
% Population in Assessment Age-Group			100%					26%
Average EPC within the Suburb ($\mu q/m^3$):	4.2E-01	4.2E-01	4.2E-01	4.2E-01	4.2E-01	4.2E-01	4.2E-01	4.2E-01
Increased Annual Incidence (case per year):	0.07			0.02		0.003		
Total Increased Annual Incidence (case per year) - All Suburbs	0.2	0.1	0.03	0.1	0.2	0.02	0.2	0.3

Abbreviations:

µg/m³: microgram per cubic meter ED: Emergency Department EPC: Exposure Point Concentration MPE: Moorebank Precinct East MPW: Moorebank Precinct West PM: Particulate Matter

Table A-7 Calculation of Increased Annual Incidence for Mortality and Morbidity Endpoints - PM2.5 (cumulative Proposal)

Endpoint:			Mortality - Ischemic Heart Disease	Mortality - Lung Cancer	Mortality - All Causes	Cardiovascular	Admissions -	Admissions -	Hospital Admissions - Cardiovascular Disease	Hospital Admissions - Ischemic Heart Disease	Hospital Admissions - COPD	Hospital Admissions - Pneumonia and Bronchitis	Hospital Admissions - Respiratory Disease	ED Visits Asthma
Exposure Period:	Annual Average	Annual Average	Annual Average	Annual Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average
Age Group:	30+ years	30+ years	30+ years	30+ years	All ages	All ages	65+ vears	65+ years	65+ vears	65+ years	65+ vears	65+ years	15-64 years	1-14 years
β (Exposure Response Function per 1 μg/m ³ Increase in PM _{2.5}):	0.006	0.014	0.024	0.014	0.0023	0.0013	0.004	0.005	0.003	0.004	0.004	0.005	0.003	0.0015
Baseline Health Incidence Rate (per person)	0.01065	0.0049	0.00067	0.00035	0.00529	0.001551	0.04476	0.09159	0.09159	0.03331	0.01489	0.01236	0.00899	0.00804
Casula														
Total Population:	14,696	14,696	14,696	14,696	14,696	14,696	14,696	14,696	14,696	14,696	14,696	14,696	14,696	14,696
% Population in Assessment Age-Group	49%	49%	49%	49%	100%	100%	10%	10%	10%	10%	10%	10%	67%	23%
Average EPC within the Suburb (µg/m ³):	2.3E-01	2.3E-01	2.3E-01	2.3E-01	2.3E-01	2.3E-01		2.3E-01	2.3E-01	2.3E-01	2.3E-01	2.3E-01	2.3E-01	2.3E-01
Increased Annual Incidence (case per year):	0.1	0.1	0.03	0.008	0.04	0.007	0.06	0.2	0.09	0.05	0.02	0.02	0.06	0.009
Glenfield														
Total Population:	7,558	7,558			7,558	7,558		7,558		7,558	7,558			7,558
% Population in Assessment Age-Group	67%	67%	67%	67%	100%	100%	14%	14%		14%	14%		67%	19%
Average EPC within the Suburb (ug/m ³): Increased Annual Incidence (case per year):	1.7E-01 0.05	1.7E-01 0.06		1.7E-01 0.004	1.7E-01 0.02	1.7E-01 0.003		1.7E-01 0.08		1.7E-01 0.02	1.7E-01 0.010	1.7E-01 0.01	1.7E-01 0.02	1.7E-01 0.003
Increased Annual Incidence (case per year):	0.05	0.06	0.01	0.004	0.02	0.003	0.03	0.08	0.05	0.02	0.010	0.01	0.02	0.003
Moorebank														
Total Population:	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595
% Population in Assessment Age-Group	60%	60%	60%	60%	100%	100%	13%	13%	13%	13%	13%	13%	66%	22%
Average EPC within the Suburb (µg/m ³):	3.5E-01	3.5E-01	3.5E-01	3.5E-01	3.5E-01	3.5E-01	3.5E-01	3.5E-01	3.5E-01	3.5E-01	3.5E-01	3.5E-01	3.5E-01	3.5E-01
Increased Annual Incidence (case per vear):	0.10	0.1	0.03	0.008	0.03	0.005	0.06	0.2	0.09	0.05	0.02	0.02	0.05	0.007
Wattle Grove	8,192	8.192	8.192	8,192	8,192	8,192	0.403	8.192	0.403	0.407	8.192	0.400	8,192	0.400
Total Population: % Population in Assessment Age-Group	8,192	8,192			8,192	8,192	8,192				8,192			8,192 26%
Average EPC within the Suburb (µg/m ³):	4.0F-01	4.0F-01		4.0F-01	4.0E-01	4.0F-01		4.0F-01		4.0F-01	4.0F-01		4.0F-01	4.0E-01
Average EPC within the Suburb (µq/m): Increased Annual Incidence (case per year):	4.0E-01 0.09	4.0E-01 0.10			4.0E-01 0.04	4.0E-01 0.007		4.0E-01 0.08		4.0E-01 0.02	4.0E-01 0.010			4.0E-01 0.010
increased Annual Incluence (case per year).	0.05	0.10	0.02	0.007	0.04	0.007	0.03	0.08	0.05	0.02	0.010	0.010	0.00	0.010
Total Increased Annual Incidence (case per year) - All Suburbs	0.4	0.4	0.09	0.03	0.1	0.02	0.2	0.5	0.3	0.1	0.06	i 0.06	0.2	0.03

Abbreviations: µa/m³: microaram per cubic meter COPD: Chronic Obstructive Pulmonary Disease ED: Emergency Department EPC: Exoosure Point Concentration MPE: Moorebank Precinct East MPW: Moorebank Precinct West PM: Particulate Matter

Table A-8 Calculation of Increased Annual Incidence for Mortality and Morbidity Endpoints - NO2 (cumulative Proposal)

Endpoint:					Mortality-	Mortality - Cardiovascular Disease	Admissions - Respiratory		Hospital Admissions - Respiratory Disease	ED Visits Asthma
Exposure Period:	Annual Average	Annual Average	Annual Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average	24-Hour Average
Age Group:	30+ years	30+ years	30+ years	All ages	All ages	All ages	65+ years	65+ years	15-64 years	1-14 years
β (Exposure Response Function per 1 μg/m ³ Increase in NO ₂):			0.0028	0.001	0.0023	0.001	0.003	0.0014	0.001	0.0006
Baseline Health Incidence Rate (per person)		0.002987	0.00050	0.00529	0.00050	0.001551	0.04476	0.09159	0.00899	0.00804
	0.01005	0.002907	0.00050	0.00323	0.00050	0.001331	0.04470	0.09139	0.00033	0.00004
Casula										
Total Population:	14,696	14,696	14,696	14,696	14,696	14,696	14,696	14,696	14,696	14,696
% Population in Assessment Age-Group	49%	49%	49%	100%	100%	100%	10%	10%	67%	23%
Average EPC within the Suburb (µg/m ³):	6.6E+00	6.6E+00	6.6E+00	6.6E+00	6.6E+00	6.6E+00	6.6E+00	6.6E+00	6.6E+00	6.6E+00
Increased Annual Incidence (case per year):	1.4	0.4	0.07	0.5	0.1	0.1	1.3	1.3	0.6	0.1
Glenfield										
Total Population:	7,558							7,558		
% Population in Assessment Age-Group	67%	67%	67%	100%	100%	100%				19%
Average EPC within the Suburb (µg/m ³):	5.0E+00	5.0E+00	5.0E+00	5.0E+00	5.0E+00	5.0E+00	5.0E+00	5.0E+00		5.0E+00
Increased Annual Incidence (case per year):	0.8	0.2	0.04	0.2	0.04	0.06	0.7	0.7	0.2	0.03
Moorebank										
Total Population:	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595	7,595
% Population in Assessment Age-Group	60%	60%	60%	100%	100%	100%	13%	13%		22%
Average EPC within the Suburb (µg/m ³):	1.0E+01	1.0E+01	1.0E+01	1.0E+01	1.0E+01	1.0E+01	1.0E+01	1.0E+01	1.0E+01	1.0E+01
Increased Annual Incidence (case per year):	1.4	0.4	0.07	0.4	0.09	0.1	1.3	1.3	0.5	0.08
Wattle Grove										
Total Population:	8,192									
% Population in Assessment Age-Group	45%	45% 1.1E+01	45%	100%	100%	100%	5.2%	5.2%		26%
	Average EPC within the Suburb (µq/m ³): 1.1E+01		1.1E+01	1.1E+01	1.1E+01	1.1E+01	1.1E+01	1.1E+01		1.1E+01
Increased Annual Incidence (case per year):	Increased Annual Incidence (case per year): 1.2 0.3		0.06	0.5	0.1	0.1	0.6	0.6	0.6	0.1
Total Increased Annual Incidence (case per year) - All Suburbs	4.8	1.3	0.2	1.6	0.4	0.5	4.0	3.8	1.8	0.3

Abbreviations: µg/m³: microgram per cubic meter ED: Emergency Department EPC: Exposure Point Concentration MPE: Moorebank Precinct East MPW: Moorebank Precinct West NO2: Nitrogen Dioxide

Table A-9 Calculation of Increased Annual Incidence for Mortality and Morbidity Endpoints - SO2 (cumulative Proposal)

Endpoint:	All Causes	Mortality- Respiratory	Mortality - Cardiovascular Disease	Hospital Admissions - Respiratory Disease	ED Visits Asthma
Exposure Period:	24-Hour Average	24-Hour Average	24-Hour Average	1-Hour Maximum	24-Hour Average
Age Group:	All ages	All ages	All ages	15-64 years	1-14 years
β (Exposure Response Function per 1 µg/m ³ Increase in SO ₂):	0.0006	0.0013	0.0008	0.002	0.008
Baseline Health Incidence Rate (per person)	0.00529	0.00050	0.001551	0.00899	0.00804
Casula					
Total Population:					
% Population in Assessment Age-Group				67%	
Average EPC within the Suburb (µg/m ³):		3.4E-02		3.4E-02	
Increased Annual Incidence (case per year):	0.002	0.0003	0.0006	0.006	0.007
Glenfield		1			
Total Population:				7,558	7,558
% Population in Assessment Age-Group	100%	100%	100%	67%	19%
Average EPC within the Suburb (µg/m ³):	2.5E-02	2.5E-02	2.5E-02	2.5E-02	2.5E-02
Increased Annual Incidence (case per year):		0.00012	0.0002	0.002	
Moorebank					
Total Population:	7,595	7,595	7,595	7,595	7,595
% Population in Assessment Age-Group					
Average EPC within the Suburb (µg/m ³):		5.9E-02	5.9E-02	5.9E-02	5.9E-02
Increased Annual Incidence (case per year):	0.0014	0.0003	0.0006	0.005	
Wattle Grove	<u> </u>				1
Total Population:	8,192	8,192	8,192	8,192	8,192
% Population in Assessment Age-Group				69%	
Average EPC within the Suburb (µg/m ³):		6.1E-02			
Increased Annual Incidence (case per year):					
Total Increased Annual Incidence (case per year) - All Suburbs	0.005	0.0011	0.002	0.02	0.02

Abbreviations:

µg/m³: microgram per cubic meter ED: Emergency Department EPC: Exposure Point Concentration MPE: Moorebank Precinct East MPW: Moorebank Precinct West SO₂: Sulfur Dioxide Table A-10 Calculation of Increased Annual Incidence for Mortality and Morbidity Endpoints - CO (cumulative Proposal)

Endpoint:	Mortality -	Hospital Admissions -	Hospital Admissions - Cardiovascular Disease
Exposure Period:	8-Hour Average	8-Hour Average	8-Hour Average
Age Group:	30+ years	65+ years	65+ years
β (Exposure Response Function per 1 µg/m ³ Increase in CO):	0.000001	0.000003	0.0000014
Baseline Health Incidence Rate (per person)	0.01065	0.09159	0.01236

Casula			
Total Population:	14,696	14,696	14,696
% Population in Assessment Age-Group	49%	10%	10%
Average EPC within the Suburb (µg/m ³):	4.1E+00	4.1E+00	4.1E+00
Increased Annual Incidence (case per year):	0.0003	0.002	0.00011
Glenfield			
Total Population:	7,558	7,558	7,558
% Population in Assessment Age-Group	67%	14%	14%
Average EPC within the Suburb (µg/m ³):	3.2E+00	3.2E+00	3.2E+00
Increased Annual Incidence (case per year):	0.0002	0.0009	0.00006
Moorebank			
Total Population:	7,595	7,595	7,595
% Population in Assessment Age-Group	60%	13%	13%
Average EPC within the Suburb (µg/m ³):	6.8E+00	6.8E+00	6.8E+00
Increased Annual Incidence (case per year):	0.0003	0.002	0.00011
Wattle Grove			
Total Population:	8,192	8,192	8,192
% Population in Assessment Age-Group	45%	5.2%	5.2%
Average EPC within the Suburb (µg/m ³):	8.0E+00	8.0E+00	8.0E+00
Increased Annual Incidence (case per year):	0.0003	0.0009	0.00006
Total Increased Annual Incidence (case per year) - All Suburbs	0.0011	0.005	0.0003

Abbreviations: µg/m³: microgram per cubic meter CO: Carbon Monoxide EPC: Exposure Point Concentration MPE: Moorebank Precinct East

MPW: Moorebank Precinct West

APPENDIX 3 CALCULATION OF EXCESS LIFETIME CANCER RISK

Table B-1 Calculation of Excess Lifetime Cancer Risks (Proposal)

																	Chemical:	DPM	Benzene	1,3-Butadiene	PAHs (as BaP TEQ)
																UR	F (µg/m ³) ⁻¹	0.0003	0.000029	0.00017	0.0011
																	URF Source:	Cal/EPA (2016)	Cal/EPA (2016)	Cal/EPA (2016)	Cal/EPA (2016)
																	ona oourcer	00,2177 (2010)	COI) 21 / (2010)	Cdi/2177 (2010)	Cul/ 21/1 (2010)
					٨٣	nual Averag		(m ³)													
				MDE OL					2 ()W					EP	C (µg/m³)		Inhalation				
Suburb	Receptor Location	Receptor Type		MPE Stag	e 2 (IMT Facility			MPE Stage	2 (Warehousing		MAF ^a						Intake		Excess Lifetin	e Cancer Risk	
			DPM	Benzene	1,3-Butadiene	PAHs (as BaP TEQ)	DPM		1,3-Butadiene	PAHs (as BaP TEQ)		DPM	Ben	zene	1,3-Butadiene	PAHs (as BaP TEQ)	Factor ^b				
Casula	Lakewood Crescent	Residential	3.5E-02	5.0E-03	2.1E-03	2.4E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	3.5E-	02 5.0	0E-03	2.1E-03	2.4E-06	0.50			1.8E-07	
Casula	St Andrews Boulevard	Residential	4.0E-02	6.0E-03	2.5E-03	2.8E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	4.0E-		0E-03	2.5E-03	2.8E-06	0.50		8.8E-08	2.1E-07	
Casula	Buckland Road	Residential	3.7E-02	6.1E-03	2.5E-03	2.5E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	3.7E-		1E-03	2.5E-03	2.5E-06	0.50		8.8E-08	2.2E-07	1.4E-09
Casula	Dunmore Crescent	Residential	2.5E-02		1.9E-03	1.7E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	2.5E-		5E-03	1.9E-03	1.7E-06	0.50		6.5E-08	1.6E-07	
Casula	Leacocks Lane	Residential	4.5E-03	8.6E-04	3.6E-04	3.1E-07	0.0E+00	0.0E+00	0.0E+00	0.0E+00		4.5E-		6E-04	3.6E-04	3.1E-07	0.50			3.1E-08	
Casula	Leacocks Lane Mid	Residential	7.7E-03	1.5E-03	6.2E-04	5.3E-07	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	7.7E-		5E-03	6.2E-04	5.3E-07	0.50			5.3E-08	2.9E-10
Casula	Slessor Road	Residential	1.2E-02	2.2E-03	9.0E-04	8.0E-07	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	1.2E-		2E-03	9.0E-04	8.0E-07	0.50			7.7E-08	4.4E-10
Casula	Maple Grove Retirement Village	Residential	4.8E-03	9.0E-04	3.8E-04	3.3E-07	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	4.8E-		0E-04	3.8E-04	3.3E-07	0.50			3.2E-08	1.8E-10
Casula	All Saints Catholic College	Residential/School	7.0E-03	1.3E-03	5.6E-04	4.8E-07	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	7.0E-		3E-03	5.6E-04	4.8E-07	0.50			4.8E-08	2.7E-10
Casula	Casula High School	Residential/School	4.1E-03	7.7E-04	3.2E-04	2.8E-07	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	4.1E-		7E-04	3.2E-04	2.8E-07	0.50			2.7E-08	1.5E-10
Casula	Casula Primary School	Residential/School	1.2E-02	2.0E-03	8.6E-04 6.9E-04	7.9E-07 6.0E-07	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	1.2E-		0E-03 7E-03	8.6E-04 6.9E-04	7.9E-07 6.0E-07	0.50		3.0E-08	7.3E-08	4.4E-10 1.6E-11
Casula Glenfield	Casula Powerhouse Arts Centre Canterbury Road	Recreational Residential	8.7E-03	1.7E-03 1.5E-03	6.9E-04 6.2E-04	5.5E-07	0.0E+00 0.0E+00	0.0E+00	0.0E+00 0.0E+00	0.0E+00	1.3	8.7E-		7E-03	6.9E-04	5.5E-07	0.024		1.1E-09 2.2E-08	2.8E-09 5.3E-08	
Glenfield	Ferguson Street	Residential	7.9E-03 8 3E-03	1.6E-03	6.7E-04	5.8E-07		0.0E+00	0.0E+00	0.0E+00	1.0	7.9E-		6E-03	6.7E-04	5.8E-07	0.50			5.7E-08	3.0E-10 3.2E-10
Glenfield	Goodenough Street	Residential	0.3E-03	2.0E-03	8.5E-04	7.5E-07	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	0.3E-		0E-03	8.5E-04	7 5E-07	0.50			7.3E-08	4.1E-10
Glenfield	Cambridge Avenue	Residential	1.0E-02	1.9E-03	7.9E-04	7.0E-07	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	1.1L-	2.	9E-03	7.9E-04	7.0E-07	0.50			6.7E-08	3.8E-10
Glenfield	Glenwood Public School	Residential/School	5.4E-02	1.0E-03	4.3E-04	3.8F-07	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	5.4F-		0F-03	7.9E-04 4.3E-04	3.8F-07	0.50			3.6F-08	2.1E-10
Glenfield	Glenfield Public School	Residential/School	6.2E-03		4.3L-04 4.8E-04	4 3E-07		0.0E+00	0.0E+00	0.0E+00	1.0	6.2E-		2E-03	4.8E-04	4 3E-07	0.50			4.1F-08	
Glenfield	Hurlstone Agricultural School	Residential/School	5.2E-03	9.8F-04	4.1E-04	3.6F-07	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	5.2E-		8E-04	4.1E-04	3.6F-07	0.50			3.5E-08	
Glenfield	Glenfield Rise Development	Residential	8.6E-02	1.8F-02	7.4E-03	5.9E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	8.6E-		8E-04	7.4E-03	5.9E-06	0.50		2.6E-07	6.3E-07	3.2E-09
Glenfield	Playground Learning Centre	Residential	3.3E-02	6.3E-03	2.6F-03	2.3E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	3.3E-		3E-03	2.6E-03	2.3E-06	0.50	5.0E-06		2.2E-07	1.2E-09
Moorebank	Anzac Road	Residential	7.7F-02	1.4E-02	5.9E-03	5.1E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	7 7E-		4F-02	5.9E-03	5.1E-06	0.50		2.0E-07	5.0E-07	2.8E-09
Moorebank	Anzac Road	Commercial/Industrial	7 7E-02		5.9E-03	5 1E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1 3	7 7E-		4F-02	5.9E-03	5 1E-06	0.094			9.4E-08	5 2E-10
Moorebank	Church Road	Residential	2.5E-02	3.7E-03	1.6E-03	1.7E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	2 5E-	02 3	7E-03	1.6E-03	1.7E-06	0.50	3.7E-06	5.4E-08	1.3E-07	9.5E-10
Moorebank	New DNSDC Facility	Commercial/Industrial	6.0E-03	1.1E-03	4.7E-04	4.2E-07	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.3	6.0E-		1E-03	4.7E-04	4.2E-07	0.094			7.5E-09	
Wattle Grove	Wallcliff Court	Residential	3.8E-02		3.1E-03	2.8E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	3.8E-		4E-03	3.1E-03	2.8E-06	0.50			2.6E-07	
Wattle Grove	Corryton Court	Residential	6.0E-02	1.2E-02	4.9E-03	4.5E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	6.0E-	02 1.	2E-02	4.9E-03	4.5E-06	0.50		1.7E-07	4.2E-07	2.5E-09
Wattle Grove	Martindale Court	Residential	6.0E-02	1.2E-02	5.0E-03	4.4E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	6.0E-	02 1.	2E-02	5.0E-03	4.4E-06	0.50	9.0E-06	1.7E-07	4.2E-07	2.4E-09
Wattle Grove	Yallum Court	Residential	5.2E-02	1.0E-02	4.3E-03	4.0E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	5.2E-	02 1.0	0E-02	4.3E-03	4.0E-06	0.50		1.5E-07	3.6E-07	2.2E-09
Wattle Grove	Wattle Grove Public School	Residential/School	4.3E-02	8.5E-03	3.6E-03	3.3E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	4.3E-	02 8.	5E-03	3.6E-03	3.3E-06	0.50	6.5E-06	1.2E-07	3.0E-07	1.8E-09
Wattle Grove	St Marks Coptic College	Residential/School	2.8E-02	5.4E-03	2.3E-03	2.0E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	2.8E-	02 5.4	4E-03	2.3E-03	2.0E-06	0.50	4.1E-06	7.8E-08	1.9E-07	1.1E-09
Wattle Grove	Wattle Grove Long Day Care Centre	Residential	3.4E-02	6.1E-03	2.5E-03	2.4E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0	3.4E-	02 6.	1E-03	2.5E-03	2.4E-06	0.50	5.2E-06	8.8E-08	2.2E-07	1.3E-09
Wattle Grove	Wattle Grove Long Day Care Centre	Recreational	3.4E-02	6.1E-03	2.5E-03	2.4E-06	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.3	3.4E-	02 6.1	1E-03	2.5E-03	2.4E-06	0.024	2.5E-07	4.2E-09	1.0E-08	6.2E-11

Maximum Residential/School Receptors	1.3E-05	2.6E-07	6.3E-07	3.2E-09
Maximum Recreational Recepto	2.5E-07	4.2E-09	1.0E-08	6.2E-11
Maximum Commercial/Industrial Recepto	2.2E-06	3.8E-08	9.4E-08	5.2E-10

Abbreviations and Notes:

Jud/m²: microgram per cubic meter BaP: Benzo(a)pyrene Cal/EPA: California Environmental Protection Agency DPM: Diesel Particulate Matter EPC: Exposure Point Concentration GLC: Ground Level Concentration IMEX: Import Export IMT: Intermodal Terminal

MAF: Modeling Adjustment Factor MPW: Moorebank Precinct West

PAH: Polycyclic Aromatic Hydrocarbon TEQ: Toxicity Equivalent

URF: Unit Risk Factor

¹⁰¹ The MPX State 2 warehousing would operate 18 hours per day, while the model predicted GLCs were annualized over 24 hours per day. Therefore, the annual average GLCs were lower than the actual air concentrations emitted from the source. Commercial/industrial and recreational receptors may not be continuously exposed to chemicals in air, and using annual average GLCs instead of the actual air concentrations by multiplying a MAF (calculated as 24 hours/18 hours = 1.3). ¹¹ Exposure time (ET) was assumed to be 24 hours per day, and 4 hours per day for residential/school, commercial/industrial, and recreational receptors, respectively. Exposure frequency (EF) was assumed to be 365 days per year, and 104 days per year for residential/school, commercial/industrial, and recreational receptors, respectively. Inhalation Intake Factor was calculated as ET x EF x ED/CF/AT, where AT is averaging time for cancer risk (i.e. 70 years or 25550 days), and CF is conversion factor (24 hours/dav).

Source: California Environmental Protection Agency (Cal/EPA) Office of Environmental Health Hazard Assessment (OEHHA). 2016. OEHHA Toxicity Criteria Database. Available at: http://oehha.ca.gov/tcdb/index.asp

																											Chemical:	DPM	Benzene	1,3-Butadiene	PAHs (as E TEQ)
																											F (µg/m ³) ⁻¹	0.0003	0.000029	0.00017	0.0011
																										U	JRF Source: 0	Cal/EPA (2016)	Cal/EPA (2016)	Cal/EPA (2016) C	Cal/EPA (20
																							- 1								
Suburb	Receptor Location	Receptor Type											rage GLC (µg/m	¹)										EPC (µg/m ³)			Inhalation				
			MPE Stage 2 (IMT)			MPE Stage 2 (Warehousing)				MPW Stage 2 (IMT Facility)			MPW Stage 2 (Warehousing)				MPE Stage 1 (IMEX Facility)			M	AF *				Intake		Excess Lifetime	e Cancer Risk			
			DPM	Benzene 1,3-Butadi		PAHs (as BaP TEO)	DPM	Benzene	1,3-Butadiene	PAHs (as	DPM	Benzene	1,3-Butadiene	PAHs (as	DPM	Benzene 1,3	1,3-Butadiene	PAHs (as	DPM	Benzene	1,3-Butadiene	PAHs (as BaP TEO)	DPM	M Benzene		PAHs (as BaP TEO)	Factor ^b				
ula	Lakewood Crescent	Residential	3.5E-02	5.0E-03	2 1E-03		0.05.00	0.0E+00	0.0E+00	0.0E+00	3.55.01	6.8E-03	1.4E-03	Bap TEOT	4.8F-02	7 4E-03	2.15.02	2 SE 07	8 5E-02	2.05.02	6.4E-04	Bap TEOT	1.0 4.15.01	2 2E-02	7.25.02	6 1E-06	0.50	6.2E-05	2.25.07	6.2E-07	3.
	St Andrews Boulevard	Residential	4.0E-02			2.4E-0			0.0E+00	0.0E+00	3 2E-01	9.2E-03	1.9E-03	1.5E-06		1.2E-02	4 9F-03		1.1E-01	3.8E-03	8.2E-04	2.2E-06		3.1E-02	1.0E-02	7.6E-06	0.50	8.2E-05	3.2E-07 4.4E-07	8.7E-07	4
	Buckland Road		3.7E-02			2.8E-0			0.0E+00	0.0E+00	3.2E-01	9.8E-03	2 1E-03	1.82-06		1.2E-02	6 1E-03		1.3E-01		9.9E-04	3.3E-06		3.5E-02	1.2E-02	8 1E-06	0.50	9.1E-05	5.1E-07	9.9E-07	4
la	Dunmore Crescent	Residential	2.5E-02		1.9E-03			0.0E+00	0.0E+00	0.0E+00	3.3E-01	5.7E-03	1 3E-03	1.75-06	5.1E-02	8.0E-02	3.4E-03		1.3E-01		8.5E-04	2.7E-06		2 2E-02	7.4E-03	5.6E-06	0.50	6.0E-05	3.2E-07	6.3E-07	
la	Leacocks Lane		4.5E-02		3.6E-04		7 0.0E+00		0.0E+00	0.0E+00	3.2E-01	8.1E-04	1.9E-04	1.5E-07			4.0E-04		1.9E-02		1.3E-04	4.0E-07	1.0 6.1E-02		1.1E-03		0.50	9.2E-06	4.7E-08	9.2E-08	4
la la	Leacocks Lane Mid	Residential	4.5E-03 7.7E-03		6.2E-04			0.0E+00	0.0E+00	0.0E+00	4.7E-02	8.1E-04 1 1E-03	2.8E-04			9.4E-04	4.0E-04 5.3E-04		3.4E-02		2.4E-04	4.0E-07		4.9E-03	1.7E-03	9.0E-07	0.50	9.2E-06 1.4E-05	4.7E-08 7.1E-08	9.2E-08	-
	Electoric Road	Residential	1.2E-02		9.0F-04			0.0E+00	0.0E+00		4.7E-02	1.1E-03	2.8E-04 5.3E-04	2.3E-07			5.3E-04 8.4F-04		3.4E-02 8.3E-02		2.4E-04 5.8E-04		1.0 9.6E-02		2.9E-03	2.9E-06	0.50	3.0E-05	1.2E-07	2.4E-07	
	Maple Grove Retirement Village	Residential	4.8E-03	9.0E-04	3.8E-04			0.0E+00	0.0E+00	0.0E+00	9.0E-02	7.1E-04	5.3E-04 1.7E-04	4.3E-07 1.3E-07		2.0E-03 8.0E-04	3.4E-04		8.3E-02 1.9E-02		5.8E-04 1.4E-04	4.3E-05	1.0 2.0E-01		2.9E-03 1.0E-03	2.9E-06 9.2E-07	0.50	3.0E-05 8.5E-06	4.4E-08	2.4E-07 8.7E-08	
	All Saints Catholic College	Residential/School	4.8E-03 7.0E-03		3.8E-04			0.0E+00	0.0E+00	0.0E+00	4 3E-02	7.1E-04 1.1E-03	2.6E-04				5.2E-04		2.8E-02		2.0E-04	4.3E-07	1.0 S./E-02		1.5E-03	1.3E-06	0.50	1.3E-05	4.4E-08	1.3E-07	
	Casula High School	Residential/School	4.1E-03	1.3E-03			7 0.0E+00 7 0.0E+00			0.0E+00	4.3E-02	6.5E-04							2.8E-02			3 SE-07	1.0 8.6E-02		1.5E-03 8.9E-04	7.7E-05	0.50		6.6E-08		
a			4.1E-03	7.7E-04	3.2E-04				0.0E+00		2.5E-02 7.5E-02		1.5E-04	1.2E-07			3.1E-04				1.1E-04				8.9E-04	7.7E-07		7.4E-06	3.9E-08	7.6E-08	
3	Casula Primary School	Residential/School	1.2E-02	2.0E-03	8.6E-04			0.0E+00	0.0E+00	0.0E+00		2.0E-03	4.5E-04	3.7E-07	1.6E-02	2.6E-03	1.1E-03		4.7E-02		3.5E-04	1.1E-06		8.2E-03	2.7E-03	2.3E-06	0.50	2.3E-05	1.2E-07	2.3E-07	
	Casula Powerhouse Arts Centre	Recreational	8.7E-03	1.7E-03	6.9E-04	6.0E-0		0.0E+00	0.0E+00	0.0E+00	5.5E-02	1.4E-03	3.3E-04	2.7E-07	1.0E-02	1.6E-03	6.6E-04	5.2E-08	3.8E-02	1.2E-03	2.7E-04	8.0E-07		6.3E-03	2.2E-03	1.7E-06	0.024	8.2E-07	4.3E-09	8.8E-09	
	Canterbury Road	Residential	7.9E-03	1.5E-03	6.2E-04		7 0.0E+00	0.0E+00	0.0E+00	0.0E+00	4.9E-02	1.1E-03	2.9E-04	2.3E-07	7.6E-03	1.2E-03	5.0E-04		4.8E-02	1.6E-03	3.4E-04	9.8E-07	1.0 1.1E-01		1.8E-03	1.8E-06	0.50	1.7E-05	7.8E-08 8.2E-08	1.5E-07	
ield	Ferguson Street	Residential	8.3E-03	1.6E-03	6.7E-04	5.8E-0	7 0.0E+00	0.0E+00	0.0E+00	0.0E+00	5.1E-02	1.1E-03	3.0E-04	2.4E-07		1.2E-03	5.2E-04		5.2E-02		3.7E-04	1.1E-06	1.0 1.2E-01	5.7E-03	1.9E-03	1.9E-06	0.50	1.8E-05	8.2E-08	1.6E-07	
field	Goodenough Street	Residential	1.1E-02	2.0E-03	8.5E-04	7.5E-0	7 0.0E+00	0.0E+00	0.0E+00	0.0E+00	6.5E-02	1.4E-03	3.9E-04	3.1E-07		1.5E-03	6.3E-04	5.0E-08	7.3E-02	2.4E-03	5.2E-04	1.4E-06	1.0 1.6E-01	7.3E-03	2.4E-03	2.6E-06	0.50	2.4E-05	1.1E-07	2.0E-07	
ield	Cambridge Avenue	Residential	1.0E-02	1.9E-03	7.9E-04		7 0.0E+00		0.0E+00	0.0E+00	6.3E-02	1.4E-03	3.8E-04	3.0E-07		1.5E-03	6.2E-04		6.3E-02		4.5E-04	1.2E-06	1.0 1.5E-01		2.2E-03	2.3E-06	0.50	2.2E-05	9.8E-08	1.9E-07	
ield	Glenwood Public School	Residential/School	5.4E-03	1.0E-03	4.3E-04	3.8E-0	7 0.0E+00	0.0E+00	0.0E+00	0.0E+00	2.8E-02	6.8E-04	1.7E-04	1.4E-07	4.6E-03	7.2E-04	3.0E-04	2.4E-08	2.7E-02	9.3E-04	2.0E-04	5.7E-07	1.0 6.6E-02	3.3E-03	1.1E-03	1.1E-06	0.50	9.8E-06	4.9E-08	9.3E-08	
field	Glenfield Public School	Residential/School	6.2E-03	1.2E-03	4.8E-04	4.3E-0	7 0.0E+00	0.0E+00	0.0E+00	0.0E+00	3.6E-02	8.4E-04	2.1E-04	1.7E-07		9.1E-04	3.8E-04	3.0E-08	3.4E-02	1.2E-03	2.5E-04	7.2E-07	1.0 8.2E-02		1.3E-03	1.3E-06	0.50	1.2E-05	5.9E-08	1.1E-07	
field field field	Hurlstone Agricultural School		5.2E-03	9.8E-04	4.1E-04		7 0.0E+00		0.0E+00	0.0E+00	3.0E-02	7.1E-04	1.8E-04 1.3E-03	1.4E-07			3.2E-04		2.8E-02		2.0E-04 1.3E-03	5.9E-07 4.5E-06	1.0 6.8E-02		1.1E-03	1.1E-06	0.50	1.0E-05 7.7E-05	4.9E-08 5.0E-07	9.4E-08	
ield	Glenfield Rise Development	Residential	8.6E-02	1.8E-02	7.4E-03			0.0E+00	0.0E+00	0.0E+00	2.2E-01	5.9E-03			3.1E-02	4.9E-03	2.1E-03		1.8E-01				1.0 5.2E-01	3.5E-02	1.2E-02	1.2E-05	0.50		5.0E-07		
ield	Playground Learning Centre	Residential	3.3E-02			2.3E-0			0.0E+00	0.0E+00	1.3E-01	3.4E-03	7.7E-04			3.0E-03			8.5E-02		6.3E-04	2.1E-06	1.0 2.7E-01	1.6E-02	5.3E-03	5.1E-06	0.50	4.0E-05	2.3E-07	4.5E-07	
ebank	Anzac Road	Residential	7.7E-02			5.1E-0	6 0.0E+00	0.0E+00		0.0E+00	3.3E-01	8.7E-03	1.9E-03	1.6E-06	4.4E-02	6.9E-03			1.3E-01		9.9E-04		1.0 5.8E-01	3.4E-02	1.2E-02	1.0E-05	0.50	8.7E-05	5.0E-07	1.0E-06	
ebank	Anzac Road	Commercial/Industrial	7.7E-02	1.4E-02	5.9E-03	5.1E-0	6 0.0E+00	0.0E+00	0.0E+00	0.0E+00	3.3E-01	8.7E-03	1.9E-03	1.6E-06	4.4E-02	6.9E-03	2.9E-03	2.3E-07	1.3E-01	4.6E-03	9.9E-04	3.5E-06	1.3 5.9E-01	3.7E-02	1.3E-02	1.0E-05	0.094	1.7E-05	1.0E-07	2.0E-07	
ebank	Church Road	Residential	2.5E-02		1.6E-03	1.7E-0	6 0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.2E-01	3.1E-03	6.9E-04		1.8E-02	2.9E-03	1.2E-03	9.7E-08	5.5E-02	1.9E-03	4.1E-04	1.4E-06	1.0 2.2E-01	1.2E-02	3.9E-03	3.8E-06	0.50	3.3E-05	1.7E-07	3.3E-07	
ebank	New DNSDC Facility	Commercial/Industrial	6.0E-03	1.1E-03	4.7E-04	4.2E-0	7 0.0E+00	0.0E+00	0.0E+00	0.0E+00	3.4E-02	8.0E-04	2.0E-04	1.6E-07	5.5E-03	8.6E-04	3.6E-04	2.9E-08	3.3E-02	1.1E-03	2.3E-04	6.8E-07	1.3 8.0E-02	4.2E-03	1.4E-03	1.3E-06	0.094	2.3E-06	1.1E-08	2.2E-08	
Grove	Wallcliff Court	Residential	3.8E-02		3.1E-03		6 0.0E+00		0.0E+00	0.0E+00	9.7E-02	2.4E-03	5.9E-04	4.6E-07	1.5E-02	2.3E-03	9.7E-04	7.7E-08	1.6E-01	6.0E-03	1.2E-03	3.5E-06	1.0 3.1E-01	1.8E-02	5.8E-03	6.8E-06	0.50	4.7E-05	2.6E-07	5.0E-07	
e Grove	Corryton Court	Residential	6.0E-02		4.9E-03	4.5E-0	6 0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.3E-01	3.4E-03	7.8E-04	6.1E-07	1.9E-02	3.0E-03	1.3E-03	1.0E-07	1.9E-01	6.9E-03	1.4E-03	4.2E-06	1.0 4.0E-01	2.5E-02	8.3E-03	9.5E-06	0.50	6.0E-05	3.6E-07	7.1E-07	
Grove	Martindale Court	Residential		1.2E-02	5.0E-03			0.0E+00	0.0E+00	0.0E+00	1 4E-01	3.8E-03	8.7E-04	6.8E-07	2 1E-02	3 3E-03	1.4E-03		1.7E-01		1.3E-03		1.0 4.0E-01	2 SE-02	8 5E-03		0.50	5.9E-05	3.7E-07	7.2E-07	
e Grove	Yallum Court	Residential	5.2E-02		4 3E-03			0.0E+00	0.0E+00	0.0E+00	1.2E-01	2.9E-03	7 0F-04	5.5E-07		2 7E-03	1.1E-03		2.0E-01		1.5E-03	4.4E-06		2.3E-02	7.6E-03	9.0E-06	0.50	5.8E-05	3.4E-07	6 5E-07	
e Grove	Wattle Grove Public School	Residential/School		8.5E-03		3.3E-0			0.0E+00	0.0E+00	1.1E-01	2.7E-03	6.3E-04			2.5E-03			1.5E-01		1.1E-03	3.3E-06		1.9E-02	6.3E-03		0.50	4.7E-05	2.8E-07	5.4E-07	
Grove	St Marks Coptic College	Residential/School	2.8E-02	5.4E-03		2.0E-0			0.0E+00	0.0E+00	8 1E-02	2.1E-03	4.9E-04	3.9E-07					9.3E-02		6.9E-04		1.0 2.1E-01		4 3E-03		0.50	3.2E-05	1.9E-07	3.6E-07	
Grove	Wattle Grove Long Day Care Centre	Residential	3.4E-02		2.5E-03			0.0E+00	0.0E+00	0.0E+00	3 1E-01	8.6E-03	1.9E-03	1.5E-06		1.3E-02	5 SE-03		1.7E-01		1.3E-03	4.0E-06		3.4F-02	4.3E-03	8.4E-06	0.50	9.0E-05	4.9E-07	9.5E-07	
	Wattle Grove Long Day Care Centre	Recreational	3.4E-02		2.5E-03		5 0.0E+00		0.0E+00	0.0E+00	3.1E-01	8.6E-03	1.9E-03	1.5E-06		1.3E-02	5.5E-03		1.7E-01		1.3E-03	4.0E-06	1.3 6.3E-01		1.1E-02		0.024	4.5E-06	2.6E-08	5.3E-08	
e giove	wattle Grove Lond Dav Care Centre	Recreational	3.46-02	0.12-03	2.32-03	2.46-0	0.02700	0.02700	0.02700	0.02700	3.12-01	8.06-03	1.92-03	1.32-00	0.46-02	1.36*02	3.35-03	4.46*07	1.72-01	3.92-03	1.32-03	N.UE-00	1.3 0.32-01	3.0E-UZ	1.32*02	8.36-00	0.024	4.32-00	2.02-08	3.35-08	
																									Maximum Reside	ential/School	Receptors	9.1E-05	5.1E-07	1.0E-06	
																									Maximur	n Recreation	al Receptor	4.5E-06	2.6E-08	5.3E-08	2
																									aximum Commer	int (Testuctule	al Basantas	1.7E-05	1.0E-07	2.0E-07	1

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Source: California Environmental Protection Agency (Cal/EPA) Office of Environmental Health Hazard Assessment (OEHHA). 2016. OEHHA Toxicity Criteria Database. Available at: http://oehha.ca.gov/tcdb/index.asp

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